Peripheral Vestibular Vertigo: Clinical Spectrum And Review Of Aetiological Factors.

OA Lasisi
Department of Otorhinolaryngology, University of Ibadan, Ibadan, Nigeria.

Abstract

Vertigo constitutes a significant proportion of specialist otorhinolaryngological (ORL) outpatient practice although management may be multidisciplinary. Accurate diagnosis remains a challenge, often depending on clinical/neuro-otological examination, often with limited availability of costly and sophisticated diagnostic facilities, the yields of which are often low.This report aims at creating a reference database for management of vertigo in the (ORL) Clinic.

This is a retrospective chart review of vertigo patients seen in ORL outpatient clinic through 2001 to 2006. Clinical/neuroo-otological examination, radiology and audiometry were the major modalities of investigation.

The prevalence of vertigo in the Oto-Rhino-Laryngology outpatient Clinic was 15% (134). There were 79 males and 55 females with peak age at presentation between the 3rd and 4th decade of life. Benign paroxysmal positional vertigo 33 (25%), Labyrinthitis 23(17%), menieres disease 19(14%) and cervical spondylosis 14(10.5%) were the leading clinical diagnoses. Trauma accounted for 35% of the aetiological factors while 17% was idiopathic.

Clinical examination remains the prime technique in accurate diagnosis of vertigo although contemporary diagnostic facilities may help. The spectrum of the non-traumatic and idiopathic cases suggest a need for immunogenetic study in the diagnosis.

Key Words: Vertigo, clinical diagnosis, aetiology, outpatient review

Correspondence to:

Dr O.A Lasisi
P.O. Box 22040,
University of Ibadan, Ibadan, Nigeria
E-MAIL: sakeemng@yahoo.com

Introduction

Balance is a complex mechanism comprising of sensory inputs from the vestibular apparatus, vision, and proprioception. These inputs pass into the central nervous system, integrated and modulated by activity of the cerebellum, the extrapyramidal system, the limbic system, and the cerebral cortex, the disorders are thus classified into central and peripheral.

The reported incidence of vertigo is 5-10%, reaching 40% in patients older than 40 years[1, 2]. In the U.S, It is among the 25 leading causes for clinic visit affecting about 42 % of the population and the cost of medical care has been estimated to exceed $1 billion per year [1, 2]. It accounts for more than 5 million dizziness-vertigo visits a year in U. S. A and seven million clinic visits a year in South Africa [1-4].

Peripheral vestibular pathology constitute 40 – 50% of the causes of balance disorder [1, 2, 5, 6]. About 50-60% are idiopathic, however BPPV, labyrinthitis and meneres disease are the common causes[1, 2, 7, 8]. The aetiopathogenesis are mainly due to trauma, although in about one-third of cases, this is also unknown[7-11].

In the evaluation of the patients, history and physical examination remain the prime clinical strategies. However, additional tests are commonly considered, these include audiometry, electrocochleography, auditory brainstem response, otoacoustic emissions, vestibular tests, blood tests, Computerised Tomography Scan, and Magnetic Resonance Imaging. The yields of these tests are low and their routine use is probably not cost-effective.

This paper reports the spectrum of clinical diagnosis and reviews the aetiology of vertigo seen in the patients presenting to our Oto-Rhino-Laryngology Clinic.

Materials And Methods

This is a retrospective chart review of patients who presented at the otorhinolaryngology Outpatient clinic with complaints of vertigo/dizziness through the year 2000 - 2006.

The hospital charts were computerized making retrieval of data easy. The biodata, clinical features and laboratory investigations were retrieved in order to document the clinical diagnosis and aetiological factors. The laboratory investigations included complete blood count and film, serum sodium, potassium, chloride, bicarbonate and urea, serology for syphilis; and plain radiography and computerized tomography scan of the skull base. Serum rheumatoid factor was requested in cases with unknown aetiology and high electrolyte sedimentation rate. Pure tone audiometry was done by a speech pathologist who also had audiology training. The World Health Organization classification were used for auditory assessment. The aetiological factors and clinical diagnosis were determined using simple definitions(Appendix 1). These were analysed and the results presented in pictorial forms.

Results

Vertigo was the major symptom in 134(15.4%) out of 869 patients seen in the otorhinolarynglogic outpatient clinic during the period of study. This comprised of 79 males and 55 females (1.4: 1). The age at presentation ranged between 16years and 74 years, mean of 48 years, the 3rd and 4th accounted for 64 (47.8%), Figure 1. The clinical diagnoses were Benign paroxysmal positional vertigo (BPPV) 33 (25%), Labyrinthitis 23(17%), menieres disease 19(14%) and cervical spondylosis 14(10.5%) others Table.1.

