Published in final edited form as:

J Vestib Res. 2016 January 28; 25(5-6): 233–239. doi:10.3233/VES-150563.

Retrospective data suggests that the higher prevalence of benign paroxysmal positional vertigo in Individuals with type 2 diabetes is mediated by hypertension

Linda J. D'Silva, PT,

Department of Physical Therapy and Rehabilitation Science, University of Kansas Medical Center, 3901 Rainbow Blvd, Kansas City, KS 66160. USA. Phone number: (913)-945-6630; Fax number (913)-588-9428; ldsilvakumc.edu

Hinrich Staecker, MD, PhD,

Department of Otolaryngology-Head and Neck Surgery, University of Kansas Medical Center, Kansas City, KS 66160

James Lin, MD,

Department of Otolaryngology-Head and Neck Surgery, University of Kansas Medical Center, Kansas City, KS 66160

Kevin J. Sykes, PhD,

Department of Otolaryngology-Head and Neck Surgery, University of Kansas Medical Center, Kansas City, KS 66160

Milind A. Phadnis, PhD,

Department of Biostatistics, University of Kansas Medical Center, Kansas City, KS 66160

Tamara M. McMahon, MS,

Division of Medical Informatics, University of Kansas Medical Center, Kansas City, KS 66160

Dan Connolly, BS,

Division of Medical Informatics, University of Kansas Medical Center, Kansas City, KS 66160

Carla H. Sabus, PT, PhD,

Department of Physical Therapy and Rehabilitation Science, University of Kansas Medical Center, Kansas City, KS 66160

Susan L. Whitney, PT, PhD, and

University of Pittsburgh, School of Health and Rehabilitation Sciences, Pittsburgh, PA 15260; Secondary affiliation, Rehabilitation Research Chair, Department of Rehabilitation Sciences, King Saud University, Riyadh, Saudi Arabia

Patricia M. Kluding, PT, PhD

Department of Physical Therapy and Rehabilitation Science, University of Kansas Medical Center, Kansas City, KS 66160

Abstract

Correspondence to: Linda J. D'Silva.

Objective—Benign Paroxysmal Positional Vertigo (BPPV) has been linked to comorbidities like diabetes and hypertension. However, the relationship between type 2 diabetes (DM) and BPPV is unclear. The purpose of this retrospective study was to examine the relationship between DM and BPPV in the presence of known contributors like age, gender and hypertension.

Methods—A retrospective review of the records of 3933 individuals was categorized by the specific vestibular diagnosis and for the presence of type 2 DM and hypertension. As the prevalence of BPPV was higher in people with type 2 DM compared to those without DM, multivariable logistic regressions were used to identify variables predictive of BPPV. The relationship between type 2 DM, hypertension and BPPV was analyzed using mediation analysis.

Results—BPPV was seen in 46% of individuals with type 2 DM, compared to 37% of individuals without DM ($p < 0.001$). Forty two percent of the association between type 2 DM and BPPV was mediated by hypertension, and supported hypertension as a complete mediator in the relationship between type 2 DM and BPPV.

Conclusions—Hypertension may provide the mediating pathway by which diabetes affects the vestibular system. Individuals with complaints of dizziness, with comorbidities including hypertension and diabetes, may benefit from a screening for BPPV.

Keywords

Type 2 Diabetes; hypertension; benign paroxysmal positional vertigo

Introduction

Benign paroxysmal positional vertigo (BPPV) is a common vestibular disorder, affecting about 1.6% of the adult population per year [33]. The main symptom of BPPV is vertigo which is position dependent and transient [5]. Patients typically develop vertigo when looking overhead, getting in or out of bed, and bending. When symptomatic, 86% of individuals with BPPV have been shown to restrict daily activities, while 18% avoided leaving their homes [33]. Fortunately, BPPV is easily treated with appropriate repositioning maneuvers, which are very effective [12, 18, 27].

