Migration Letters

Volume: 19, No: S2 (2022), pp. 681-688

ISSN: 1741-8984 (Print) ISSN: 1741-8992 (Online)

www.migrationletters.com

Evaluation, Causes And Management Of Acute Spontaneous Dizziness

Emad A. Subahi[1*], Adel Saleh Alanazi[2], Laila Khaled Alanazi[2], Mohammed Khalid Alharbi[3] , Hussain Ali Bu Shehab[4], Fares Abdullah Alnakhli[5], Maryam Abdullah Tawhari[6], Sulaiman Mulfi Alshammari[7], Mohammed Munir Alshahrani[8], Abdulmajeed Salem Alfaifi[8], Bandar Mohammed Almotairi[5]

Abstract

Although acute dizziness is a very frequent reason for admissions to emergency care, its differential diagnosis is difficult to establish due to many factors. Dizziness may mean diverse sensations such as vertigo, lightheadedness, presyncope, disequilibrium, or simply feeling unwell. The classical approach to dizziness initiates with characterizing the form of dizziness according to the belief that every kind of dizziness represents a particular underlying etiology: vertigo is vestibular, presyncope is cardiovascular, disequilibrium is neurological, and nonspecific dizziness is psychogenic or metabolic. Development of acute dizziness/vertigo unaccompanied by precip[1]itating features occurs in patients of acute spontaneous dizziness. The dizziness/vertigo mainly presents with autonomic symptoms like imbalance, nausea, and vomiting. Dizziness/vertigo and imbalance are the commonest manifestations in vertebrobasilar ischemia, which accounts for over 20% of all ischemic strokes. It is essential to distinguish between isolated vascular vertigo from less severe disorders related to the inner ear as the treatment plan and prognosis vary among these problems. The development of diffusion-weighted magnetic resonance imaging (MRI) has improved the infarction identification in cases with vascular dizziness/vertigo, particularly from undermined posterior blood flow. Nonetheless, proper bedside neurotologic assessment which includes components such as head impulse/thrust test, head shaking nystagmus test and ocular tilt reaction test demonstrate higher sensitivity than imaging in diagnosing acute cerebrovascular accidents as an etiology of abruptly occurring vertigo lasting longer than one day, particularly in the initial 48 hours.

Keywords: *dizziness, vertigo, emergency medicine, stroke*

Introduction

Although acute dizziness is a very frequent reason for admissions to the emergency care (1), its differential diagnosis is difficult to establish due to many factors. Firstly, it can be a presentation of diseases ranging from serious conditions to normal physiological responses.

[1]Department of Emergency Medicine, Al Noor Specialist Hospital, Mecca, Saudi Arabia [2]Department of Emergency Medicine, King Salman Armed Forces Hospital, Tabuk, Saudi Arabia [3]Department of Internal Medicine, Hera General Hospital, Mecca, Saudi Arabia
[4]College of Medicine, University of Pécs, Pécs, Hungary
[5]College of Medicine, Shaqra University, Shaqra, Saudi Arabia
[6]College of Medicine, Princess Nourah Bint Abdul Rahman University, Riyadh, Saudi Arabia [7]Department of Ophthalmology, King Khalid Hospital, Hail, Saudi Arabia
[8]Department of Emergency Medicine, King Salman Hospital, Riyadh, Saudi Arabia