Trauma was the most significant aetiological factors, 47 (35%) while the cause was unknown in 23 (17%), the others are as in Fig 2.

Discussion

A prevalence of 14.7% suggests that balance disorder is significant in the outpatient consultation. This compares with 18% reported by Guilemany et al[5] and other authors which reported 1 in 7 and 1 in 10 patients in the Oto-Rhino-Laryngology Clinic[6 - 9]. Similarly, peak age at presentation was 3rd and 4th decade of life accounting for about 50% of the cases.

Diagnosis in this study was based on clinical judgment, careful neurotologic examination, audiometry and radiological studies. We feel the availability of sophisticated diagnostic modalities such as bithermal caloric test, electronystagmography, Video-based electronystagmography, chair and dynamic posturography would have further substantiated our report. However, the yields from these studies have been reported to be less than 1% [10, 11].

BPPV, labyrinthitis and Meniere's disease were most common clinical diagnoses and no etiology could be established in 19%. This is similar to other reports[12, 13, 14].

BPPV was first described by Adler in 1897, it is the most common cause of positional vertigo, accounting for about 90% of vertigo patients and 17-40% of patients in specialised vertigo clinics.[15, 16,17] . The idiopathic cases of BPPV accounted for 50%–70%[17,18]; among the secondary cases, head trauma was most frequent accounting for 7%–17%[17, 18, 19]. Hoffer et al[20] had reported BPPV in 28% of persons with post-traumatic vertigo. The mechanism is thought to be release of otoconia into the endolymph leading to vertigo[17, 18, 19]. Others are viral infection, degeneration of the peripheral end organ due to ageing, surgical damage to the labyrinth and minor strokes involving anterior inferior cerebellar artery[21, 22].

Two theories have been developed to explain its pathophysiology; cupulolithiasis and canalithiasis which is more widely accepted. The dizziness and other symptoms is due to detachment and displacement of calcium carbonate debris also referred to as a "canalith" from the utricle to the semicircular canals. In our own protocol the Dix-Hallpike positioning test was used as the key to diagnosis, similar to other workers[21, 22, 23 24]. The aim of the positioning test is to make otoconia in the posterior semicircular canal move and so provoke vertigo and nystagmus. The prevalence of BPPV increases with advancing age; women are affected almost twice as often as men. The posterior canal is the most commonly involved, about 90%, Korres et al[25] reported lateral canal BPPV in 3-9% and anterior canal BPPV in 2%.

Acute labyrinthitis seen in our series is sequel to otitis media in about 50% of cases, the rest were viral, traumatic and idiopathic. Schuknecht[7] classified labyrinthine sequelae in otitis media thus: toxic labyrinthitis due to biochemical or bacterial toxins from the middle ear, suppurative (acute and chronic otogenic suppurative) labyrinthitis, in which bacteria have invaded the otic capsule and meningogenic suppurative labyrinthitis, which is the invasion of bacteria from the subarachnoid space into the labyrinth and others include labyrinthine fistula and labyrinthitis ossificans. All these follow spreads through the oval or round window; a

preexisting temporal bone fracture; an area of bone erosion by cholesteatoma, tumor, or chronic infection; a congenital defect; iatrogenic or meningitis. The impression in our patients is toxic labyrinthitis, as all of them had had otitis media for more than 10 years and no evidence of fistula was seen using a Siegle pneumatic otoscope and computed tomographic scan. However, more sophisticated methods of detecting fistula which were not available to us include electronystagmography, electrocochleography and the use of Beta 2-transferrin to detect the presence of perilymph in the middle ear[26, 27, 28].

Vestibular neuronitis has been reported with an incidence of 170/100,000 people[9, 29]. Its etiology remains unknown although it is believed to be of viral etiology, acute localized ischemia have been proposed to be an important cause. It is sometimes referred to as vestibular neuropathy because condition is not clearly inflammatory in nature.

Ménière disease was first described In 1861 by Prosper Ménière. Diagnosis is usually based on history of episodic vertigo, tinnitus and audiometric evidence of low frequency hearing loss which may fluctuate. However glycerol test and electrocochleography have been reported useful in confirmation of disease and selection for surgery[30 - 37]. It affects white people predominantly, with a prevalence in the United Kingdom of 1/ 1000 of the population, both sexes being represented equally[32]. The peak age of onset is between 20 and 50 years, although it may occur in children[38] ; and bilaterality has been reported in over 40% of cases[39]. The aetiology was idiopathic in about 45 % of cases.

Allergy was first proposed to be a possible etiologic factor by Duke and other workers, Pulec reported a prevalence of 14%[40 - 44].