Known factors associated with a higher prevalence of BPPV include age, female sex, history of head trauma, other inner ear disease including Meniere's disease and vestibular neuritis, osteoporosis, and migraines [3, 15, 21, 31, 33]. Vasospasm of the labyrinthine arteries may cause detachment of otoconia from the macula, seen in conditions like migraine [32]. BPPV is associated with hypertension and hyperlipidemia, due to possible vascular damage to the inner ear, and is a known sequel of labyrinthine ischemia that precipitates detachment of the otoconia [7, 33]. The relationship between diabetes (DM) and BPPV is not well understood. However, in people with type 2 DM, the risk of diabetic complications have been shown to increase significantly when systolic blood pressure is high [2]. The prevalence of hypertension is 40–60% higher in people with type 2 DM between 45–70 years of age, compared to the nondiabetic population [1]. Cohen et al. reported DM as being unusually prevalent in patients with BPPV [8], and comorbidities like hypertension and diabetes, have been shown to significantly increase the recurrence rate of BPPV [10], however Warninghoff et al. did not detect an increased prevalence of DM or hypertension in

individuals with vertigo [37]. By the year 2030, the prevalence of DM globally is estimated to rise to 366 million people [40], with type 2 DM accounting for 95% of all cases of DM diagnosed in adults [11]. It is necessary to understand the relationship between BPPV, hypertension and type 2 DM because making a diagnosis of BPPV in older adults, with multiple comorbidities is difficult [19]. In fact, the prevalence of unrecognized BPPV in elderly people with multiple chronic diseases is as high as 9% [26], which could potentially delay treatment maneuvers.

This study was designed as a retrospective, observational study that investigates the interaction between type 2 DM and vestibular disorders as they naturally occur within individuals managing chronic disease through their adult lifespan. Because animal research has shown morphological changes in the saccule of diabetic rats [24], and clinical research has shown a higher prevalence of BPPV in type 1 and type 2 DM [8, 41], our hypothesis was that in people with type 2 DM, the prevalence of BPPV may be higher. We examined the role of known contributing factors such as age, gender, and hypertension; as well as the potential mediating relationship between type 2 DM, hypertension, and BPPV.

Materials and Methods

Following Institutional Review Board approval from the University of Kansas Medical Center (KUMC), data was collected using the Healthcare Enterprise Repository for Ontological Narration (HERON), which is a medical records system. HERON is built upon the i2b2 integrated clinical data repository software [35]. This database integrates data from electronic medical records, as well as the billing system to provide de-identified medical data pieces, preserving patient privacy [23]. Using a query search in Heron, data for this study was extracted from the University of Kansas Hospital inpatient and emergency room records as well as from area outpatient clinics affiliated with KUMC, that use the electronic medical record system.

Data was obtained on individuals 18 years and older. Our population of interest was patients with a vestibular diagnosis for their complaints of dizziness (ICD9 code 386.1–386.5) between October 2006 and December 2012. Data were collected on age at the time of the vestibular diagnosis, sex, race, smoking history, and specific vestibular diagnosis. The presence or absence of type 2 diabetes and hypertension were collected within the data query, making sure to identify the date of first diagnosis of diabetes and hypertension. This was used to determine if diabetes and hypertension were present before or after the vestibular diagnosis, thus helping us classify the cases as having type 2 diabetes and hypertension or not. Our query included a population inclusive of a range of vestibular diseases with and without diabetes. All individuals with type 1 DM were excluded from the analysis.

Statistical Analysis

Chi-square tests were used to compare the frequency of specific vestibular diagnoses in people with and without type 2 DM. Descriptive statistics describe the presence of comorbidities in individuals with BPPV.

Univariate logistic regression was used to identify the variables with significant relationship to BPPV. Variables with significant relationship to BPPV were included in a multivariable logistic regression analysis. Factors included in the model were age, sex, race, presence of hypertension and diabetes, which were entered in a backward stepwise regression model.

The mediating effect of hypertension on the relationship between type 2 DM and BPPV was tested using multiple regression analyses. According to Baron and Kenney [4], mediation is demonstrated when the following relationships are observed: 1) the main independent variable (type 2 DM) is significantly associated with the main dependent variable (BPPV), 2) the independent variable (type 2 DM) is significantly associated with the mediator variable (hypertension), 3) the mediator variable (hypertension) is significantly associated with the dependent variable (BPPV) when the independent variable (type 2 DM) is controlled. All analyses were conducted at the 0.05 level of significance a priori, using SPSS 20.0 for Windows (Chicago, IL).