Secondly, there is no diagnostic confirmatory aid existing for most conditions inducing dizziness. Therefore, the diagnosis is mainly built on a sequence of clinical characteristics acquired through diligent history taking and bedside evaluation (2). The most common types of dizziness in the emergency department are benign paroxysmal positional vertigo, cerebellar stroke and vestibular neuritis (3). Neither hematological testing nor neuroimaging are cost-efficient when administered to dizzy patients non-selectively (4). Thirdly, the term "dizziness" may mean diverse sensations such as vertigo, lightheadedness, presyncope, disequilibrium, or simply feeling unwell (5). The classical approach to dizziness initiates with characterizing the form of dizziness according to the belief that every kind of dizziness represents a particular underlying etiology: vertigo is vestibular, presyncope is cardiovascular, disequilibrium is neurological, and nonspecific dizziness is psychogenic or metabolic (5). However, certain individuals face challenges explaining their particular kind of dizziness (6). Furthermore, vestibular conditions can present with different patterns of dizziness and heart conditions/systemic hypotension may cause vertigo in addition to vestibular presentations (7). Hence, the classical method that depends heavily on the symptomatology poses difficulties (4). A recent suggested method to diagnosing dizziness starts with categorizing the kind of dizziness/vertigo into acute extended abrupt dizziness/vertigo, recurring abrupt dizziness/vertigo, recurring positional vertigo, or chronic continuing dizziness and disequilibrium. This novel method is regarded more rational and more useful in organization of a differential diagnosis in every group.

METHODOLOGY

No specific criteria were selected beforehand to determine which publications would be incorporated in this review. Google Scholar search engine was utilized to look for scientific publications containing "acute spontaneous dizziness" and "acute spontaneous vertigo". After a preliminary scanning of abstracts, full-lengths of relevant articles from peer-reviewed journals were acquired. The references sections of these articles were also screened for pertinent citations which were referred to for additional review.

DISCUSSION

Development of acute dizziness/vertigo unaccompanied by precipitating features occurs in patients in acute spontaneous dizziness (2). The dizziness/vertigo mainly presents with autonomic symptoms like imbalance, nausea, and vomiting. The event can be transient (lasting less than a day) or of an extended duration. Vestibular neuritis, which is a benign inflammation of the vestibular labyrinth, and stroke are the main diseases for differential diagnosis in this phenotype.

As opposed to the classical assumption about dizziness/vertigo generally co-occurring with other neurological manifestations in cerebrovascular diseases, detection of solitarily occurring cases of vascular vertigo are rising due to modern advancements in neurotology and imaging (8). Dizziness/vertigo and imbalance are the commonest manifestations in vertebrobasilar ischemia, which accounts for over 20% of all ischemic strokes (9, 10). It is essential to distinguish between isolated vascular vertigo from less severe disorders related to the inner ear as the treatment plan and prognosis vary among these problems (11). Misdiagnosed acute cerebrovascular incidents can cause substantial debilitation and even death, whereas excessive clinical detection of vascular vertigo can lead to expensive investigations and pharmaceutical therapy (11). The development of diffusion-weighted magnetic resonance imaging (MRI) has improved the infarction identification in cases with vascular dizziness/vertigo, particularly from undermined posterior blood flow (12). Nonetheless, proper bedside neurotologic assessment has higher sensitivity than imaging in diagnosing acute cerebrovascular accidents

as an etiology of abruptly occurring vertigo lasting longer than one day, particularly in the initial 48 hours (13-15).

History taking

Although neurotological evaluation is important for finding potential causes of acute abrupt extended dizziness/ vertigo, medical record must comprise previous infections, vascular predisposing elements (for instance; hypertension, diabetes mellitus, dyslipidemia, tobacco smoking, and heart disease), and related discomfort in the head and neck region. In the absence of other clinical features indicating autonomous nervous system problem, the onset of acute dizziness or vertigo accompanied by extremely intense aching in the head and neck region firmly suggests a cerebral disease (16).

Such individuals coming into the emergency care showed twice the risk of experiencing cerebrovascular and cardiovascular accidents compared to cases without dizziness/ vertigo, according to one article (17). Further, individuals with vertigo and ≥3 vascular predisposing determinants showed a 5.51-times greater susceptibility to strokes as compared to their counterparts lacking the predisposing elements (17). Additional research implementing the $ABCD^2$ point system (18), a medical prognostic resource for evaluating the predisposition to cerebrovascular events following a transient ischemic event noted these events only in 1% of emergency cases experiencing dizziness and ≤3 points in comparison to 8.1% in cases with ≥4 points (19). Notably, ~27% cases scoring six or seven had experienced cerebral accidents (19). Thus, the $ABCD^2$ score can estimate the possibility of cerebrovascular events in individuals experiencing transient vertigo.

