Is Posttraumatic Benign Paroxysmal Positional Vertigo Different From the Idiopathic Form?

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Background: Although head trauma is considered a common cause of benign paroxysmal positional vertigo (BPPV), clinical presentation and outcome of traumatic BPPV (t-BPPV) have not been systematically evaluated.

Objectives: To compare the clinical presentation, patient’s response to physical treatment, and outcome of patients with t-BPPV with those with the idiopathic form (i-BPPV).

Setting: Tertiary referral neuro-otology outpatient clinic.

Methods: We reviewed the clinical records of 247 consecutive patients with posterior canal BPPV during the years 1997 to 2000. All patients were diagnosed using the Dix-Hallpike test and treated using the particle repositioning maneuver. Patients with an onset of positional vertigo within 3 days of well-documented head trauma were included in the t-BPPV group. The outcome was compared with the outcome of 42 patients with i-BPPV who were similarly treated and followed up.

Results: Twenty-one (8.5%) of the 247 patients with BPPV fulfilled the diagnostic criteria for t-BPPV. The most common cause of head trauma was motor vehicle crash, documented in 57% of the cases; half of the patients additionally suffered from a whiplash injury. While the other causes were diverse, common falls were predominant. Only 2 of the patients involved in motor vehicle crashes experienced brief loss of consciousness. Sixty-seven percent of patients with t-BPPV required repeated physical treatments for complete resolution of signs and symptoms in comparison to 14% of patients with i-BPPV ($P<.001$). During a mean ±SD follow-up of 21.7±9.7 months, 57% of t-BPPV patients and 19% of i-BPPV controls had recurrent attacks ($P<.004$).

Conclusions: The nature and severity of the traumas causing t-BPPV are diverse, ranging from minor head injuries to more severe head and neck trauma with brief loss of consciousness. It appears that t-BPPV is more difficult to treat than i-BPPV, and also has a greater tendency to recur.

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BENIGN PAROXYSMAL POSIT- TIONAL vertigo (BPPV) is a very common form of vertigo caused by dislodged otoconia making its way from the utricle mainly into the posterior semicircular canal. In 5% to 22% of cases, the otoconia will be displaced into the horizontal canal, or more rarely, the anterior canal.1,2 The majority of patients experience attacks of rotational vertigo lasting 10 to 30 seconds that are precipitated by lying down, sitting up, turning over in bed, looking up, or bending forward. Prolonged and disabling sensations of drunkenness and light-headedness that may cause falls are also frequently reported; thus, the designation “benign” for this condition in such patients seems somewhat inappropriate.

A diagnosis of BPPV stemming from the posterior semicircular canal can be easily established at bedside by the Dix-Hallpike positional test (placing the patient in the lateral head-hanging position elicits the typical attack of vertigo accompanied by transient, up-beating, torsional nystagmus with the upper poles of the eyes beating toward the undermost affected ear).3 Establishing a diagnosis of BPPV is beneficial since it is treated by relatively simple physical maneuvers without the need for additional investigations or drug therapy. About 80% of patients with posterior canal BPPV will be free of symptoms and signs following a single physical maneuver.6-10

In several publications4,11,12 based on a large number of patients with BBPV, the cause of otoconia detachment could not be established in 34% to 66% of cases (idiopathic, or primary, BPPV). In the acquired, or secondary, form, head trauma and inner ear diseases such as viral neurolabyrinthitis and Meniere disease were considered the most common causes. None of these stud-
ies proposed definite criteria for traumatic BPPV (t-BPPV) or attempted to delineate differences in the presentation and clinical course of t-BPPV and idiopathic BPPV (i-BPPV).

Those studies that specifically mention BPPV as one of the possible causes of posttraumatic dizziness or vertigo are also lacking diagnostic clarity. In a study of 100 patients with dizziness following head trauma, a typical history of BPPV was obtained in 61 patients, but in only 25% were the diagnoses confirmed by a positional test. Similar to the other studies mentioned above, neither mode of treatment nor outcome were given.

In the last few years we had the impression that the course of t-BPPV is more severe than i-BPPV; however, this was not systematically studied. The aim of the present report is to describe the clinical presentation, the response to physical treatment, and the outcome in a series of patients with t-BPPV as compared with i-BPPV.