Results

The HERON search results identified a total of 3933 electronic medical records, of which, 12 individuals had type 1 DM and were excluded from the analysis. There were nine vertiginous conditions frequently diagnosed, BPPV was the most common among people with and without type 2 DM (n=1518). BPPV was present in 46% of the sample who had concurrent DM (n=322), and in 37% of individuals who did not have concurrent DM (n=1196), this proportion was significantly higher than expected ($p < 0.001$). (Table 1)

Descriptive Statistics of Individuals with BPPV

The mean age of people with BPPV was 59 ± 15.8 years, while those with BPPV and type 2 DM was 66 ± 12.8 years. The female to male proportion in both groups was 68% female and 32% male. Table 2 shows the frequency of hypertension and type 2 DM in people with BPPV stratified by age group. Two percent of patients (n=79) had BPPV and coexisting Meniere's disease, 1% (n=45) had BPPV and a diagnosis of central vertigo, 0.6% (n=23) had BPPV and vestibular neuronitis, while 0.9% (n=36) had BPPV and a diagnosis of labyrinthitis.

Analysis of Variables with Logistic Regression

Variables with a significant relationship to BPPV included age (independent sample t-test, $p < 0.001$), sex (chi square test, $p = 0.008$), race (chi square test, $p < 0.001$), diabetes (chi square test, $p < 0.001$) and hypertension (chi square test, $p < 0.001$). Using a backward stepwise regression model, we found that age, sex, race and hypertension were the significant variables predictive of BPPV (Table 3). The prevalence of BPPV was higher with increasing age and female gender. African-American were 1.3 times more likely to present with BPPV while Asians were 2.6 times more likely to present with BPPV compared to their Caucasian counterparts. In people with hypertension, the frequency of BPPV was 1.7 times higher; however diabetes did not influence the presence of BPPV in the multivariate analysis.

Figure 1 illustrates the mediation effect and shows that hypertension mediated the association between type 2 DM and BPPV. The effect can be expressed as $(0.39 \times 0.15) / [(0.39 \times 0.15) + 0.08] = 42.2\%$. In the presence of hypertension, the association between type 2 DM and BPPV is no longer significant, showing that hypertension is a complete mediator in the relationship between type 2 DM and BPPV. Hypertension was present in 86.8% of individuals with type 2 DM, compared to 37.2% with BPPV without type 2 DM. This relationship between type 2 DM and hypertension was seen in all age groups.

Discussion

Results of this study show that hypertension was a strong predictor of BPPV and completely mediated the effect of diabetes on BPPV. The close association between type 2 DM and hypertension is well known and they are often referred to as “the bad companions” [13]. The overall prevalence of hypertension in people with DM is as high as 71%, of which only 12% have their hypertension controlled ($<130/85$ mm of Hg) [36]. Microvascular and macrovascular complications are significantly higher in people with type 2 DM and hypertension [14]. One mechanism by which hyperglycemia increases vascular resistance is by inhibiting nitric-oxide related vasodilation, by disrupting nitric oxide signaling as well as by forming reactive oxygen species (ROS). Formation of advanced glycation end products through the polyol pathway increases formation of ROS [29], which further depletes nitric oxide. In addition, activation of the protein kinase pathway leads to cell proliferation, leading to increased stiffness of arterial walls [38]. These mechanisms are responsible for vasoconstriction of small arterioles leading to tissue ischemia. The vascular effects of hypertension and diabetes may be possible mechanisms leading to tissue hypoxia and cochleovestibular degeneration [9, 22]. Results of our study are in agreement with other studies of hypertension in vestibular dysfunction [7, 33]. Chavez-Delgado et al. report that hypertension, type 2 DM and dyslipidemia resulted in the highest percentage of cochlear and vestibular dysfunction, with hypertension present in 74% of their study population ($n=385$) [7]. Besides microvascular damage, macrovascular damage is seen in people with diabetes and hypertension. Arteriosclerotic changes were seen in the carotid arteries of 71% of people with BPPV compared to 43% of people with other vestibular disorders [34], while stenosis or occlusion of the vertebrobasilar artery was seen in 21% of people with BPPV, compared to 6.8% of people without BPPV [42]. Elderly people (mean age 57.36 ± 15.44 years) with BPPV and comorbidities like hypertension and diabetes have a higher risk of ischemic stroke (4.5% compared to 2.9% without BPPV) [17].