Evaluation

Nystagmus

Manifestations of abruptly occurring nystagmus are mainly useful in assessing cases with acute-onset dizziness or vertigo (20). For comprehensive assessment of abrupt-onset nystagmus, the clinician should note the path and the impact of gaze on the severity and orientation of the nystagmus. In cases of vestibular neuritis, abrupt-onset nystagmus is horizontal-torsional directing away from the injured side. The nystagmus generally becomes severe when one gazes along the path of the spontaneous nystagmus and reduces while gazing in the opposing way (21). As peripheral vestibular nystagmus undergoes substantial suppression due to visual fixation, for accurate identification of nystagmus visual fixation needs to be eliminated via the use of Frenzel glasses (22). On the other hand, the orientation and visual fixation manifestations can differ in central type. Subsequently, when the features are not aligned with those observed in the peripheral type, central type should be suspected (23-26). Even though one-way torsional-horizontal type undergoes suppression via fixation, caution must be taken while declaring it as peripheral type without investigations for peripheral vestibular injuries like positive head impulse (HIT) or caloric test.

Several bedside maneuvers can prompt nystagmus or regulate pre-existing spontaneous nystagmus. In cases with abrupt-onset nystagmus also, pattern variance may indicate the fundamental disease mechanism or help in diagnosing it. In cases showing compensated vestibular illness, prompting of nystagmus by different maneuvers is essential for discovering the root cause of vestibulopathy related imbalance (27, 28). Gaze- evoked nystagmus (GEN) implies nystagmus which onsets when individuals assume eccentric eye positions. GEN results from defective gaze holding (or looking) in such positions, that lead to centripetally beating nystagmus (29, 30). In individuals with acute vestibular syndrome, it is the most discerning oculomotor manifestation for central vertigo (12, 13). By utilizing either a passive (by examiner) or active (by patients) head shaking maneuver, head- shaking nystagmus (HSN) can be prompted. For bringing the horizontal semicircular canals into the stereotaxic plane of

activation, the individual's head is tilted ahead by around 30°. Thereafter, it is maneuvered at diverse angles in the horizontal plane at two to three Hz with a range of 20° for quarter of a minute (28). In unifacial peripheral vestibular disease, HSN at first contralesionally, wanes over a duration of 20 seconds, followed by undergoing a feeble reversal (31). On the other hand, HSN patterns can differ in central vestibular disorders. Usually, central HSN comprises remarkably strong HSN evoked by feeble headshaking, strong HSN in individuals in the absence of canal hypofunction, HSN on the same side as the lesion, HSN contralaterally to spontaneous nystagmus, and downward or upward beating nystagmus after maneuvering in the horizontal plane.

Head impulse test (HIT)

For the bedside evaluation of vestibular function loss, HIT ranks the highest with respect to efficiency (21). For achieving this, the patient is asked to focus on an object ahead while rapid movements of the individual's head are performed in the horizontal plane. The head impulse may be unforeseeable with a small range like between 10° to 20° and elevated rate of head rotation (32). In case of a normally functioning vestibulo-ocular reflex, HIT will produce a compensating ocular motion contralateral to the head rotation with same range, bearing the gaze steadily. Contrary to this, performing the test ipsilateral to the peripheral vestibulopathic side will lead to corrective saccade which is related to a reduced vestibulo-ocular reflex in cases with peripheral-origin vestibulopathy. Though these movements are seen in majority of cases with acute-onset peripheral-origin vestibulopathies, HIT is generally regular in central vestibulopathies (33). Therefore, one should consider a central vestibular injury when the case presenting with acute-onset vertigo and abruptly initiating nystagmus displays normal HIT (12). A refixating saccadic motion in a separate plane too is indicative of a central lesion (34-36). Nonetheless, this clinical test can produce negative results in case of incomplete vestibulopathies or saccadic camouflaging of vestibulopathies (32).

