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Red Ear Syndrome Precipitated by a Dietary Trigger: A Case Report

Chung Chi Chan[1,2*], Susmita Ghosh[3]

Abstract

Introduction: Red ear syndrome is a rare condition characterized by episodic attacks of erythema of the ear accompanied by burning ear pain. Symptoms are brought on by touch, exertion, heat or cold, stress, neck movements and washing or brushing of hair. Diagnosis and treatment of this condition are challenging. The case we report here involves a woman whose symptoms were brought on by a dietary trigger: orange juice as well as stress, causing significant physical and psychological morbidity. Avoidance of triggers resulted in symptomatic improvement.

Case presentation: A 22-year-old Caucasian woman who was a student presented twice to our department with evolving symptoms, the first time with hyperacusis (abnormal sound sensitivity arising from within the auditory system to sounds of moderate volume), intermittent right tinnitus and subjective hearing difficulties. She presented five years later with highly distressing episodes of erythematous ears, which were associated with burning pain around the ear and temporal areas, and intolerance to noise. After keeping a symptom diary, she identified orange juice and stress as triggers of her symptoms. No local head and neck pathology was present. Investigations and imaging were negative. Avoidance of triggers led to great symptomatic improvement. To the best of our knowledge, dietary triggers have not previously been reported as a trigger for this syndrome. This case shows a direct temporal link to a dietary trigger and supports a primary pathogenesis. Recognition and management of primary headache disorder and simple dietary and lifestyle changes brought about symptomatic relief.

Conclusion: Red ear syndrome is a little-known clinical syndrome of unknown etiology and management. To the best of our knowledge, our present case report is the first to describe primary red ear syndrome triggered by orange juice. Clinical benefit derived from avoidance of this trigger, which is already known to precipitate migraines, gives some insight into the pathogenesis of red ear syndrome.

Keywords: Dietary trigger, Erythema, Lifestyle modifications, Migraine, Red ear syndrome

Introduction

Red ear syndrome (RES) is a rare condition characterized by episodic erythema of the ear accompanied by burning sensation or otalgia. One or, less commonly, both ears may be affected, and erythema may extend beyond the ear to the face. Symptoms may be spontaneous or triggered by touch, exertion, heat or cold, stress, neck movements, sneezing, coughing, chewing and/or brushing of hair [1]. Recognition of this condition is important but difficult because of its rarity.

Case Presentation

A 22-year-old Caucasian woman who was a student presented to our neuro-otology clinic on two separate occasions five years apart. Her initial symptoms were a six-month history of intermittent right-sided tinnitus and bilateral hyperacusis (abnormal sound sensitivity arising from within the auditory system to normal or moderate-level ambient noise which would not trouble other people). She also reported right ear fullness and significant difficulty hearing in background noise when stressed.

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Otoscopy, a neuro-otological examination, pure-tone audiometry, tympanometry, stapedial reflexes, otoacoustic emissions (OAEs), auditory brainstem response and speech audiometry results were normal. She had particularly strong transient OAE responses and spontaneous OAE activity bilaterally. This was consistent with increased cochlear gain, suggestive of reduced efficacy of inhibitory feedback in the auditory system. Several sessions of auditory rehabilitation were carried out with a hearing therapist, involving counseling, communication tactics, tinnitus and hyperacusis retraining, advice regarding ear-level noise generators to enable desensitization, relaxation techniques and stress management. Her symptoms had greatly improved at review nine months later, and she was discharged.

She was referred again to our clinic by her general practitioner five years later. Her primary complaint was recurrent one-hour episodes of painful cutaneous erythema of the right external ear (Figures 1 and 2) that was associated with severe right temporal pain radiating down to the mastoid area with transient subjectively reduced hearing, right conjunctival injection, intolerance to noise and light, which was exacerbated during these episodes; the latter symptom was suggestive of involvement of pathways outside the auditory pathway.

These symptoms caused our patient considerable distress, resulting in weekly attendance for three months at her general practitioner's clinic, in addition to presentation at the local emergency departments and to ear, nose and throat clinics, prior to referral to our department. During the previous three months, she had also experienced continuous headaches and fatigue with occasional light-headedness during episodes of erythematous ear. She reported no nausea, visual field symptoms, tinnitus or vertigo.

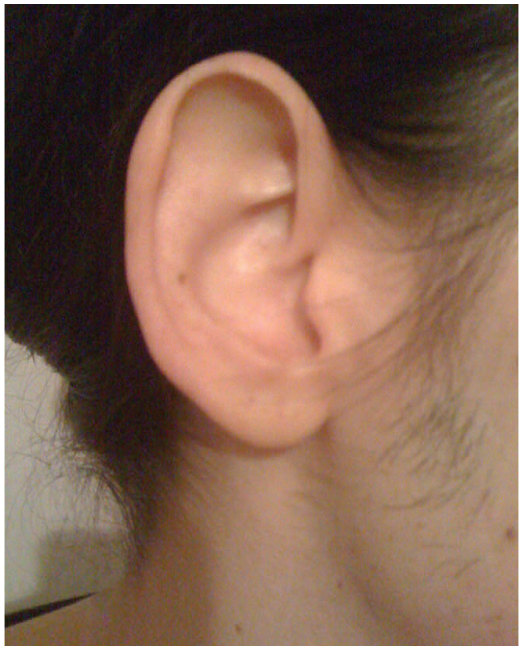

Fig. 1: Normal appearance of the right ear of the patient (photograph taken by patient).

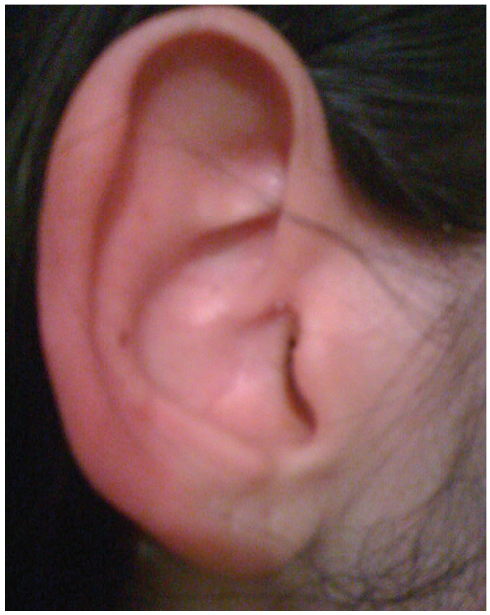

Fig. 2: Right ear during the patient's "red ear" episode (photograph taken by patient).

Differential diagnoses of dermatological, temporomandibular joint, dental, pharyngeal and cervical problems were excluded on the basis of a head and neck examination. On inspection, there was no evidence of erythema or of infection in the ear or mastoid area. The otoscopy findings were normal. A neuro-otological examination was unremarkable, including extra-ocular eye movements, cranial nerves, cerebellar function and clinic room balance tests. Pure-tone audiometry and tympanometry showed normal hearing and middle-ear function. Magnetic resonance imaging of the brain was normal. Routine blood tests were negative. She was diagnosed with RES associated with hyperacusis.

She was reassured that she had no major structural pathology. There were some migrainous features in her medical history. Management of her migraine included starting behavioral modifications, such as reducing caffeine intake, stress reduction, optimizing fluid intake, improving sleep pattern, relaxation techniques and starting exercise. She was advised to keep a symptom diary to identify further triggers. She was offered migraine prophylaxis but declined it.

On review after four months of supportive measures, she was feeling much better, with complete resolution of her headaches and much reduced frequency of episodes of erythematous ear. She reported absence of pain in the ear and less sensation of swelling. Triggers for red ear episodes identified from her symptom diary included stress and, surprisingly, orange juice. Her symptoms were managed successfully without medication for four years.