The effect of diabetes on the peripheral vestibular organs has been examined in animal models. Morphological changes were noted in the utricle and saccule of rats with experimentally induced diabetes [24, 25] with type 1 hair cell degeneration seen, particularly in the saccule. These changes were considered signs of metabolic stress due to hyperglycemia [24]; however the impact of diabetes on the dislodgement of otoconia has not been reported. However, in a histopathological study examining the temporal bones of patients with type 1 diabetes, Yoda et al. noted a higher prevalence of otoconia deposits in the semicircular canals as well as adhered to the cupula, compared to age matched controls [41].

We examined other known factors associated with a higher prevalence of BPPV like age, gender and race. Aging has been shown to cause changes in the protein and gelatinous matrix of the otolith membrane, as well as weakening of the linking filaments among otoconia with fragmentation of the otoconia [16]. This results in displacement of the otoconia into the semicircular canals. This study showed that the presence of BPPV increased by 10% for every advancing decade of life. Twice as many females were diagnosed with BPPV as compared to males. This is aligned with current literature showing that there is a marked female preponderance in BPPV [33]; this female preponderance may be linked, among other factors, to migraines or hormonal factors [15, 21, 31]. Estrogen deficiency has been shown to disturb the internal structure of the otoconia and their interconnection, and attachment to the matrix [20, 31]. Regarding race, we found that African-Americans were more likely to have BPPV compared to Caucasians. This finding could be because the age adjusted prevalence of diagnosed diabetes is higher in African-American individuals compared to Caucasians, and is highest in general, in African-American women [6]. Of interest, our study showed that BPPV was present in higher frequency in the Asian community. Previous research has shown a higher prevalence of DM and its complications in the Asian population [28]. In our study, the mean age in the Asian population was younger (56 ± 14 years) than the mean for the overall population, more commonly female (72%) and comorbidities included hypertension and diabetes 37.9% and 20.7% of the time respectively.

Another result of this study was that unspecified peripheral vertigo and other disorders of the labyrinth, conditions whose etiology is unclear, was seen more commonly in people without type 2 DM. It is possible that in people with comorbidities like diabetes and hypertension, referrals to specialists are higher, which may not be the case for people without DM. Referrals to specialists in tertiary care centers usually result in revised and specific diagnoses [30]. Thirty-four percent of our cases had a diagnosis of Meniere's disease. This may be because KUMC is a tertiary care center with specialists in the field of neuro-otology, resulting in a higher referral rate to this center. These are interesting observations that require further investigation.

Results of our study suggest a multifactorial and interrelated risk that may influence the presence of BPPV in people with DM. Being aware of the relationship between BPPV, type 2 DM and hypertension may benefit clinicians. Since hypertension, diabetes and BPPV can independently cause dizziness, it is important for the clinician to examine all potential causes of dizziness carefully. Asking specific questions, to determine if BPPV could be a source of the dizziness, may help with early diagnosis and treatment [39].

Limitations

One limitation of this study is the retrospective nature of data collection. Every effort was made to exclude type 1 DM and ensure that the diagnosis of diabetes and hypertension predated the vestibular diagnosis. We found that the prevalence of BPPV was 9% higher in those with type 2 DM compared to those without DM. Although this difference was statistically significant, we cannot establish if this is a clinically meaningful difference. This was an observational study hence the cause/ effect relationship between diabetes and BPPV

cannot be established. In this study, indicators of DM severity (e.g. HbA1c levels, fasting glucose) were not consistently available. Future studies examining relationships between DM, glycemic control, and severity of vertiginous symptoms will assist clinicians in the management of these patients.

Conclusion

In summary, this study shows that hypertension is the mediating factor contributing to the increased prevalence of BPPV in individuals with type 2 DM. Hypertension may cause vascular damage to the macula of the utricle and saccule, resulting in dislodgement of the otoliths. Improved understanding about the role of hypertension and DM as risk factors for the presence of BPPV can help generate new pathophysiological hypotheses that may ultimately help to improve patient care.

Acknowledgments

The authors thank Michael Tetwiler for his assistance in data collection. This work was supported by Award Number T32HD057850 from the Eunice Kennedy Shriver National Institute of Child Health & Human Development and by a CTSA grant from NCATS awarded to the University Of Kansas Medical Center for Frontiers: The Heartland Institute for Clinical and Translational Research # UL1TR000001 (formerly #UL1RR033179). REDCap at University of Kansas Medical Center is supported by CTSA grant from NCRR and NCATS awarded to the University of Kansas Medical Center for Frontiers: The Heartland Institute for Clinical and Translational Research. The contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH or NCATS.