Ocular misalignment

Occurring frequently in central vestibular lesions, this condition needs be examined in nine primary gaze orientations additionally with limited ranges of ocular motion. Skew deviation means vertical ocular misalignment caused by vestibular tone imbalance (37). The occurrence of this phenomenon can be confirmed through observation of vertical diplopia and can be established with the cover-uncover testing. Skew deviation may develop spontaneously in an injury in relation to the posterior cranial fossa, but most cases are related to cerebrovascular events in the brain stem. (38) The phenomenon is usually seen as a part of the ocular tilt reaction which consists of vertical deviation, bilateral ocular torsion as well as ipsilateral head and neck tilting. Generally, tilting develops ipsilateral to the inferior eye and the eyes tort in a similar manner such that superior poles bilaterally rotate toward the inferior eye (39). Injuries occurring inferior to the lower pontine region lead to an ocular tilt reaction directed ipsilateral to the hypotropic eye, whereas comparatively rostrally-situated pathologies cause ocular tilt reactions contraversively.

Balance

The intensity of disequilibrium and directional falls can offer significant hints regarding the root cause of vestibulopathy. Typically, in one-sided peripheral vestibulopathy equilibrium may be possibly maintained despite extreme dizziness/vertigo (16). On the contrary, extreme imbalance which undermines sitting or standing typically suggests a central pathology (16).

Neuroimaging

The utility of imaging investigations is rising in emergency management of dizzy patients. Nonetheless, the computed tomographic scans to detect spontaneously occurring cerebrovascular accidents are not very sensitive (26%) (40). Therefore, a negative scan alone cannot eliminate the suspicion of a cerebrovascular event. MRI is far more sensitive; however, it is the least sensitive in the first day of onset, particularly in brain stem-related or cerebellar lesions (13, 33, 41). Further, MRI cannot identify solitary labyrinthine infarctions which may proceed to encroach more brain stem or cerebellar areas connected by the anterior inferior cerebellar artery (42). Perfusion imaging can aid in the identification and gauging of cerebral perfusion defects, particularly when procedural MRI, which includes diffusion-weighted images, is normal (43). But the precise purpose of this scan requires justification in vertigo cases without focal neurological symptoms occurring secondarily to vasculopathies.

Transient vascular vertigo and solitary labyrinth infarcts

Although isolated transient vascular vertigo (ITVV)/dizziness occurs frequently in relation to vertebrobasilar (VBI) ischemic lesions, their identification is challenging (10, 44, 45). They often presents suddenly and generally continue for a number of minutes (46). One study noted that ~62% of cases with vertigo because of VBI had experienced at least a single incident of ITVV , with ~19% occurring with vertigo as the first presenting sign (45). Cases of AICA related infarcts can show isolated recurring episodes, intermittent hypoacusis as well as tinnitus as the presenting symptoms one to ten days before permanent infarction (47).

This diagnostic challenge also relates to isolated labyrinth infarcts as no confirmatory resource apart from than a pathologic testing is present at the moment for this disorder (48). Since the internal auditory artery originating from AICA typically, provides blood supply to the labyrinth, VBI related cerebrovascular events can occur with vertigo/dizziness and hypoacusis as a result of the infarct. A possibility of labyrinth infarct needs to be taken into consideration in elderly individuals presenting with acute vertigo/dizziness and one-sided hypoacusis, specifically in individuals who have experienced previous cerebrovascular accidents or possess predisposing factors. As current imaging provides poor visualization of these infarcts (48), clinicians need to take into account the results of all medical investigations to confirm the etiological source of vertigo/dizziness and hypoacusis (49).

Management

Cases suspected of vascular vertigo must be assessed promptly for their cerebral vasculature (11, 50). Since non-lacunar pathologic pathways have shown to be more prevalent than formerly believed in acute vestibulopathies from minor infarcts, advanced treatment strategies are required to avoid recurrent cerebrovascular events in vascular-origin vertigo/dizziness (14, 51).