Discussion

Red ear syndrome is a clinical diagnosis for which there is no specific diagnostic test. One hundred cases have been reported in the literature so far, with an estimated male-to-female ratio of 1:1.25 and a median age at onset of 44 years (with a wide range of ages: 4 to 92 years) [1]. Lance [2] was the first to describe RES in the literature in a 1996 report of 12 patients with recurrent RES. Pain in RES varies from mild to severe [3,4]. Its duration may be seconds [5] or hours [2]. The frequency of episodes may be several times per day, or there may be year-long remission periods [6]. Raieli and colleagues [4] reported that unilateral or bilateral 30- to 60-minute episodes can occur in isolation and be associated with migraine (before, during or after). In 10% of the cases they reported, red ears preceded onset of a painful migraine attack. In their series of 96 children admitted with headache, who ranged from 6 to 18 years of age, 55 (57%) had migraine, and RES was found in 16 migraine cases. RES did not occur in the other headache groups. It was associated with severe pain in 62.5% and neurovegetative symptomatology (nausea, vomiting, phonophobia and/or photophobia) in 50% [4].

Episodes have been reported to occur spontaneously or to be triggered by heat [2]; by entering a hot room [7]; by touch [2,5]; by neck movement [2,5]; by sneezing, coughing, hair-brushing, physical exercise, chewing, and stress [2]; and by exposure to cold and lying on the affected side [8].

There are various views regarding the pathophysiology of this condition. Lance [2] suggested that the syndrome is induced in patients with cervical disorders, predominantly C3 root discharge causing antidromic release of vasodilator peptides (peripheral mechanism). He proposed that the primary mechanism is activation of the trigeminovascular system. He pointed out, and Hirsch [9] reiterated, that parasympathetic vasodilatation is greater in the nose and cheek than in the ear; therefore, red ears must be mediated primarily by inhibition of sympathetic vasoconstriction or activation of sympathetic vasodilatation. Thus, the presence of RES suggests an underlying dysregulation of sympathetic outflow. Purdy [10] noted that, in RES, there is pain in and around the ear associated with autonomic phenomena, including erythema of the ear ipsilateral to the pain. He suggested that the condition be labeled auriculo autonomic cephalalgia or be placed in the trigeminal autonomic cephalgia group. Several authors, including Kumar and colleagues [5], have used brainstem trigeminovascular activation to explain RES associated with migraine. Lambru and colleagues suggested that it is possible that trigeminoautonomic parasympathetic activation occurs with sympathetic deficit. The imbalance between parasympathetic and sympathetic systems thus may facilitate inhibition of sympathetic tone of the ear. Sympathetic dysregulation, not parasympathetic activation as formerly believed, may be the predominant mechanism of RES [1].

Another group [7] has suggested that RES is an auricular form of erythromelalgia with similar burning pain, erythema and increased skin temperature. Erythromelalgia is a condition affecting hands and feet that might be caused by sensory and sympathetic nerve dysfunction.

Red ear syndrome has been associated with various conditions, including upper cervical pathology (arachnoiditis, facet joint spondylosis and cervical root traction), glossopharyngeal and trigeminal neuralgia, temporomandibular joint (TMJ) dysfunction and thalamic syndrome [2]. Associations have been reported with primary headache disorders, including migraine, chronic paroxysmal hemicranias [3], hemicrania continua and the short-lasting unilateral neuralgiform headache with conjunctival injection [11]. Other cases are idiopathic. Donnet and Valade proposed two types of RES: (1) a primary form that occurs in young people and is associated with migraine and (2) a secondary form that occurs in older adults and is associated with cervical disorder or trigeminal autonomic cephalalgia phenomenon [6].

Various treatments for RES have been used with varying success. Among the 12 patients Lance described, one improved with methysergide therapy. One experienced partial symptomatic relief with indometacin, and others with propranolol, application of a cold pack, amitriptyline, or imipramine [2].

Inconsistent results have been reported following treatment with non-steroidal anti-inflammatory drugs [12], topical anesthetics, cooling the ear [7], verapamil and gabapentin [8] and greater auricular nerve blockade with a combination of local anesthetics and steroids. Some authors have reported relief over an eight-week period, and others have noted no benefit [2,13]. Bender [14] suggested that in primary or idiopathic cases, treatment of the coexisting headache disorder with drugs such as propranolol, amitriptyline, imipramine and flunarizine helps to resolve these cases to varying degrees. Secondary forms may be more resistant to treatment [12]. In one review, secondary cases appeared to have a greater response to treatment than idiopathic cases [15]. In secondary cases, such as those associated with TMJ dysfunction, a dental plate was reported to be useful in relieving symptoms [2,9]. In cases associated with chronic paroxysmal hemicrania, indometacin was found to be effective [3,16].

Transient unilateral sensation of aural fullness with tinnitus was described in one of Lance's original cases [2] and blockage without tinnitus in 2 further cases of chronic paroxysmal hemicrania [3]. Aural fullness was present in our case on the first presentation; additional subjective hearing loss in background noise without evidence of hearing loss on audiometry was possibly due to central auditory pathway changes.

Various triggers of RES have been identified; however, we found no reports of dietary triggers that provoke RES. Alcohol and spicy foods are known to cause bilateral facial flushing. Gustatory flushing is mediated by an autonomic neural reflex involving the trigeminal nerve. The presence of a dietary trigger that causes neurological symptoms suggests a migrainous etiology, as such triggers in migraine are well-known and avoidance is a therapeutic mainstay. The non-concentrate orange juice that our patient consumed is a well-known brand in the United Kingdom. There is a possibility that ethyl butyrate (also known as butyric acid ethyl ester), which is often used in flavoring extracts, could be the culprit rather than the orange juice itself. In our patient, dietary, stressor and lifestyle modifications were sufficient to relieve her physical symptoms and psychological distress. We advocate examining a patient's lifestyle and encouraging the patient to keep a symptom diary to identify environmental factors that provoke RES. Clinicians should be made aware of the likelihood of migraine pathogenesis in primary RES and the range of management options available (non-pharmacological lifestyle changes as well as prophylaxis).

Red ear syndrome is a little known condition with much variation in individual patients' symptoms and variable responses to proposed treatments. It may go unrecognized and can cause the patient undue anxiety. Often patients feel ignored without a firm diagnosis or management. Patients may present repeatedly to emergency departments, general practices or various specialist departments (ear, nose and throat; dermatology; neurology; audiovestibular medicine; and/or audiology) before being diagnosed. Raised awareness of this disorder with prompt diagnosis has cost benefits by reducing the number of primary and secondary care presentations, decreasing psychological distress and speeding return to usual daily activities.

Conclusions

Red ear syndrome is a rare syndrome of diverse pathophysiology which is difficult to treat. To our knowledge, our present report is the first to describe a dietary trigger of RES. Successful management with lifestyle modifications and avoidance of migraine triggers gives insight into the pathogenesis of primary migraine-associated RES.

Consent

Written informed consent was obtained from the patient for the publication of this report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

SG reviewed the literature and wrote the initial manuscript drafts. CC managed the patient, reviewed the literature and completed the manuscript. Both authors read and approved the final manuscript.

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A Rare Cause of Excruciating Chest Pain Mimicking Acute Coronary Syndrome

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A 62-year-old male presented to the chest pain unit with chest pain and nausea, reporting that the symptoms occurred one hour after dinner. His medical history included a foudroyant event of pulmonary embolism with embolectomy in 2012.

Due to clinical deterioration and the history of pulmonary embolism, we decided to perform a contrast computed tomography angiography (CT). We could rule out aortic dissection and pulmonary embolism, However, CT revealed a mixed axial paraoesophageal upside-down stomach (UDS) compressing the left ventricle (Fig. 1).

Upside-down stomach is the rarest type of hiatal hernia and can manifest clinically in a wide variety of symptoms as demonstrated in this case [1]. As causes of chest pain, gastrointestinal disease other than peptic ulcer or reflux-related diseases which might include UDS were reported to be below 1% [2]. In UDS patients, complications such as incarceration, volvulus development as well as acute gastric bleeding can lead to a life-threatening emergency with prevalence of 30.4% and can require immediate surgery [3, 4].