References

1. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 38. UK Prospective Diabetes Study Group. *BMJ*. 1998; 317:703–713. [PubMed: 9732337]
2. Adler AI, Stratton IM, Neil HA, Yudkin JS, Matthews DR, Cull CA, Wright AD, Turner RC, Holman RR. Association of systolic blood pressure with macrovascular and microvascular complications of type 2 diabetes (UKPDS 36): prospective observational study. *BMJ*. 2000; 321:412–419. [PubMed: 10938049]
3. Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology*. 1987; 37:371–378. [PubMed: 3822129]
4. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*. 1986; 51:1173–1182. [PubMed: 3806354]
5. Bhattacharyya N, Baugh RF, Orvidas L, Barrs D, Bronston LJ, Cass S, Chalian AA, Desmond AL, Earll JM, Fife TD, Fuller DC, Judge JO, Mann NR, Rosenfeld RM, Schuring LT, Steiner RW, Whitney SL, Haidari J. Clinical practice guideline: benign paroxysmal positional vertigo. *Otolaryngology and Head and Neck Surgery*. 2008; 139:S47–S81.
6. CDC. National Diabetes Fact sheet. 2011
7. Chavez-Delgado ME, Vazquez-Granados I, Rosales-Cortes M, Velasco-Rodriguez V. Cochleovestibular dysfunction in patients with diabetes mellitus, hypertension and dyslipidemia. *Acta Otorrinolaringologica Espanola*. 2012; 63:93–101. [PubMed: 22153963]
8. Cohen HS, Kimball KT, Stewart MG. Benign paroxysmal positional vertigo and comorbid conditions. *ORL: Journal of Oto-Rhino-Laryngology and Its Related Specialties*. 2004; 66:11–15.
9. de Moraes Marchiori LL, de Almeida Rego Filho E, Matsuo T. Hypertension as a factor associated with hearing loss. *Brazilian Journal of Otorhinolaryngology*. 2006; 72:533–540. [PubMed: 17143434]
10. De Stefano A, Dispenza F, Suarez H, Perez-Fernandez N, Manrique-Huarte R, Ban JH, Kim MB, Strupp M, Feil K, Oliveira CA, Sampaio AL, Araujo MF, Bahmad F Jr, Gananca MM, Gananca