Evidence of immune complex deposition in the endolymphatic has reinforced the immune basis of ménière's disease[45]. Reactivity to sialyl-i ganglioside in the sera of patients, recombinant glutathione-S-transferase-Raf-1 protein and elevated levels of antibodies to inner ear proteins from patients have been reported, and the incidence of these antibodies correlated significantly with disease activity[46 - 51].

Data from various studies reported 5-15% heredity[30 - 33], suggesting familial predisposition. Morrison[32] reported a family history in 7.7% with an autosomal dominant mode of inheritance, penetrance of around 60%, and obvious genetic anticipation.

The genetic basis was also supported by the finding of human leukocyte antigen A2 in 90 % of Meniere's disease patients with a positive family history and 75 % with solitary disease; and major histocompatibility complex genes responsible for the type II collagen disorders have been reported in patients[52, 53]. Bernstein et al[53] reported MHC haplotype Dqw2-Dr3-c4Bsf-C4A0-G11: 15-Bf: 0.4-C2a-HSP70:7.5-TNF in 44% of patients with menieres, otosclerosis and strial presbyacusis had one particular extended, compared to only 7% of controls. In addition HLA-DRB1*04, DQA1 03 and 05 has been described in Koreans with sudden hearing loss[54, 55].

Other diseases reportedly associated with meniere's disease include adrenal pituitary insufficiency 7%, syphilis 6%, hypothyroidism 2%, vasculitis 3%, estrogen insufficiency 2% and viral etiology in 1 % of the patients[46 - 50]. Paparella et al[56] has reported that chronic otitis media may lead to endolymphatic hydrops, this was also supported by Meyerhoff et al[57] and Shojaku[58].

Autoimmune inner-ear disease (AIED) accounted for 4.5% in our review, although we adhered strictly to laboratory evidence of at least a high electrolyte sedimentation rate. However, it has been reported that clinical and laboratory evidence of a systemic disorder and laboratory markers for inner-ear antigenicity have low sensitivity and may not be present. AIED is rare, reported prevalence being less than 1% of vertigo[59, 60]. Typically AIED presents with rapidly progressive, bilateral hearing loss and vestibular hypofunction although it may be unilateral in the onset. However, the rapid progression and early bilateral involvement distinguishes it from ménière's syndrome. It is rare, accounting for less than 1% of vertigo[59, 60]. The theories include immune reactions provoked by release of cytokines from damage to the inner ear[59, 60]; and cross-reactions with antibodies or rogue T-cells causing accidental inner ear damage because the ear shares common antigens with a potentially harmful substances, viruses or bacteria[61]. Out of 52 patients examined by Hughes[62], he reported Cogan's syndrome 7, rheumatoid arthritis 4 and 1 systemic lupus erythematosus, the etiology is uncertain in the rest; in our review also, the cause of immunopathology is unknown, further research

may be needed.

Presbystasis has been proposed as a term to encompass the dysequilibrium of aging, it remains a diagnosis of exclusion. It affects about 25 - 30 percent of people above 65 and it has been reported to be at least 40% heritable. This was further reinforced by the report of genes on chromosome 3q called DFNA18, in a study of 50 pairs of elderly male fraternal twins with hearing loss[63, 64].

Trauma was the most significant aetiological factor in our review accounting for 38%. The presentation may be positional vertigo, cervical vertigo, osteophytes and menieres disease. Post-traumatic positional vertigo has been reported to be due to utricular injury, vestibular atelectasis, and various forms of central vertigo caused by cerebellar or brainstem disturbances. The mechanism in meniere syndrome is thought to be bleeding into inner ear, followed by disturbance of fluid transport. Labyrinthine concussion is diagnosed by a non-persistent hearing or labyrinthine disturbance which follows a head injury, not caused by another mechanism. Cervical vertigo was first documented by Claude Bernard in 1858[65, 66]. Several theories have reported vascular compression and alteration of sensory input to the vestibular system resulting in vertigo in cervical pathology. This could explain the mechanisms of vertigo in our cervical spondylosis, but further studies would be needed to confirm this. Radanov et al[65] reported dizziness in 20-60% of patients with cervical injury, although recovery occurred in about 75% by one year, aches and pains may persist in 20 to 45%. Degenerative problems develop after injury in about 40% of patients[62, 65, 66].

Postoperative vertigo accounted for 6.7% of cases, this was prominent in cases of severe mastoid abscess and petrositis before surgery and also the use low – speed bone drill which tend to cause significant vibration of the labyrinth. Postoperative vertigo was found in 9 out of 37 modified radical mastoidectomy done in the year under review accounting for 24%.

In conclusion , the diagnosis of vertigo still remains a major challenge in the management, the high proportion of allergy, ageing and idiopathic cases among the non-traumatic aetiology suggest a need for immunogenetic study. The predominance of peripheral vestibular pathology as causes of vertigo may be a reflection of referral pattern to the otorhinolaryngology clinic; an inter-departmental review involving neurology, neurosurgery, endocrinology and ophthalmology may be needed to be able to compare the relative prevalence of the various causes.

