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Original Research article

Demography, aetiology, and positional test findings of 187 consecutive patients diagnosed as having BPPV at ENT unit, Teaching Hospital Anuradhapura

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Abstract

Introduction

Benign Paroxysmal Positional Vertigo (BPPV) is the most common vestibular disorder encountered in balance clinics. Diagnosis by Dix-Hallpike test is straightforward. However, diagnosis can still be complicated due to the presence of number of different subtypes and negative findings. Objectives of the study are to determine demographic pattern, positional test findings in patients diagnosed as having BPPV and to discuss the reasons for positional vertigo with negative physical examination findings. A subgroup analysis was done from a cross sectional descriptive study that was carried out at ENT clinic at Teaching Hospital Anuradhapura from 1st of March 2016 to 28th February 2017. Hundred and eighty-seven (187) consecutive patients with symptoms suggestive of BPPV were assessed .Posterior semi-circular canal involvement as the commonest variant with 70% diagnosed without video Frenzels. Although symptomatic during positional testing, we could not demonstrate clear nystagmus in 24.6% patients in light. Geotrophic or apogeotrophic horizontal nystagmus on supine roll test and down beating nystagmus on Dix-Hallpike test were found 4.2% and 1% respectively.

Our findings on posterior canal and positional test negative BPPV are compatible with previously published data. However, diagnosis of lateral appears to be still inadequate. Early referral, routine use of video Frenzel recording at clinics, routine use of seated pitch test, repeated testing may help in reducing false negative rates and finding a specific diagnosis.

Key words: BPPV, Vertigo, Positional tests, Nystagmus, Anuradhapura

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Introduction.

Benign paroxysmal positional vertigo (BPPV) is the commonest peripheral vertigo syndrome having a life time prevalence of 2.4%[1,2]. BPPV can be diagnosed up to about one-third of vertigo presenting with dizziness[2,3].One of our previous studies has shown that 36.5% of dizziness patients presented to a tertiary hospital setting in Sri Lanka have got the diagnosis of BPPV[4]. The condition is characterized by short-lasting vertigo episodes (typically less than 1 minute) which are induced by a change in head position in relation to gravity[5].These episodes are typically produced by looking up, looking down, lying supine on the bed or getting up from supine position or rolling over on the bed[5]. Less commonly patients can present with imbalance[6], but detailed history usually reveals associated positional vertigo.

BPPV occurs following inappropriate stimulation of hair cells in semi-circular canals (SCC) in response to changes in head position with respect to gravity, by sequestered otoconia. Otoconia are calcium carbonate crystals, normally found embedded in the gelatinous otolithic membranes of the utricle and saccule. When free floating otoconia dislodge into semi-circular canals (canalolithiasis) or attach themselves to cupula of SCC (cupulolithiasis)[7], changes of head position will result in displacement of cupula directly or indirectly via change in endolymphatic fluid pressure[8]. Same phenomenon can occur when specific gravity of endolymph changes relative to the cupula[9,8] (heavy or light cupula). This results in vertigo and nystagmus. In cases where otoconia in posterior or anterior SCC, the nystagmus will be vertical- torsional. In contrast, the nystagmus will be horizontal in lateral SCC[8]

Posterior canal BPPV accounts for about 60-90% of the cases and lateral canal BPPV accounts for about 8-15%[10]. Anterior canal BPPV and involvement of multiple canals are rare (2-5%).

Lateral semi-circular canal BPPV is diagnosed by supine roll test, where the head is rolled to left and then to right when the patient is in supine position with the head raised to 30 degree and nystagmus is observed. Geotropic nystagmus is when the fast phase beats towards the side of head roll and when it beats to the opposite side to the head movement it is named as apo geotropic. Geotropic nystagmus is seen when free floating otoconia is in the long arm of the lateral semi-circular canal (long-arm canalolithiasis) and apo geotropic nystagmus is seen when free floating otoconia is in the short arm (short-arm canalolithiasis) of the lateral canal or when they are embedded in the cupula (cupilolithiasis)[14].

BPPV limits activities of daily living significantly, lateral canal having increased patient-perceived disability[15].

The most cost effective management modality of BPPV is carrying out correct canalith repositioning manoeuvre depending on the sub type of BPPV[16]. This needs proper nystagmus-based diagnosis.

Many population-based studies have been conducted worldwide to recognize the burden of BPPV at dizzy clinics. However, there is no Sri Lankan data available. This analysis was carried out to assess the demography, frequency of different types of BPPV, its aetiology, associations and management of patients diagnosed as having BPPV at the Ear, Nose and Throat (ENT) clinic at the teaching hospital, Anuradhapura in the North Central province of Sri Lanka. This was carried out as a subgroup analysis of a previously published study on prevalence of vestibular disorders in Sri Lankan Population.