- FF, Dorigueto R, Lee H, Kulamarva G, Mathur N, Di Giovanni P, Petrucci AG, Staniscia T, Citraro L, Croce A. A multicenter observational study on the role of comorbidities in the recurrent episodes of benign paroxysmal positional vertigo. *Auris, Nasus, Larynx*. 2013
11. Deshpande AD, Harris-Hayes M, Schootman M. Epidemiology of Diabetes and Diabetes-Related Complications. *Physical Therapy*. 2008; 88:1254–1264. [PubMed: 18801858]
 12. Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngology and Head and Neck Surgery*. 1992; 107:399–404.
 13. Epstein M. Diabetes and hypertension: the bad companions. *Journal of Hypertension*. Supplement. 1997; 15:S55–S62.
 14. Ferrannini E, Cushman WC. Diabetes and hypertension: the bad companions. *Lancet*. 2012; 380:601–610. [PubMed: 22883509]
 15. Ishiyama A, Jacobson KM, Baloh RW. Migraine and benign positional vertigo. *Annals of Otolaryngology and Laryngology*. 2000; 109:377–380.
 16. Jang YS, Hwang CH, Shin JY, Bae WY, Kim LS. Age-related changes on the morphology of the otoconia. *Laryngoscope*. 2006; 116:996–1001. [PubMed: 16735917]
 17. Kao CL, Cheng YY, Leu HB, Chen TJ, Ma HI, Chen JW, Lin SJ, Chan RC. Increased risk of ischemic stroke in patients with benign paroxysmal positional vertigo: a 9-year follow-up nationwide population study in taiwan. *Frontiers in Aging Neuroscience*. 2014; 6:108. [PubMed: 24917815]
 18. Korres SG, Balatsouras DG, Papouliakos S, Ferekidis E. Benign paroxysmal positional vertigo and its management. *Medical Science Monitor*. 2007; 13:CR275–CR282. [PubMed: 17534234]
 19. Lawson J, Johnson I, Bamiou DE, Newton JL. Benign paroxysmal positional vertigo: clinical characteristics of dizzy patients referred to a Falls and Syncope Unit. *QJM*. 2005; 98:357–364. [PubMed: 15820968]
 20. Lee SH, Kim JS. Benign paroxysmal positional vertigo. *Journal of Clinical Neurology (Seoul, Korea)*. 2010; 6:51–63.
 21. Lempert T, Leopold M, von Brevern M, Neuhauser H. Migraine and benign positional vertigo. *Annals of Otolaryngology and Laryngology*. 2000; 109:1176.
 22. Maia CA, Campos CA. Diabetes mellitus as etiological factor of hearing loss. *Brazilian Journal of Otorhinolaryngology*. 2005; 71:208–214. [PubMed: 16446919]
 23. Murphy SN, Weber G, Mendis M, Gainer V, Chueh HC, Churchill S, Kohane I. Serving the enterprise and beyond with informatics for integrating biology and the bedside (i2b2). *Journal of the American Medical Informatics Association*. 2010; 17:124–130. [PubMed: 20190053]
 24. Myers SF, Ross MD. Morphological evidence of vestibular pathology in long-term experimental diabetes mellitus. II. Connective tissue and neuroepithelial pathology. *Acta Oto-Laryngologica*. 1987; 104:40–49. [PubMed: 3499049]
 25. Myers SF, Ross MD, Jokelainen P, Graham MD, McClatchey KD. Morphological evidence of vestibular pathology in long-term experimental diabetes mellitus. I. Microvascular changes. *Acta Oto-Laryngologica*. 1985; 100:351–364. [PubMed: 4082974]
 26. Oghalai JS, Manolidis S, Barth JL, Stewart MG, Jenkins HA. Unrecognized benign paroxysmal positional vertigo in elderly patients. *Otolaryngology and Head and Neck Surgery*. 2000; 122:630–634.
 27. Prokopakis E, Vlastos IM, Tsagournisakis M, Christodoulou P, Kawauchi H, Velegrakis G. Canalith repositioning procedures among 965 patients with benign paroxysmal positional vertigo. *Audiology and Neuro-Otology*. 2013; 18:83–88. [PubMed: 23147839]
 28. Shah A, Kanaya AM. Diabetes and associated complications in the South Asian population. *Current Cardiology Reports*. 2014; 16:476. [PubMed: 24643902]
 29. Singh R, Barden A, Mori T, Beilin L. Advanced glycation end-products: a review. *Diabetologia*. 2001; 44:129–146. [PubMed: 11270668]
 30. van Leeuwen RB, van der Zaag-Loonen H. Referrals to a specialised dizziness clinic often result in revised diagnoses and new therapeutic advice. *European Neurology*. 2015; 73:20–22. [PubMed: 25376792]