The therapy for vestibular neuritis usually involves symptomatic therapy during the initial period, corticosteroids, and targeted vestibular therapy (52). Supportive therapy with suppressing agents needs to be offered exclusively in the initial period during which individuals are severely nauseous and facing extreme vertigo because these drugs may cause delayed central compensation (21). The efficaciousness of corticosteroids is disputed. According to one report, there is presently inadequate proof in favor of the administration of steroids in cases of idiopathic abrupt onset vestibulopathy (53). Use of valacyclovir exclusively, or along with glucocorticoids failed to provide any relief (54). On the contrary, vestibular therapy sped convalescence (55).

CONCLUSION

The assessment and comprehensive treatment of acute dizziness/vertigo are essential in the emergency department. Special care needs to be taken for possibly fatal diseases underlying the presentation of acute dizziness/vertigo. One current treatment proposal for dizziness/vertigo

suggests beginning by categorizing the kind of dizziness/vertigo manifestations. Emergency medicine staff needs to be acquainted with investigations for identification of signs of central vestibulopathy like head impulse/thrust test and direction changing positional nystagmus in cases of abrupt-onset, extended dizziness/vertigo. These features demonstrate higher sensitivity than neuroimaging for the purpose of identification of cerebrovascular incidents accompanying abrupt-onset and solitarily occurring dizziness/vertigo. In the previous years, remarkable advances have been made with regard to the assessment and treatment of acute dizziness/vertigo. Despite these achievements in neuroimaging sciences, the diagnostic evaluation of acute dizziness/vertigo heavily relies on clinical testing.

REFERENCES

1. Cappello M, Di Blasi U, Di Piazza L, Ducato G, Ferrara A, Franco S, et al. Dizziness and vertigo in a department of emergency medicine. European journal of emergency medicine: official journal of the European Society for Emergency Medicine. 1995;2(4):201-11.
2. Baloh RW. Vertigo. The Lancet. 1998;352(9143):1841-6.
3. Zarachi A, Pezoulas V, Lianou A, Tsikou A, Tsiakas I, Dinaki K, et al. Dizziness in the Emergency Department: Insights and Epidemiological Data - a Population Based Study. Maedica. 2022;17(1):122-8.
4. Newman-Toker DE, Cannon LM, Stofferahn ME, Rothman RE, Hsieh Y-H, Zee DS, editors. Imprecision in patient reports of dizziness symptom quality: a cross-sectional study conducted in an acute care setting. Mayo Clinic Proceedings; 2007: Elsevier.
5. Drachman DA, Hart CW. An approach to the dizzy patient. Neurology. 1972.
6. Hanley K, O'Dowd T. Symptoms of vertigo in general practice: a prospective study of diagnosis. British journal of general practice. 2002;52(483):809-12.
7. Choi JH, Seo JD, Kim MJ, Choi BY, Choi Y, Cho B, et al. Vertigo and nystagmus in orthostatic hypotension. European Journal of Neurology. 2015;22(4):648-55.
8. Kim SH, Park SH, Kim HJ, Kim JS. Isolated central vestibular syndrome. Annals of the New York Academy of Sciences. 2015;1343(1):80-9.
9. Savitz SI, Caplan LR. Vertebrobasilar disease. New England Journal of Medicine. 2005;352(25):2618-26.
10. Paul NL, Simoni M, Rothwell PM. Transient isolated brainstem symptoms preceding posterior circulation stroke: a population-based study. The Lancet Neurology. 2013;12(1):65-71.
11. Choi K-D, Lee H, Kim J-S. Vertigo in brainstem and cerebellar strokes. Current opinion in neurology. 2013;26(1):90-5.
12. Lee H, Sohn S-I, Cho Y-W, Lee S-R, Ahn B-H, Park B-R, et al. Cerebellar infarction presenting isolated vertigo: frequency and vascular topographical patterns. Neurology. 2006;67(7):1178-83.
13. Kattah JC, Talkad AV, Wang DZ, Hsieh Y-H, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. Stroke. 2009;40(11):3504-10.
14. Newman-Toker DE, Saber Tehrani AS, Mantokoudis G, Pula JH, Guede CI, Kerber KA, et al. Quantitative video-oculography to help diagnose stroke in acute vertigo and dizziness: toward an ECG for the eyes. Stroke. 2013;44(4):1158-61.
15. Tehrani ASS, Kattah JC, Mantokoudis G, Pula JH, Nair D, Blitz A, et al. Small strokes causing severe vertigo: frequency of false-negative MRIs and nonlacunar mechanisms. Neurology. 2014;83(2):169-73.
16. Baloh RW. Differentiating between peripheral and central causes of vertigo. Otolaryngology—Head and Neck Surgery. 1998;119(1):55-9.
17. Lee C-C, Su Y-C, Ho H-C, Hung S-K, Lee M-S, Chou P, et al. Risk of stroke in patients hospitalized for isolated vertigo: a four-year follow-up study. Stroke. 2011;42(1):48-52.
18. Johnston SC, Rothwell PM, Nguyen-Huynh MN, Giles MF, Elkins JS, Bernstein AL, et al. Validation and refinement of scores to predict very early stroke risk after transient ischaemic attack. The Lancet. 2007;369(9558):283-92.