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Material and Methods

This study was done as a part of a cross sectional descriptive study that was conducted at Ear nose and throat clinic at Teaching Hospital Anuradhapura during one-year period from 01/03/ 2016 to 28/02/2017. The study sample included all the patients presented to the ENT clinic with short-lasting positional vertigo/dizziness and diagnosed as having BPPV. A detailed history was taken from all the patients presented with vertigo/dizziness and full vestibular examination had been carried out. Dix-Hallpike maneuver was routinely done on both sides and waited for 30 seconds to observe any nystagmus. Patient was asked if he or she has developed any vertigo while on Dix-Hallpike position and on reversal. The worse symptomatic side was noted when nystagmus was not demonstrated in light. Then supine roll test was done, and same pattern followed. Video Frenzel was not routinely used during examination. All the patients were seen by a consultant Otolaryngologist to arrive at a final diagnosis. Diagnoses were made according to the accepted guidelines as per consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society[8].

Ethical clearance was obtained for the main study from the ethics review committee of Faculty of Medicine and Allied Sciences, Rajarata University of Sri Lanka.

Results

A total of 187 patients were diagnosed as having BPPV during this time period. 98 (52.4%) were males. Mean age of presentation was 52.5 years (SD=14.1).

Age and sex distribution are given in table 1

Age (years)	N	%
<12	0	0
12-19	01	0.5
20-39	38	20.3
40-59	92	49.2
>59	56	29.9
Sex		
Male	98	52.4
Female	89	47.6

TABLE 1 :Age and sex distribution

Of these 187 patients, 33 (17.6%) gave a recent history of accidental blunt trauma to ear. One patient (0.6%) developed posterior canal BPPV following stapedotomy of the same side ear and another 4 patients (2.1%) had a history of vestibular migraine. Idiopathic BPPV was diagnosed for 149 patients (table 1).

Aetiology	N	%
Accidental blunt trauma	33	17.6
Vestibular migraine	4	2.1
Post Stapedotomy	1	0.005
Idiopathic	149	79.7

TABLE 2: Aetiology of BPPV among the study participants

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141 (75.4%) patients showed nystagmus during one of the positional tests enabling us to diagnose BPPV with confidence (table 2). However, forty-six (24.6%) patients became vertiginous during Dix-Hallpike test without positional nystagmus and their supine roll test were also negative. They were included into a category of positional test negative BPPV after excluding neurological and other otological causes by detailed history, clinical examination and investigations. They also improved symptomatically by single or multiple Epley's canalith-repositioning manoeuvres. 8 (4.2%) patients had horizontal nystagmus on supine roll test (geotrophoc or apogeotrophic) and diagnosed as having lateral canal BPPV. All showed clinical resolution with Lempert canalith repositioning manoeuvre or forced prolonged positioning or combination. Two patients had positional downbeat nystagmus on Dix-Hallpike manoeuvre suggesting either superior canal BPPV or Apo geotropic posterior canal BPPV.

Findings on positional tests	**Diagnosis**	**N**	**%**
Geotropic torsional up beating nystagmus	Posterior canal BPPV	131	70.00
No gross nystagmus	Positional test negative BPPV	46	24.50
Geotropic or apo geotropic horizontal nystagmus	Lateral canal BPPV	8	4.20
Down beating nystagmus	Superior canal BPPV	2	1.06

TABLE 3:Positional test findings BPPV among the study participants

Discussion

Posterior semi-circular canal BPPV is the commonest variant accounting for 60-90% cases depending on the study[8,17]. In our study 70% of the patients were diagnosed as having posterior semi-circular canal BPPV by demonstrating geotropic torsional upbeat nystagmus at Dix-Hallpike test.

Although positional tests are co considered as diagnostic tests in diagnosing BPPV[18], we have seen 46 patients (24.6%) with positional short-lasting vertigo only became vertiginous but did not develop any nystagmus on positional tests. Prevalence of positional test negative BPPV has been reported as ranging from 20-26% according to previous studies[19] and our data is also confirms this. One study showed that about half of the patient who did not show nystagmus on initial examination managed to demonstrate a positive Dix- Hallpike test, usually within a follow up period of 10 days[20]. Another study managed to demonstrate positive findings in 11 out of 13 patients with positional test negative BPPV after head shake manoeuvre[21].

The Dix-Hallpike manoeuvre has a positive predictive value of 83% and negative predictive value of 52% for the diagnosis of posterior and anterior semi-circular canal BPPV[1]. Therefore, negative test does not rule out the diagnosis of BPPV. Those with negative Dix Hallpike test should have a repeated test done at another setting or repeated after head shaking manoeuvre to reduce false negative rate.

The results of positional tests can be affected by technical factors like the speed of the maneuver and the plane of head movement. In our study, performing positional tests on each patient on two different occasions by two different examiners minimized this performance bias.