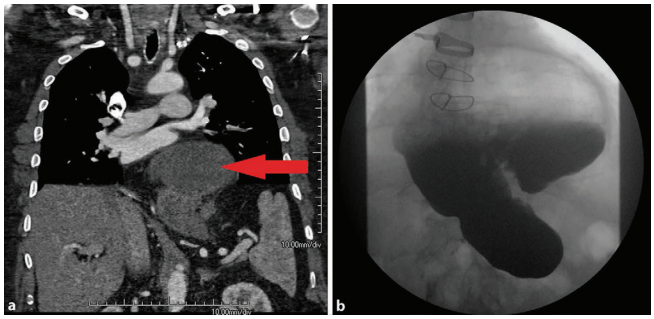

Fig. 1: Computed tomography (**a**) and gastrointestinal contrast series (**b**) show a mixed axial and para-oesophageal upside-down stomach (*red arrow*) compressing the left ventricle, without any incarcerated portions of the stomach.

Conflict of interest L. Hobohm, D. Krompiec, R. Michel, Y. Yang, F. Schmidt, C. Düber, T. Münzel and P. Wenzel declare that they have no competing interest.

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Benign Paroxysmal Positional Vertigo After Nonotologic Surgery: Case Series

Leyla Kansu, Erdinc Aydin, Kamran Gulsahi

Benign paroxysmal positional vertigo is one of the most common types of vertigo caused by peripheral vestibular dysfunction. Although head trauma, migraine, long-term bed rest, Ménière disease, viral labyrinthitis, and upper respiratory tract infections are believed to be predisposing factors, most cases of benign paroxysmal positional vertigo are idiopathic. Ear surgery is another cause, but after nonotologic surgery, attacks of benign paroxysmal positional vertigo are rare. We describe three cases of benign paroxysmal positional vertigo attacks after non-otologic surgery (one patient after a nasal septoplasty and two patients after dental endodontic treatment) and discuss the pathophysiological mechanism of benign paroxysmal positional vertigo seen after non-otologic surgery, its diagnosis and treatment.

Keywords: Benign paroxysmal positional vertigo, Dental surgery, Nasal septoplasty

Introduction

Benign paroxysmal positional vertigo (BPPV) is one of the most common types of vertigo caused by peripheral vestibular dysfunction. It is characterized by short, intense vertigo episodes associated with predominantly horizontal-rotational nystagmus. They are provoked by a quick change in head position such as lying down, rolling over in bed, bending over, or looking up [1–3]. Although head trauma, migraine disease, long-term bed rest, extended travel, Ménière disease, viral labyrinthitis, vestibular neuronitis, and upper respiratory infection are believed to predisposing factors, most cases of BPPV (50 to 70%) are idiopathic [4, 5]. Ear surgery is another causative factor in BPPV [6].

The pathophysiologic mechanism of BPPV can be caused either by canalithiasis or cupulolithiasis. Canalithiasis is most commonly accepted; the endolymph system of posterior or lateral semicircular canals is disturbed by free-floating otoliths, which detach from the utricle or saccule and accumulate in the long arm of the posterior semicircular canal. It moves with gravity. This concept was first described in 1979 by Hall, Ruby, and McClure, and the phenomenon was first demonstrated *in vivo* by Parnes and McClure in 1992 [4, 7].

Conversely, Schuknecht explained the pathophysiology of the cupulolithiasis by saying that particles detached from otoconial membrane are deposited in the cupula of the posterior semicircular canal. The detached otoconial material remains free in the utricle until it enters the semicircular canal [2, 8].

We describe three cases of BPPV after non-otologic surgery (one patient after septoplasty, two patients after dental endodontic treatment) and discuss the pathophysiologic mechanism of BPPV, its diagnosis and treatment.

Clinical Cases

Case 1

A 38-year-old man came to the endodontics clinic for tooth pain. The results of his clinical and radiologic evaluations established tooth decay in his left upper first molar tooth. No other systemic disorder was found that could affect his balance. Treatment of root canals was begun. We began treatment with a low-powered surgical drill. After pulp extirpation, the canals were expanded and the tooth was filled. The surgery lasted about 40 min.

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While sitting up after surgery, the patients experienced intense vertigo with nausea, especially when he changed the position of his head. He sat in the dental chair and rested for 30 min, but the vertigo remained. The dentist referred the patient to the emergency department. The results of standard laboratory analyses, and his neurologic examination were within normal limits. His oculomotor examination was normal. No spontaneous nystagmus was observed. The Dix–Hallpike test was performed and during the left-sided swing, the patient experienced vertigo and rotatory nystagmus was observed. The nystagmus had a 4- to 7-second latency and lasted ~25 s. The Epley maneuver with mastoid oscillation was performed on the left side, and he was sent home. After the procedure, the patient was advised to avoid moving his head abruptly, to sleep in a slightly elevated position, and to avoid turning during sleep toward the affected ear for 48 h. A cervical collar or medication for BPPV was not used. After the first Epley maneuver, the patient was asked to revisit our clinic in 3 days. The results of a control Dix–Hallpike test were negative, and the patient was symptom-free.

Case 2

A 44-year-old healthy woman came to the endodontics clinic with tooth pain. After clinical and radiographic examination, tooth decay was established in right upper first and second molar tooth. Like first case, a low-powered surgical drill was used to preserve healthy tooth tissue. The surgery lasted about 35 to 40 min. After the procedure, the patient was sent home. Her husband called us after 2 or 3 h and he said that his wife had intense vertigo and nausea. The patient was referred to otolaryngology department for diagnosis and treatment of vertigo. The history of central nervous system or otological disease was negative in her past medical history. There was no systemic disease like hypertension, anemia, increased cholesterol level, coronary artery disease or diabetes mellitus. The otological symptoms as hearing loss, tinnitus or aural pressure were established. A Dix–Hallpike test was done. During the right swing, the patient had severe vertigo, and rotatory nystagmus started 4 to 6 s after and was observed for 25 to 30 s. An Epley maneuver was performed on the right side. After the procedure, the patient was advised to avoid moving her head abruptly, to sleep in a slightly elevated position, and to avoid turning during sleep toward the affected ear for 48 h. A cervical collar or medication for BPPV was not used. The results of a control Dix–Hallpike test were negative.

In both cases, a low-powered drill was used for endodontic treatment. This drill is used to keep the tooth tissue healthy, but when it touches the tooth, high vibration would be caused in the tooth and the maxilla.

Case 3

A 30-year-old female nurse came to the otolaryngology clinic with a nose obstruction since 2 to 3 years. On examination, we found she had a deviated septum and bilateral inferior turbinate hyperplasia. The patient was in good physical health. She was taking no medications. With the patient under general anesthesia, she underwent a nasal septoplasty, and radiofrequency was applied bilaterally to the inferior turbinate. The day after the operation, she had vertigo and nausea. There was no hearing loss, tinnitus or vomiting. The physical and neurological examinations were normal. No spontaneous nystagmus was noted. The results of a Dix–Hallpike test demonstrated vertigo, and torsional nystagmus was noted when she hung her head to the right. The canalith repositioning maneuver with mastoid oscillation was done on the right side. After the procedure, the patient was advised to avoid moving her head abruptly, to sleep in a slightly elevated position, and to avoid turning during sleep toward the affected ear for 48 h. A cervical collar or medication for BPPV was not used. On follow-up 3 days later, she felt better. The results of the Dix–Hallpike maneuver were normal. None of the patients re-experienced BPPV during 1-year follow-up.

Discussion

Although BPPV is usually idiopathic, cases have been discovered after traffic accidents, head trauma, otologic surgery, or other surgical interventions with prolonged bed rest. More recently, BPPV has been reported as a complication of surgical procedures involving the cochlea, such as a stapedectomy, a stapedotomy, and a cochlear implant. Atacan and associates found a 6.3%, and Magliulo and associates found an 8.5% incidence of BPPV after stapedectomy [6, 9]. During these surgical procedures, the occurence of BPPV could be explained in 2 different ways by the pathophysiological mechanisms in the literature—direct trauma or indirect trauma (vibration induced by the drill) [5]. The tip of the piston could be affected by direct trauma. The vibration of the drill on the cochlea would be sufficient to dislodge several otoconia into the labyrinth, where they could cause canalolithiasis.