19. Navi BB, Kamel H, Shah MP, Grossman AW, Wong C, Poisson SN, et al. Application of the ABCD2 score to identify cerebrovascular causes of dizziness in the emergency department. Stroke. 2012;43(6):1484-9.
20. Serra A, Leigh R. Diagnostic value of nystagmus: spontaneous and induced ocular oscillations. Journal of Neurology, Neurosurgery & Psychiatry. 2002;73(6):615-8.
21. Robinson DA, Zee DS, Hain TC, Holmes A, Rosenberg LF. Alexander's law: Its behavior and origin in the human vestibulo-ocular reflex. Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society. 1984;16(6):714-22.
22. Hotson JR, Baloh RW. Acute vestibular syndrome. New England Journal of Medicine. 1998;339(10):680-5.
23. Baloh R, Yee R. Spontaneous vertical nystagmus. Revue neurologique. 1989;145(8-9):527-32.
24. Böhmer A, Straumann D. Pathomechanism of mammalian downbeat nystagmus due to cerebellar lesion: a simple hypothesis. Neuroscience letters. 1998;250(2):127-30.
25. Baloh RW, Spooner JW. Downbeat nystagmus: a type of central vestibular nystagmus. Neurology. 1981;31(3):304-.
26. Glasauer S, Hoshi M, Kempermann U, Eggert T, Buttner U. Three-dimensional eye position and slow phase velocity in humans with downbeat nystagmus. Journal of Neurophysiology. 2003;89(1):338-54.
27. Huh Y-E, Kim J-S. Bedside evaluation of dizzy patients. Journal of Clinical Neurology. 2013;9(4):203-13.
28. Choi KD, Oh SY, Kim HJ, Koo JW, Cho BM, Kim JS. Recovery of vestibular imbalances after vestibular neuritis. The Laryngoscope. 2007;117(7):1307-12.
29. Büttner U, Grundei T. Gaze-evoked nystagmus and smooth pursuit deficits: their relationship studied in 52 patients. Journal of neurology. 1995;242(6):384-9.
30. Leigh RJ, Zee DS. The neurology of eye movements: Contemporary Neurology; 2015.
31. Hain T, Fetter M, Zee D. Head-shaking nystagmus in patients with unilateral peripheral vestibular lesions. American journal of otolaryngology. 1987;8(1):36-47.
32. Weber K, Aw S, Todd M, McGarvie L, Curthoys I, Halmagyi G. Head impulse test in unilateral vestibular loss: vestibulo-ocular reflex and catch-up saccades. Neurology. 2008;70(6):454-63.
33. Newman-Toker DE, Kattah JC, Alvernia JE, Wang DZ. Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis. Neurology. 2008;70(24 Part 2):2378-85.
34. Walker MF, Zee DS. Cerebellar disease alters the axis of the high-acceleration vestibuloocular reflex. Journal of neurophysiology. 2005;94(5):3417-29.
35. Walker MF, Zee DS. Directional abnormalities of vestibular and optokinetic responses in cerebellar disease. Annals of the New York Academy of Sciences. 1999;871(1):205-20.
36. Jeong S-H, Kim J-S, Baek IC, Shin JW, Jo H, Lee AY, et al. Perverted head impulse test in cerebellar ataxia. The Cerebellum. 2013;12(5):773-5.
37. Brodsky MC, Donahue SP, Vaphiades M, Brandt T. Skew deviation revisited. Survey of ophthalmology. 2006;51(2):105-28.
38. Smith JL, David NJ, Klintworth G. Skew deviation. Neurology. 1964;14(2):96-.
39. Dieterich M, Brandt T. Ocular torsion and tilt of subjective visual vertical are sensitive brainstem signs. Annals of neurology. 1993;33(3):292-9.
40. Chalela JA, Kidwell CS, Nentwich LM, Luby M, Butman JA, Demchuk AM, et al. Magnetic resonance imaging and computed tomography in emergency assessment of patients with suspected acute stroke: a prospective comparison. The Lancet. 2007;369(9558):293-8.
41. Oppenheim C, Stanescu R, Dormont D, Crozier S, Marro B, Samson Y, et al. False-negative diffusion-weighted MR findings in acute ischemic stroke. American Journal of Neuroradiology. 2000;21(8):1434-40.
42. Kim JS, Cho K-H, Lee H. Isolated labyrinthine infarction as a harbinger of anterior inferior cerebellar artery territory infarction with normal diffusion-weighted brain MRI. Journal of the neurological sciences. 2009;278(1-2):82-4.
43. Kim DU, Han M-K, Kim JS. Isolated recurrent vertigo from stenotic posterior inferior cerebellar artery. Otology & Neurotology. 2011;32(1):180-2.
44. Grad A, Baloh RW. Vertigo of vascular origin: clinical and electronystagmographic features in 84 cases. Archives of Neurology. 1989;46(3):281-4.