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FIGURE 1: AGE DISTRIBUTION OF PATIENTS

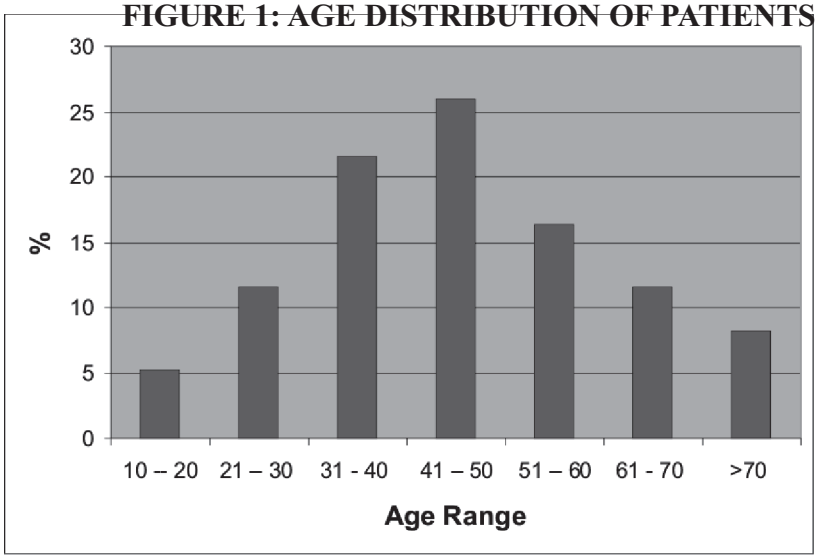

Table 1: Clinical diagnosis and aetiological factors of Vertigo

CLINICAL DIAGNOSIS n= 134	AETIOLOGICAL FACTOR
BPPV 33 (25%)	Trauma 21(16%) ;Idiopathic 6(4.5%) Ageing 6(4.5%)
Labyrinthitis 23 (17%)	Bacterial Otitis media 12 (9%) Measles 4 (3%); Mumps 3 (2.2%) Trauma 2(1.5%); Idiopathic 2(1.5%)
Menieres disease 19 (14%)	Trauma 14(10.5%); Idiopathic 5(3.7%)
cervical spondylosis 14 (10.5%)	Allergy 3(2.2%) ; Trauma 5(3.7%) Virus 3(2.2%) ; Idiopathic 3(2.2%)
Vestibular Neuronitis 10 (7.5%)	Virus 8(6%) ; Idiopathic 2(1.5%)
Presbystasis 7 (5.2%)	Ageing 7(5.2%)
Autoimmune inner-ear disease 6 (4.5%)	Idiopathic 6(4.5%)
Otoxicity 4 (3%)	Chloramphenicol 3 (2.2%); Genticin 1 (1%)
Labyrinthine concussion 3 (2.2%)	Trauma 3(2.2%)
Temporal bone fracture 2 (1.5%)	Trauma 2 (1.5%)
Acoustic Neuroma 2 (1.5%)	Idiopathic 2(1.5%)
Postoperative vertigo	9 (6.7%)
TOTAL	**134 (100%)**

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Appendix

Definitions

Mennieres Disease – Presence of episodic vertigo, tinnitus and hearing impairment with or without nausea, anorexia and vomiting and audiometry showing low frequency hearing loss.

Labyrinthitis - Presence of sudden, progressive, or fluctuating vertigo with hearing loss or other auditory symptoms.

Vestibular neuronitis - Vertigo without auditory symptoms lasting for several days with or without a prodrome of upper respiratory tract illness.

Benign Paroxysmal Positional Vertigo - Brief episodes of vertigo that appears in specific head positions with positive positional tests.

Cervical Vertigo – Presence of vertigo, neck pain or discomfort with radiologic evidence of cervical osteophytes

Autoimmune inner-ear disease – Presence of progressive, bilateral /unilateral vestibular hypofunction and hearing loss in addition to raised ESR with or without immune factors.

Presbystasis – Presence of momentary positional vertigo or generalized unsteadiness in any

direction in an elderly patient with associated age changes such as poor vision, arthritis in the absence of other causes.

Measles infection: Presence of fever, skin rashes with or without other constitutional symptoms.

Mumps: presence of unilateral or bilateral parotid swelling with or without fever.

Meningitis: Presence of fever and neck stiffness or we rely on the diagnosis of the physician who managed the patient before referring to our clinic.

Ototoxicity: History of consumption of a drug in normal or high doses for at least one week particularly known ototoxic drugs whether prescribed by a physician or quack..