The vibratory trauma affecting the cochlea during use of the drill plays a fundamental role in developing paroxysmal vertigo in patients with dental surgery. The vibrations involving the cochlea are sufficient to dislodge otoconia, as reported in the case of a dental implant, performed with the use of osteotomes [5]. In our two patients with endodontic treatment, we used the low-powered drill.

Indirect trauma on the posterior labyrinth is linked with use of either a drill or a hammer and a chisel on the maxilla.

Vibrations are propagated throughout the bone structures, eventually reaching the posterior labyrinth. At this level, mechanical energy would travel through endolymphatic fluids or bone eventually leading to macular trauma. The membranous structures of the inner ear, which are contained in bony chambers, are particularly vulnerable to traumatic lesions owing to the traveling of a mechanical wave. Even mild trauma, when caused by rotating structures whose vibrations are prolonged, can damage semicircular canals. The vibrations dislodge otoliths, which then enter canal causing BPPV [10, 11].

Dieago and associates think that use of the bone expansion technique with osteotomes in dental surgery can increase the incidence of BPPV. To prevent this, they recommended use of a surgical fraise in combination with osteotomes [2]. We used a dental drill for the surgery on our patients, but BPPV was seen.

Su and associates defended a theory that percussive force may detach otoliths from the utricle or saccule of the vestibular system in the inner ear. The posteriorly displaced otoliths may then induce BPPV. During surgical positioning of the patient face up with his head hyper-extended may facilitate displacement of the detached otoliths into the posterior semicircular canal [7, 11].

Kaplan and associates thought that the use of an osteotome during rhinoplasty was sufficient to dislodge otoconia and produce BPPV [8]. The presumed cause of BPPV in our third case was blunt head trauma caused by the osteotomy. Also, tilting the head, particularly in patients having a nasal septoplasty or in those who need to be intubated during general anesthesia, may cause BPPV [10, 11].

The diagnosis of BPPV is easily made by the Dix–Hallpike test, which produces vertigo and nystagmus after the patient is rapidly moved from a sitting to head-hanging position [11]. Patients with BPPV experience vertigo when moved rapidly into a supine position with the head turned, so that the affected ear is 30 to 45º below the horizontal plane. Vertigo occurs 1 to 40 s after the patient has been placed in such a position. The patient also develops a characteristic nystagmus, with the eyes directed toward the affected side. The vertigo and nystagmus disappear in 30 to 60 s [11].

Although BPPV is a self-limiting disorder and commonly resolves within a couple of months, the symptoms are unpleasant. Treatment consists fundamentally of maneuvers to restore the calcium carbonate crystals from the anomalous location in the semicircular canal to their correct place in the utricle. Advocated treatments are maneuvers of canalith repositioning. The Epley maneuver is the most common. Here, the patient is seated with the operator behind. The head is placed over the end of the table and turned to affected ear 45º. While the head is tilted downward, it is rotated 45º to the unaffected ear. The head and body are rotated until they face downward 135º from a supine position. While the head is turned to the unaffected side, the patient is brought to a sitting position. The head is turned forward, with the chin down 20º. This maneuver is repeated as necessary at weekly intervals until the vertigo symptoms have cleared and Dix– Hallpike maneuver is negative [12].

Dentists and surgeons must bear in mind that after dental treatment and surgical procedure, an episode of BPPV may occur.

Acknowledgments Conflicts of interest The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

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Bilateral Sudden Sensorineural Deafness with Vertigo as the Sole Presenting Symptoms of Diabetes Mellitus - A Case Report

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This paper reports a late uncontrolled diabetic presenting to an otolaryngologist with sudden severe sensorineural hearing loss of immediate origin with vertigo and tinnitus as the symptoms. Appropriate investigative and treatment measure resulted in deterioration of hearing in the right ear and mild improvement of hearing in the left ear, with no recovery of imbalance.

Keywords: Sudden severe sensorineural hearing loss, Vertigo, Tinnitus, Diabetes mellitus (NIDDM)

Case History

A 55-year-old engineer was admitted to CSJMM University, upgraded KGM University, Lucknow, as an emergency with sudden loss of hearing in both ears with vertigo and tinnitus. He noticed a very brief sound in his right ear lasting about a second. He then became completely deaf in both ears and complained of vertigo and high pitched tinnitus. There was no history of facial weakness or discharge from his ears. There was no history of external trauma or sudden straining prior to the onset. There was no history suggestive of a viral infection. There was no family history of deafness. He was a known late diabetic and a hypertensive. There was no other significant past medical history. He was not on any regular medication and described himself as perfectly healthy prior to admission. The hearing in both his ears was known to be normal 12 months before this recent episode.

General physical examination revealed no abnormality in any system. Cardiovascular in central nervous system were normal. ENT examination was normal apart from tuning fork tests which indicated a sensorineural hearing loss in both ears.

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Hematological investigations including total blood count, ESR, urea and electrolytes, blood glycosylated HbA_{1C} and serology for syphilis were done. An audiogram revealed a 110 dB sensorineural hearing loss at 4 KHz. A 55 dB sensorineural hearing loss at 2 KHz and a 45 dB sensorineural hearing loss at 1 KHz in the left ear. It also revealed a 65 dB sensorineural hearing loss at 4 KHz. A 70 dB sensorineural hearing loss at 2 KHz and a 50 dB sensorineural hearing loss at 1 KHz in the right ear. Test for recruitment and tone decay indicated a cochlear loss. Blood glycosylated Hb was 12% without ketonuria. The other investigations mentioned above were with in normal limits. The patient was put on insulin drip and anti-hypertensive treatment was begun. Eight days later there was impairment showing deterioration of hearing in the right ear and mild improvement of hearing in the left ear (Figs. 1 and 2). Vestibular tests revealed left beating spontaneous nystagmus present. On performing Binaural Bithermal Caloric test – Cawthorne Hallpike Fitzgerald test: Left beating nystagmus was present. Abnormal canal paresis was found.

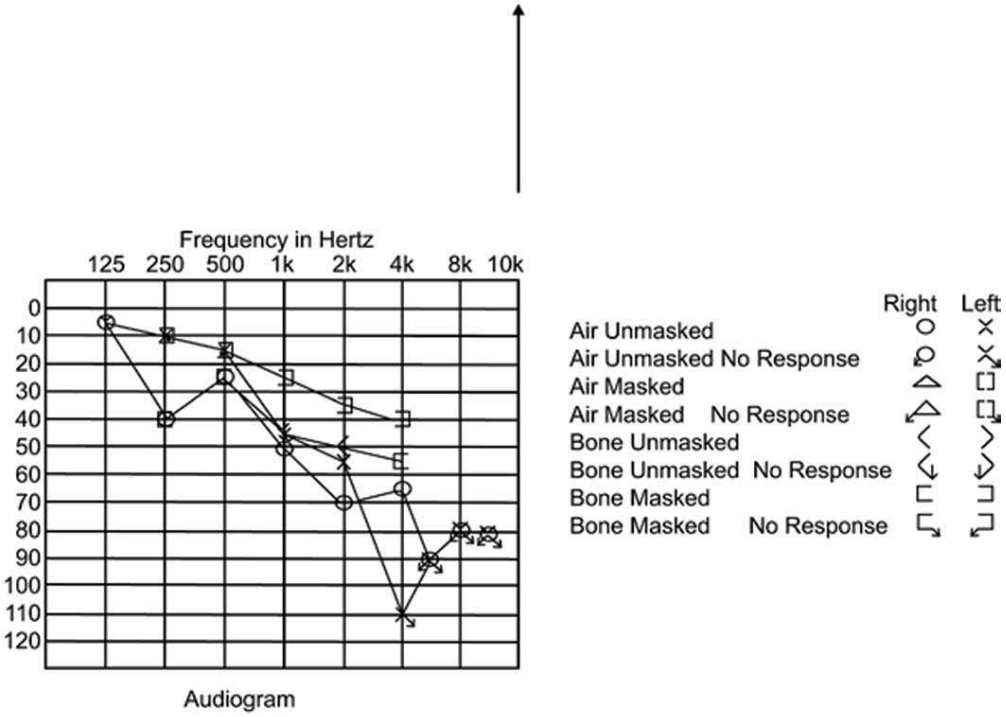
CP (n) = + 17% (right)

CP (d) = + 17% (right)

Abnormal directional preponderance was found.