Secondly it has been suggested that patients with short-arm canalolithiasis of the posterior canal would not show nystagmus during Dix Hallpike test but vertigo/body sway during getting up from the Dix Hallpike position[22,12].Prevalence of this 'subjective' vertigo seems to around 20% and they

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respond to multiple Epley or similar manoeuvres[22]. We have noticed this phenomenon commonly in our cohort and some of those patients would have had this diagnosis.

Lateral semi-circular canal BPPV (LSCC BPPV) was diagnosed only in 8(4.2%) patients by demonstrating horizontal nystagmus (geotropic or apogeotrophic) at supine roll test. However, prevalence of LSSC BPPV has been described as high as 5-30% [8,17,10]. Its known that LSSC BPPV can remit spontaneously compared to PSSC BPPV as a result of sleeping on one side or the other and therefore prevalence of LSSC BPPV tends to be less prevalent in late presenters. Presentation of BPPV patients to ENT department is delayed in most of the times in Sri Lankan setting as BPPV is managed by medical specialties and general practitioners. Further, supine role test is usually done after Dix Hallpike test when the latter is negative. However newer protocols suggest looking for pseudo-spontaneous nystagmus, carrying out seated pitch tests (bow-and-lean test) with video Frenzel recording before proceeding to supine roll test (supine yaw test) for better sensitivity[10,23]. This type of a paradigm and repeated testing may uncover more subtle forms of LSCC BPPV.

Two patients (1.06%) had down beating nystagmus and positional down beating nystagmus has been documented in about 1-2% of dizzy patients in the literature[8,24]. Our first patient had severe vegetative symptoms. This patient eventually responded to combination of Yacovino and Epley maneuvers and it was difficulty to know which one resulted in cure.

The second patient had persistent (no adaptation) down beating nystagmus on Dix Hallpike test which did not respond to any of the correcting maneuvers. Neurology referral and MRI did not find any neurological cause and nystagmus settled over time. This suggests cupulolithiasis of posterior canals or heavy cupula as the etiology[9].

There are few possibilities that one should consider when down beating torsional nystagmus is observed during Dix-Hallpike maneuver. Firstly, anterior canal BPPV, where stimulation of anterior canal results in down beating torsional nystagmus. Here down beating nystagmus is seen bilaterally and the torsional component is usually not prominent. Torsional component should direct to the affected ear when it is present. Down beating nystagmus is seen bilaterally because anterior canals are oriented more parallel to the sagittal plane[24] (smaller angle,41^0 with sagittal plane compared to posterior canals, 56^0). The down beating nystagmus of anterior canal BPPV is also evident in supine head-hanging position therefore (deep head-hanging test). This position would stimulate both the anterior canals at the same time. These patients respond very well to the modified Yacovino maneuver[25] where the patient is bought back to the sitting position and then head is flexed, making chin to touch the chest. Recording of nystagmus with video Frenzels during the test is very important in these patients as some of these patients may respond to sitting up position after the head hanging test.

The second possibility is positional down beating torsional nystagmus due to inhibition of the posterior canal BPPV. Inhibition occurs due to ampullopeatal endolymph movement according to Ewald's third law. This may be due to canalolithiasis in the non- ampullary segment of the long arm of the posterior canal close to the common crus[13]. Once again down beating torsional nystagmus is bilateral, but stronger when the affected ear is down, but torsional component rotates towards the normal unaffected uppermost ear[26]. These patients will have more vegetative symptoms and they may respond to the Yacovino maneuver or its modifications[12]. Sometimes, Yacovino maneuver might convert non- ampullary posterior canal BPPV to classic posterior canal BPPV with upbeat torsional nystagmus, which can be then cured by Epley's maneuver. We believe our first patient had this variety.

Sometimes nystagmus can be persistent with virtually no adaptation. This can be explained by or cupulolithiasis/heavy cupula which bends away from the utricle which has the same inhibitory action[12] but nystagmus would be long lasting or persistent. Possibly our second patient with persistent down beating nystagmus had this variety.

Finally, it is extremely important to rule out central pathologies like Chiari malformation, Spino-cerebellar degeneration, Posterior fossa pathologies when positional downbeat nystagmus is observed, especially when it does not show adaptation and fatiguability[27].

Conclusion

Our findings on posterior canal, positional test negative(subjective) and even superior canal BPPV are compatible with previously published data. However, diagnosis of lateral canal BPPV appears to be sill inadequate. Early referral and routine use of pseudo-spontaneous nystagmus and carrying out seated pitch tests (bow-and-lean test) with video Frenzel recording before proceeding to supine roll test may diagnose more cases of lateral canal BPPV. Early positional tests with video Frenzel recording may detect more positional downbeat nystagmus and subtle forms of nystagmus too.

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