31. Vibert D, Kompis M, Hausler R. Benign paroxysmal positional vertigo in older women may be related to osteoporosis and osteopenia. *Annals of Otology, Rhinology and Laryngology*. 2003; 112:885–889.
32. von Brevern M, Neuhauser H. Epidemiological evidence for a link between vertigo and migraine. *Journal of Vestibular Research*. 2011; 21:299–304. [PubMed: 22348934]
33. von Brevern M, Radtke A, Lezius F, Feldmann M, Ziese T, Lempert T, Neuhauser H. Epidemiology of benign paroxysmal positional vertigo: a population based study. *Journal of Neurology, Neurosurgery and Psychiatry*. 2007; 78:710–715.
34. Wada M, Naganuma H, Tokumasu K, Hashimoto S, Ito A, Okamoto M. Arteriosclerotic changes as background factors in patients with peripheral vestibular disorders. *Int Tinnitus J*. 2008; 14:131–134. [PubMed: 19205164]
35. Waitman LR, Warren JJ, Manos EL, Connolly DW. Expressing observations from electronic medical record flowsheets in an i2b2 based clinical data repository to support research and quality improvement. *AMIA Annu Symp Proc*. 2011; 2011:1454–1463. [PubMed: 22195209]
36. Wang TJ, Vasan RS. Epidemiology of uncontrolled hypertension in the United States. *Circulation*. 2005; 112:1651–1662. [PubMed: 16157784]
37. Warninghoff JC, Bayer O, Ferrari U, Straube A. Co-morbidities of vertiginous diseases. *BMC Neurology*. 2009; 9:29. [PubMed: 19583869]
38. Way KJ, Katai N, King GL. Protein kinase C and the development of diabetic vascular complications. *Diabetic Medicine*. 2001; 18:945–959. [PubMed: 11903393]
39. Whitney SL, Marchetti GF, Morris LO. Usefulness of the dizziness handicap inventory in the screening for benign paroxysmal positional vertigo. *Otology & Neurotology*. 2005; 26:1027–1033. [PubMed: 16151354]
40. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care*. 2004; 27:1047–1053. [PubMed: 15111519]
41. Yoda S, Cureoglu S, Yildirim-Baylan M, Morita N, Fukushima H, Harada T, Paparella MM. Association between type 1 diabetes mellitus and deposits in the semicircular canals. *Otolaryngology and Head and Neck Surgery*. 2011; 145:458–462.
42. Zhang D, Zhang S, Zhang H, Xu Y, Fu S, Yu M, Ji P. Evaluation of vertebrobasilar artery changes in patients with benign paroxysmal positional vertigo. *Neuroreport*. 2013; 24:741–745. [PubMed: 23903461]

Figure 1.

Hypertension is the mediator of the association between type 2 diabetes and benign paroxysmal positional vertigo. a = coefficient relating type 2 DM to hypertension, b =coefficient relating hypertension to BPPV, c = coefficient relating type 2 DM to BPPV, c' = coefficient relating type 2 DM to BPPV while adjusting for hypertension.

Table 1

Frequency of vertiginous diagnoses in people with and without type 2 DM

	With Type 2 DM (n= 699)	Without Type 2 DM (n= 3222)	p
BPPV	46.1%	37.1%	p<0.001 *
Peripheral vertigo unspecified	12.7%	16.7%	p=0.01 *
Meniere's disease	28.3%	35.4%	p< 0.001 *
Other disorders of labyrinth	1.8%	4.7%	p<0.001 *
Labyrinthine fistula	0.4%	0.5%	p= 0.82
Labyrinthine dysfunction	3.4%	2.3%	p=0.07
Vestibular neuritis	4.0%	3.7%	p=0.69
Central vertigo	7.7%	8.7%	p=0.39
Labyrinthitis	10.7%	8.9%	p=0.29

Chi-square tests were used to examine differences in frequency between groups.

* Significant difference between groups is based on p<0.05

Table 2

Presence of hypertension and type 2 diabetes in people with BPPV (n=1518).

People with BPPV	Hypertension: yes	Hypertension: no
Between 18–40 years		
Diabetes: yes (n=10)	50% (n=5)	50% (n=5)
Diabetes: no (n=161)	11.2% (n=18)	88.8% (n=143)
Between 41–65 years		
Diabetes: yes (n=131)	80.2% (n=105)	19.8% (n=26)
Diabetes: no (n=611)	30.6% (n=187)	69.4% (n=424)
People > 65 years		
Diabetes: yes (n= 181)	90.1% (n=163)	9.9% (n=18)
Diabetes: no (n= 424)	56.6% (n= 240)	43.4% (n= 184)

Table 3

Logistic linear regression model showing the variables predictive of and odds of developing benign paroxysmal positional vertigo

Factors	B	Odds Ratio (95% CI)	p-value
Age (by decade)	0.01	1.10(1.06–1.15)	0.01
Gender (female-reference) n= 2603	0.18	1.19(1.04–1.38)	0.01
Race (White-reference) n= 2977			
Race (African American) n= 396	0.24	1.3 (1.05–1.63)	0.02
Race (Hispanic) n= 13	–0.01	0.98 (0.32–3.06)	0.98
Race (Asian) n= 50	0.94	2.56 (1.44–4.54)	0.001
Race (other/ unknown) n= 485	0.24	1.27 (1.04– 1.55)	0.02
Hypertension (no) n= 2418	–0.55	0.58 (0.49–0.66)	0.00

95% CI= 95% confidence interval, significant p-value is <0.05