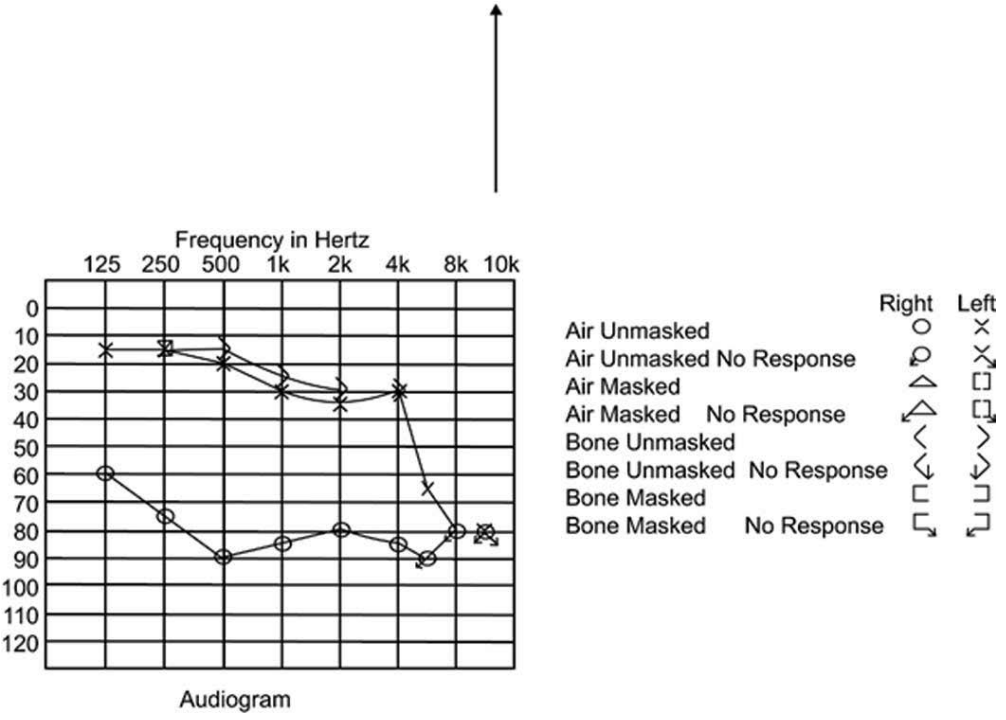
DP = + 30% (right)

On performing Unterberger's test cranio corporography angular rotation = 110° right, was extrapolated. Angular deviation = 80° right was extrapolated. Significant right sway was seen on performing Babinski's heel to toe stepping test. Audio vestibular tests therefore suggest a hemorrhagic labyrinthopathy in the



Remarks :
B/L DOWN SLOPING CURVE B/L S.N.H.L.

Fig. 1: Audiometry tracing of patient presenting with SSNHL B/L-severe.



Remarks :
B/L DOWN SLOPING CURVE
RT-A/C PROFOUND HEARING LOSS
B/C HEARING THROUGH LT SIDE
L.T-MODERATE S.N.H.L.

Fig. 2: Audiometry tracing of patient presenting with B/L sudden onset hearing impairment 8 days after previous tracing showing deterioration of hearing loss of the right ear.

right ear. There was no need to dissect the right ear as when the patient was discharged he was Hale and Harty. Slight improvement in hearing in the left ear after 8 days of therapy suggested control of hypertension and diabetes.

Discussion

Sudden sensorineural hearing loss (SSNHL) is defined as a deterioration of more than 35 dB. In at least three different frequencies occurring with in 3 days, with allowance for a longer period of onset if the loss is more severe [1]. There are many causes of SSNHL. These include trauma and labyrinthine membrane rupture, viral and bacterial infections, vascular lesions, immune complex diseases and acoustic neuroma. Diabetes and other metabolic disorders are also well known causes of sensorineural hearing loss.

In our patient the hearing deteriorated at all frequencies after 8 days of therapy in the right ear. In the literature, there are several papers which investigated the relationship between diabetes and hearing loss and vertigo. There is no reported case of any patient presenting with bilateral sudden deafness with vertigo and tinnitus as the only symptoms of diabetes mellitus. Some authors found significant hearing loss in low and middle frequencies in diabetes compared to controls [2–3]. Others found the hearing loss to be significant in high frequencies [4–5].

Audiological tests in our patient showed the hearing loss to be cochlear. The site of lesion in diabetes is thought to be cochlear [6–7], retrocochlear [8–9] or both.

Most of the available experimental and clinical evidence suggests that the complications of diabetes mellitus are a consequence of the metabolic derangements, mainly hyperglycemia [10]. Hyperglycemia can cause complications by two important mechanisms:

- Non-enzymatic glycosylation in which glucose chemically attaches to the amino-groups of proteins without the aid of enzymes and after a series of chemical rearrangements, forms irreversible advanced glycosylation end products (AGE). In capillaries for example, plasma proteins such as albumin bind to the glycosylated basement membrane accounting for the increased basement membrane thickening of diabetic microangiopathy. AGEs can also bind to receptors on many cell types resulting in a variety of biological activities including increased procoagulant activity on endothelial cells and enhanced proliferation of fibroblasts and smooth muscle cells.
- Intracellular hyperglycemia occurs in some tissues (e.g., nerve, lens, blood vessels and kidney) that do not require insulin for glucose transport. This mechanism may be responsible for damage to Schwann cells and to pericytes of capillaries with resultant neuropathy and microaneurysms resulting in hemorrhage as is suggested in this patients right membranous labyrinthopathy.

Platelets from diabetic patients show an exaggerated tendency to aggregate, perhaps mediated by altered prostaglandin metabolism. Plasma and whole blood viscosity are increased whereas red blood cell deformability is decreased. All these defects may cause stasis in the microvasculature, leading to increased intravascular pressure and to tissue hypoxia [11] again predisposing to hemorrhage as is suggested in this patients' right membranous labyrinthopathy.

In diabetes, fat may be released from tissues to enter the blood stream and cause fat embolism [12].

Mechanism of Simultaneous Bilateral Cochlear Hearing Loss in this Particular Patient

Neuropathy [8], angiopathy [9] and a combination of both may be the underlying pathology of hearing loss in diabetes. Disturbances in the microcirculation of the cochlear end vessels may be prominent aetiological factor [4]. Ischemia of the VIIIth nerve secondary to involvement of small intraneural vessels may be the aetiology in some diabetic patients [9] Wackym and Linthicum [13] suggest that vascular thickening found around the endolymphatic sac may cause accumulation of toxic waste products in endolymph, which in turn could cause hair cell dysfunction.

About 25–50% of patients with SSNHL recover their hearing partially or completely without any treatment. In the patient under report there was a probable hemorrhagic labyrinthopathy in the right ear, hearing deteriorated inspite of therapy. Since hyperglycemia is the main cause for reversible or irreversible complications, insulin therapy presumably played a major role in partial recovery of hearing in the patient's left cochlea.

This patient presented immediately after the onset of bilateral sudden onset hearing loss with vertigo and tinnitus. It appears to be advisable to hospitalize all such patients of diabetes.

The diagnosis is NIDDM/Type 2 diabetes with cochleopathy left ear and labyrinthopathy right ear.