45. Hoshino T, Nagao T, Mizuno S, Shimizu S, Uchiyama S. Transient neurological attack before vertebrobasilar stroke. Journal of the neurological sciences. 2013;325(1-2):39-42.
46. Fisher C. Vertigo in cerebrovascular disease. Archives of Otolaryngology. 1967;85(5):529-34.
47. Lee H, Cho Y. Auditory disturbance as a prodrome of anterior inferior cerebellar artery infarction. Journal of Neurology, Neurosurgery & Psychiatry. 2003;74(12):1644-8.
48. Kim J, Lopez I, DiPatre P, Liu F, Ishiyama A, Baloh RW. Internal auditory artery infarction: clinicopathologic correlation. Neurology. 1999;52(1):40-.
49. Kim H-A, Lee S-R, Lee H. Acute peripheral vestibular syndrome of a vascular cause. Journal of the neurological sciences. 2007;254(1-2):99-101.
50. Kim JS, Lee H, editors. Vertigo due to posterior circulation stroke. Seminars in neurology; 2013: Thieme Medical Publishers.
51. Jackson C, Sudlow C. Comparing risks of death and recurrent vascular events between lacunar and non-lacunar infarction. Brain. 2005;128(11):2507-17.
52. Jeong S-H, Kim H-J, Kim J-S, editors. Vestibular neuritis. Seminars in neurology; 2013: Thieme Medical Publishers.
53. Fishman JM, Burgess C, Waddell A. Corticosteroids for the treatment of idiopathic acute vestibular dysfunction (vestibular neuritis). Cochrane Database of Systematic Reviews. 2011(5).
54. Strupp M, Zingler VC, Arbusow V, Niklas D, Maag KP, Dieterich M, et al. Methylprednisolone, valacyclovir, or the combination for vestibular neuritis. New England Journal of Medicine. 2004;351(4):354-61.
55. Strupp M, Arbusow V, Maag K, Gall C, Brandt T. Vestibular exercises improve central vestibulospinal compensation after vestibular neuritis. Neurology. 1998;51(3):838-44.