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Isolated Transient Vertigo: Posterior Circulation Ischemia or Benign Origin?

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Background: Isolated transient vertigo can be the only symptom of posterior circulation ischemia. Thus, it is important to differentiate isolated vertigo of a cerebrovascular origin from that of more benign origins, as patients with cerebral ischemia have a much higher risk for future stroke than do those with 'peripheral' vertigo. The current study aims to identify risk factors for cerebrovascular origin of isolated transient vertigo, and for future cerebrovascular events.

Methods: From the files of 339 outpatients with isolated transient vertigo we extracted history, clinical and technical findings, diagnosis, and follow-up information on subsequent stroke or transient ischemic attack (TIA). Risk factors were analyzed using multivariate regression models (logistic or Cox) and reconfirmed in univariate analyses.

Results: On first presentation, 48 (14.2%) patients received the diagnosis 'probable or definite cerebrovascular vertigo'. During follow-up, 41 patients suffered stroke or TIA (event rate 7.9 per 100 person years, 95% confidence interval [CI] 5.5–10.4), 26 in the posterior circulation (event rate 4.8 per 100 person years, 95% CI 3.0–6.7). The diagnosis was not associated with follow-up cerebrovascular events. In multivariate models testing multiple potential determinants, only the presentation mode was consistently associated with the diagnosis and stroke risk: patients who presented because of vertigo (rather than reporting vertigo when they presented for other reasons) had a significantly higher risk for future stroke or TIA ($p = 0.028$, event rate 13.4 vs. 5.4 per 100 person years) and for future posterior circulation stroke or TIA ($p = 0.044$, event rate 7.8 vs. 3.5 per 100 person years).

Conclusions: We here report for the first time follow-up stroke rates in patients with transient isolated vertigo. In such patients, the identification of those with cerebrovascular origin remains difficult, and presentation mode was found to be the only consistent risk factor. Confirmation in an independent prospective sample is needed.

Keywords: Cerebrovascular, Stroke, Transient, Transient ischemic attack, Vertigo

Background

Vertigo is a frequent reason for emergency presentation [1–5] with manifold causes [3, 4, 6–8]. If focal neurological signs or symptoms occur, transient ischemic attack (TIA) may be diagnosed; otherwise cerebral ischemia seems unlikely [6–8]. In unselected samples, (proven) cerebrovascular cause in isolated vertigo is rare [9] and risk for future stroke is low [10], however higher than in other emergency patients [11], in particular when vascular risk factors (VRFs) are present [11]. In the last decade, increasing evidence has been put forward to show that posterior circulation ischemia can present with isolated vertigo without focal signs [9, 12]. Even more disconcerting are findings from the OxVasc study: 22% of posterior circulation stroke patients reported subtle transient neurological symptoms in the 90 days preceding their stroke, most frequently vertigo [13]. To preclude future strokes it is crucial to identify those patients whose vertigo episode was a subtle TIA.

For the clinician, there are two typical situations: the patient with acute onset, ongoing vertigo, and the patient free of symptoms on presentation, who reports (often multiple) transient

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episodes of vertigo. On a population basis, the lifetime prevalence of the latter is at least as high as that of the former [5]. The former patient is usually seen in a hospital emergency department, whereas the latter may consult a practitioner or an outpatient clinic. In symptomatic emergency patients we have the opportunity to notice subtle clinical (mainly neuro-ophthalmological) findings that may give important clues, as reflected by the HINTS algorithm [14], and – if stroke is likely – acute magnetic resonance imaging (MRI) including diffusion-weighted imaging (DWI) can often prove, but rarely exclude, this diagnosis [15–21]. In outpatients with recurrent transient vertigo, neuro-ophthalmological findings during the attack usually resolve unseen and other information may be lost or biased by recall. DWI may help only when residual microinfarctions persist; mostly MRI is negative in this situation [17].

The identification of patients with cerebrovascular cause of recurrent transient isolated vertigo remains an unsolved clinical problem. The purpose of the current study was to find determinants of cerebrovascular origin, find risk factors for future stroke, and – if possible - construct a predictive model to help guide the diagnosis in patients who presented with isolated transient vertigo in a tertiary cerebrovascular outpatient unit.

Methods

We screened the patient files of our department's neurovascular clinic (1 July 2007 to 30 June 2014) for adults who reported isolated vertigo without focal neurological symptoms (dysarthria, focal weakness, sensory symptoms, limb ataxia, diplopia and hemianopia; unsteady gait was allowed). We extracted history, clinical findings, and technical results. The diagnosis (made by a neurologist in training (third year or later) and a consultant with neurovascular specialization) in the initial report was categorized as 'definite', 'probable' or 'improbable cerebrovascular vertigo'.

Follow-up information on subsequent stroke or TIA was obtained from the files and a telephone interview. For each endpoint, the observation time was censored at the time of an endpoint event (whereas follow-up was continued for other endpoints), or at the end of a patient's follow-up.

Associations between clinical and technical variables, diagnosis and follow-up events were addressed with multivariate logistic and Cox regression models, and confirmed with the Kaplan-Meier log rank test and univariate logistic and Cox regression. To limit the number of statistical tests, a stepwise analysis strategy was defined a priori. Although originally planned, we did not attempt to construct a predictive model, as we found no consistent risk factors. All statistical calculations were made with SPSS® (IBM Inc., Armonk, NY, USA).

Results

From the electronic files of 4714 outpatient contacts, we identified 1355 patient contacts with the relevant keywords and 339 eligible patients (Table 1); 183 (54%) patients had two or more modifiable risk factors and 215 (63%) had coronary, cerebral or peripheral atherosclerosis.

On initial contact 187 (55.2%) patients came to see a neurologist because of vertigo, 152 for other reasons (mainly routine follow-up of known cerebrovascular disease), but reported transient isolated vertigo when asked about new symptoms. Most vertigo episodes lasted less than a minute, details are summarized in Table 2. Neurological examination was unremarkable in 193 (56.9%) patients. Gait ataxia was present in 88 patients (26.0%, prevalent conditions excluded). In 112 patients, the Epley manoeuvre was performed on presentation and was suggestive of benign paroxysmal positional vertigo (BPPV) in 20 (17.9%) patients. Head impulse test was never documented, presumably because the vertigo had subsided by the time of presentation in all cases. Caloric vestibular testing was done in only 15 cases and showed under-excitability in three cases (all diagnosed as vestibular neuritis), and over-excitability in one case (diagnosed

Table 1: Baseline characteristics of patients.

	Total population (*n* = 339)
Age, years (mean ± SD)	63.4 ± 12.7
Female gender, n (%)	143 (42.2)
Male gender, n (%)	196 (57.8)
Hypertension, n (%)	247 (72.9)
Diabetes, n (%)	74 (21.8)
Smoking, n (%)	77 (22.7)
Hypercholesterolemia, n (%)	125 (36.9)
Hypertriglyceridemia, n (%)	35 (10.3)
Number of modifiable VRFs, #: n (%)	
(Including hypertension, diabetes, smoking, hypercholesterolemia, and hypertriglyceridemia)	0: 49 (14.5) 1: 107 (31.6) 2: 123 (36.3) 3: 37 (10.9) 4: 21 (6.2) 5: 2 (0.6)
Coronary heart disease (CHD), n (%)	65 (19.2)
Peripheral arterial occlusive disease (AOD), n (%)	36 (10.6)
Prevalent Stroke or TIA (CVD), n (%)	177 (52.2)
Stroke or TIA in the last 6 months, n (%)	38 (11.2)
At least one vessel disease (CHD, AOD or CVD), n (%)	215 (63.4)

Table 2: Properties of vertigo.

	Total population (*n* = 339)
Type of vertigo[a]	
Illusion of rotational movement, n (%)	120 (35.4)
Illusion of swaying movement, n (%)	132 (38.9)
Unclassifiable, n (%)	101 (29.8)
Vertigo frequency	
Median, range (n/week)	5/week, 1/week – 35/week
Below median, n (%)	40 (35.6 of noted)
Median or higher, n (%)	73 (64.4 of noted)
Not noted, n (%)	226 (66.7)
Vertigo duration	
Median, range (seconds)	< 60s, 1 s – 10.800 s
Below median, n (%)	84 (56.4 of noted)
Median or higher, n (%)	65 (43.6 of noted)
Not noted, n (%)	190 (56.0)
Vertigo trigger[a]	
Spontaneous, n (%)	200 (59.0)
Turning the head, n (%)	61 (18.0)
Other change in body position, n (%)	123 (36.3)
Orthostatic stress, n (%)	37 (10.9)
Blood pressure-lowering situation, n (%)	40 (11.8)

[a]Classification not exclusive as some patients reported multiple types

as benign positional vertigo). Due to the high proportion of missing information, we did not attempt statistical calculations on caloric testing. Menière's disease and labyrinthitis were not diagnosed in this cohort.

Brain imaging included cranial computed tomography (CT) in 66 (19.5%) and in 150 (44.2%) patients; CT or MRI showed definite new cerebral infarction in eight (2.4%) patients. Vessel imaging included duplex sonography in 327 (96.4%), CT angiography in ten (2.9%), MR angiography in 57 (16.8%) and digital subtraction angiography (DSA) in 14 (4.1%) patients. Vessel findings (Table 3) were assembled from all available information.

Diagnosis and Follow-Up

In the medical report from the initial contact, vertigo was considered as 'definitely cerebrovascular' in 19 (5.6%) patients and 'definitely or probably cerebrovascular' in 48 (14.2%) patients.

In 214 (63.1%) patients our files included at least one further presentation after the initial contact. The remaining 125 patients were contacted and 26 did not respond or refused to participate in the survey. Ninety-nine patients gave their consent and provided follow-up information (response rate 79.2%). Overall, we obtained follow-up information from 289 of 339 patients (85.3%, follow-up period 3 days to 7.7 years, 563 person years). The number of endpoint events and the resulting event rates are shown in Table 4.

Determinants of the Clinical Diagnosis

Age was positively associated with a higher risk for the diagnosis 'definite or probable cerebrovascular vertigo' (multivariate and univariate $p = 0.024$). Presentation because of vertigo was positively related with the diagnosis 'definite cerebrovascular vertigo' (multivariate $p = 0.013$; univariate $p = 0.014$). Patients with fewer than five vertigo attacks per week were more likely to be diagnosed with 'definite or probable cerebrovascular vertigo' (multivariate and univariate $p < 0.001$). Patients with bilateral vertebral stenosis or basilar stenosis and patients with any stenosis were more likely to be diagnosed with 'definite or probable cerebrovascular vertigo' (multivariate $p = 0.047$ and $p = 0.048$; univariate $p < 0.001$ each).

Determinants of Future Cerebrovascular Events

In patients who presented because of vertigo, the future risk for stroke or TIA was significantly higher than in patients who presented for other reasons (multivariate $p = 0.028$, univariate $p = 0.005$, adjusted HR 2.07 [1.11–3.84]; Fig. 1). When tested for the other endpoints (univariate), the reason for presentation was seen to be a significant determinator of stroke or TIA in the

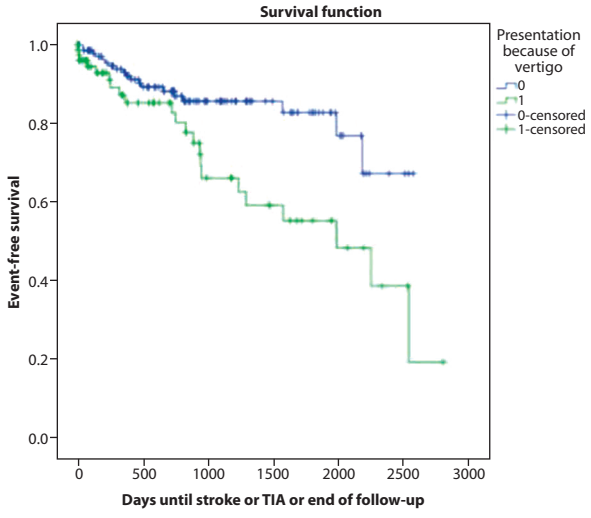

Fig. 1: Kaplan-Meier plot of the endpoint 'any stroke or TIA', stratified by presentation mode. Event rates (presentation because of vertigo (1) vs. other reasons (0)): 13.4 (95% CI 7.8-19.0) vs. 5.4 per 100 person years (95% CI 3.0-7.8).

Table 3: Findings in cervical and cerebral arteries with focus on posterior circulation (*n* = 339).

	Right	Left
Subclavian artery		
Stenosis, n (%)	23 (6.8)	37 (10.9)
No stenosis, n (%)	198 (58.4)	184 (54.3)
Not examined, n (%)	118 (34.8)	118 (34.8)
Vertebral artery V0-V4		
Stenosis ≥50%, n (%)	76 (22.4)	71 (20.9)
Stenosis <50%, n (%)	26 (7.7)	15 (4.4)
No stenosis, n (%)	225 (66.4)	241 (71.1)
Not examined, n (%)	12 (3.5)	12 (3.5)
Vertebral artery V0-V1		
Stenosis ≥50%, n (%)	41 (12.1)	39 (11.5)
Stenosis <50%, n (%)	18 (5.3)	7 (2.1)
No stenosis, n (%)	265 (78.2)	281 (82.9)
Not examined, n (%)	15 (4.4)	12 (3.5)
Vertebral artery V2		
Stenosis ≥50%, n (%)	22 (6.5)	24 (7.1)
Stenosis <50%, n (%)	3 (0.9)	4 (1.2)
No stenosis, n (%)	302 (89.1)	299 (88.2)
Not examined, n (%)	12 (3.5)	12 (3.5)
Vertebral artery V3-V4		
Stenosis ≥50%, n (%)	42 (12.4)	37 (10.9)
Stenosis <50%, n (%)	9 (2.7)	7 (2.1)
No stenosis, n (%)	274 (80.8)	282 (83.2)
Not examined, n (%)	14 (4.1)	13 (3.8)
Basilar artery		
Stenosis ≥50%, n (%)	27 (8.0)	
Stenosis <50%, n (%)	16 (4.7)	
No stenosis, n (%)	283 (83.5)	
Other findings		
Basilar head aneurysm, n (%)	2 (0.6)	
Megadolichobasilaris, n (%)	1 (0.3)	
Not examined, n (%)	13 (3.8)	
Internal carotid		
Stenosis ≥70% (NASCET), n (%)	32 (9.4)	28 (8.3)
Stenosis <70%, n (%)	30 (8.8)	50 (14.7)
No stenosis, n (%)	265 (78.2)	248 (73.2)
Other finding		
Pseudoaneurysm, n (%)	0 (0.0)	1 (0.3)
Not examined, n (%)	12 (3.5)	13 (3.8)
Intracranial artery[a]		
Stenosis ≥50%, n (%)	4 (1.2)	
No stenosis or <50%, n (%)	324 (95.6)	
Other findings		
Right PICA occlusion, n (%)	1 (0.3)	
Untreated right MCA aneurysm, n (%)	1 (0.3)	
Coiled AcomA aneurysm, n (%)	1 (0.3)	
Not examined, n (%)	11 (3.2)	
Any vertebral or basilar artery		
Stenosis ≥50%, n (%)		119 (35.1)
No stenosis or <50%, n (%)		209 (61.7)
Not examined, n (%)		11 (3.2)
Bilateral vertebral or basilar artery		
Stenosis ≥50%, n (%)		57 (16.8)
No stenosis or <50%, n (%)		271 (79.9)
Not examined, n (%)		11 (3.2)
Any pathologic finding (any degree)b		
Pathologic finding, n (%)		205 (60.5)
Normal, n (%)		123 (36.3)
Not examined, n (%)		11 (3.2)
Any stenosis ≥ 50%[b]		
Stenosis ≥50%, n (%)		191 (56.3)
No stenosis or <50%, n (%)		137 (40.4)
Not examined, n (%)		11 (3.2)

[a]Other than basilar artery
[b]Extra- or intracranial

Table 4: Stroke endpoints during follow-up.

	Number of events	Event rate per 100 person years (95% CI)
Any Stroke	22	4.0 (2.3–5.6)
Posterior circulation stroke	12	2.1 (0.9–3.3)
Any stroke or TIA	41	7.9 (5.5–10.4)
Posterior circulation stroke or TIA	26	4.8 (3.0–6.7)

posterior circulation (p = 0.044, event rate 7.8 vs. 3.5 per 100 person years), but not of 'any stroke' or 'posterior circulation stroke'.

Patients whose vertigo was provoked by a change in body position had a smaller risk for posterior circulation stroke or TIA (multivariate p = 0.037; univariate p = 0.017; event rates 1.9 [95% CI 0.0–3.8] vs. 5.4 per 100 person years [3.9–9.4]). Patients with any stenosis were less likely to suffer stroke (multivariate p = 0.032; univariate p = 0.013, event rate 2.6 vs. 7.5 per 100 person years). The diagnosis was not associated with the risk for future cerebrovascular events (tested in eight univariate Cox regression models, all p > 0.3).

Discussion

Aim of the present study was to find determinants to identify those with subtle TIAs among patients with isolated transient vertigo, and to identify risk factors for future stroke or TIA during follow-up. Our sample of 339 persons was a high-risk population with multiple VRFs and a high proportion of vascular organ damage. Usually, patients are referred to this unit because the referring physician believes there is a problem with the cervical or cerebral arteries.

A somewhat comparable population may be patients presenting with acute onset vertigo, although there are important differences: their vertigo episode may be singular (all recurrent in our cohort), possibly more severe (as acute patients called an ambulance immediately, whereas our patients consulted their physician days or weeks later), and presumably longer (in acute patients, the symptoms are mostly still present on arrival at the hospital emergency department).

In comparison with the literature data on acute vertigo, absolute follow-up stroke rates were higher in our cohort (any stroke in 4% of the cohort per year) as compared to 1.4% per year in patients who presented with 'non-stroke dizziness' to an emergency department in Texas, U.S.A. [10], and 1.7% per year in patients hospitalized for vertigo in Taiwan [11]. This discrepancy may be largely explained by the high risk factor load in our cohort: a subgroup of Taiwanese patients hospitalized for vertigo with the most VRFs had a 3.5% annual stroke rate [11], which is very similar to our results. The proportion of patients with stroke as the cause of the initial episode was 0.7% in the Texas cohort [9], whereas in our cohort stroke or TIA was the definite cause in 5.6% and the probable or definite cause of vertigo in 14.2% of our cohort. Again, the main reason for this discrepancy is most likely the large number of VRFs among our patients.

In the search for determinants of the diagnosis *cerebrovascular vertigo*, we identified age and precerebral artery stenosis, which were both expected. Unexpectedly, both these determinants were inconsistent for the stroke endpoints: here age was not identified at all as a predictor, and stenosis of any cervical or cerebral vessel was even negatively associated with future stroke. For the diagnosis on initial presentation, these two 'obvious' risk factors may have influenced the neurologist's judgment. The frequency of vertigo attacks was predictive for the diagnosis 'definite or probable cerebrovascular vertigo', where lower frequency was associated with cerebrovascular origin. As many uniform episodes are difficult to explain as TIAs, this factor may also have influenced the judgment of the diagnosing neurologist.

'Reason for presentation' was the only determinant that was consistently and significantly associated with the diagnosis 'cerebrovascular vertigo' and with future posterior circulation stroke and TIA. It is possible that vertigo, which is due to a subtle brainstem or cerebellar TIA, may be more intense or impressive than vertigo of other causes, thus persuading the patient or his primary care physician to make an urgent clinic appointment.

A very interesting influential factor was identified only in the endpoint analysis: the provocation factor 'changes in body position', which was associated with lower stroke risk. This association may tell us that in our cohort peripheral positional vertigo (for example BPPV or benign disabling vertigo [22]) may be more frequent than (ischemic) central positional vertigo (e.g. pseudo-BPPV in vermis stroke/TIA [23]). The lack of an association between 'head rotation' as a trigger of vertigo and future stroke can mean either that vertigo caused by functional vertebral artery compression [22] is rare in our patients or – if it occurs – that it rarely causes stroke.

The most surprising result of our work is that the judgment of the vascular neurologist was not correlated with future stroke risk. At first glance, this challenges our view of the world: are our sophisticated pathophysiological considerations out of sync with reality? A more comfortable explanation may be that the originally elevated stroke risk for patients correctly classified as 'subtle vertigo TIA' was counteracted by the risk factor management we subsequently recommended. Such a hypothetical treatment bias might even explain the inverse association between 'any stenosis' and future stroke, as escalations of risk factor management (e.g. tightening low-density lipoprotein cholesterol [LDLc] goals) may be triggered by finding stenoses.

What can we learn from these data for the management of our patients? First, we must be careful not to overinterpret these results, as they are explorative and require external validation. Given our sparse and somewhat inconsistent findings, we refrained from constructing an originally planned predictive model. On the basis of the risk factor 'reason for presentation' we may consider doing an MRI in patients with isolated transient vertigo, who are worried enough to see the doctor because of this symptom. A clinical benefit of this measure has yet to be proven. Replication of this study in a prospective design may yield the necessary information for constructing a predictive model and developing a refined clinical algorithm.

Limitations

Some limitations of this study arise from the retrospective design. For example, some important variables characterizing the vertigo (frequency and duration) were incompletely documented (33% and 44%, respectively). A complete documentation of these variables in a prospective setting may yield interesting results. Furthermore, in only 60% of the patients was a pathologic finding detected with vessel imaging, which is

unexpectedly low in such a high-risk cohort. In particular, low-grade stenoses (<50% or <70%) were relatively rare. Possibly minor vessel changes were under-reported, as the ultrasound examiner may have focused on 'relevant' high-grade findings. However, as our analysis focused on high-grade stenoses (≥50% or ≥70%), it is unlikely that this caused relevant bias.

Importantly, the nature of our analyses was explorative, as many determinants were tested for multiple dependent variables. Despite provisions to reduce the number of statistical tests (see Methods section), 15 determinants were tested for six dependent variables, resulting in 90 primary tests. A Bonferroni correction would have reduced the *p* value to 0.0005, which is an unlikely *p* value in multivariate models in a cohort of this size. Therefore, all results have to be interpreted with caution.

Conclusions

We here reported for the first time follow-up stroke rates in patients with isolated transient vertigo. Identifying patients with cerebrovascular vertigo remains difficult. Presentation mode (patients who presented because of vertigo) was found to be the only consistent risk factor for cerebrovascular vertigo and future risk for stroke or TIA. However, the clinical benefit of this finding may be limited. Confirmation in an independent prospective sample is needed.

Abbreviations
BPPV: Benign paroxysmal positional vertigo; CI: Confidence interval; CT: Computed tomography; DSA: Digital subtraction angiography; DWI: Diffusion-weighted imaging; LDLc: Low-density lipoprotein cholesterol; MRI: Magnetic resonance imaging; TIA: Transient ischemic attack; VRFs: Vascular risk factors

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Availability of data and materials
The datasets used and analyzed in the current study are available from the corresponding author on reasonable request.

Authors' contributions
CH participated in the design of the study and collected and reviewed data. LW collected data and performed statistical analysis. TB collected data, performed statistical analysis, interpreted data and drafted the manuscript. ML designed the study, performed statistical analysis, interpreted data, drafted and critically revised the manuscript. All authors read and approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Consent for publication
Not applicable.

Ethics approval and consent to participate
The study was conducted according to the principles of the Declaration of Helsinki, and approved by the ethics committee of the Faculty of Medicine, Goethe-University Frankfurt (Application No. 237/14).

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Notes