**METHODS**

We reviewed the medical records of 247 patients with posterior canal BPPV who were seen in our tertiary referral neuro-otology outpatient clinic during the years 1997 to 2000. Each patient underwent a complete neurological examination and received a detailed ocular motor and neuro-otological examination according to Zee and Fletcher. This included eye movement testing (alignment, range of motion, vergence, saccades, smooth pursuit, optokinetic nystagmus, and visual cancellation of the vestibulo-ocular reflex [VOR];) evaluation of spontaneous nystagmus with and without visual fixation (using Frenzel lenses or ophthalmoscopy with the other eye occluded to prevent fixation); evaluation of dynamic VOR function (dynamic visual acuity, head thrust or impulse test, ophthalmoscopy during head rotation, and assessment for the presence of nystagmus following prolonged head shaking); and a positioning test for posterior and horizontal canal BPPV.

All 247 patients had clinical features compatible with posterior canal BPPV that were confirmed by the Dix-Hallpike test. Only patients in whom the onset of positional vertigo was within 3 days of well-documented head trauma were diagnosed with t-BPPV. Included in the i-BPPV group were patients without a history of head trauma who were similarly treated and followed up in our neuro-otology clinic during the same period of time (2 idiopathic cases for each t-BPPV patient). Patients with a history of inner ear disease, migraines, or cerebrovascular disease were not included in either group. Following the confirmation of a BPPV diagnosis, all patients were immediately treated by a single particle repositioning maneuver (PRM). Patients were instructed to avoid bending over and to stay in the upright position for the remainder of the day (8-10 hours). A repeated examination 1 week later, which is the routine in our clinic, was performed to determine the PRM efficacy. When the results from the Dix-Hallpike test were positive for vertigo, PRM was performed and the patient was reexamined after 1 week. Once patients tested negative for vertigo, they were reexamined after 3, 6, and 12 months, and then contacted by telephone for further outcome information. All patients were treated identically and were urged to contact our clinic and arrange an immediate office visit if they had recurrence of positional vertigo.

**RESULTS**

Twenty-one (8.5%) of the 247 patients with BPPV fulfilled the diagnostic and historical criteria for t-BPPV. The clinical features and response to physical treatment of these patients are summarized in the **Table**. There were 11 women and 10 men; the mean ±SD age was 56.3 ± 15.6 years (range, 24-84 years). The control group of 42 subjects with i-BPPV included 32 women and 10 men; the mean ±SD age was 56.3 ± 15.6 years.
age was 61.1±22.3 years (range, 34-83 years). Except for testing positive for vertigo using the Dix-Hallpike test, in all 63 subjects the findings from the complete neurological, ocular motor, and neuro-otological examinations were normal and brain and inner ear computed tomographic scans or magnetic resonance imaging were unremarkable.

In 12 cases, the causes of head trauma were motor vehicle crash (MVC); 6 of the patients had additional whiplash injuries. The other causes were diverse (fall, blow to the head, ventriculoperitoneal [VP] shunting, a violent speedboat trip, removal of occipital osteoma, and dental surgery). Two of the patients with MVC head trauma experienced brief loss of consciousness.

Only 2 patients were diagnosed with BPPV prior to the referral to our clinic. The other 19 received other diagnoses, such as unspecified or posttraumatic dizziness (8), whiplash-associated dizziness (6), cervical vertigo (4), and transient ischemic attack (1). When first examined, 2 patients had paroxysmal horizontal canal apogeotropic nystagmus, which was more intense when the affected ear was uppermost. Both were immediately treated with a single 270° rotation around the supine patient’s yaw axis (“barbecue maneuver”) in the direction of the healthy ear. The reexamined 24 hours later, a typical posterior canal BPPV was apparent and treated accordingly. These 2 patients were also reexamined 72 hours after treatment.

Four patients (19%) had bilateral BPPV (both ears simultaneously affected). These patients were randomly treated for 1 ear at the first visit and for the other ear 72 hours later.

Thirty-six (86%) of the 42 unilateral i-BPPV controls had complete resolution of symptoms and signs (results were negative for vertigo on the Dix-Hallpike test) following a single PRM. Seven patients (9 of 25 ears; 36%) with t-BPPV (33%) were free of symptoms and signs after a single PRM. The proportion of success after a single treatment to an affected ear was significantly different in the 2 groups (P<.001, Fisher exact test).

During the 6- to 42-month follow-up (mean±SD, 21.7±9.7 months), 12 patients (14 of 25 ears) with t-BPPV (57%) and 8 with i-BPPV (19%) had recurrent attacks of BPPV (P<.004, Fisher exact test). Three of 7 t-BPPV patients (42%) responded successfully with a single PRM and 9 of 14 (64%) who required multiple PRMs had recurrence (Table). This difference was not statistically significant (P=.53).

**COMMENT**

This group of carefully documented patients with t-BPPV represents only 8.5% of our patient population with BPPV evaluated between 1997 to 2000. The nature and severity of the traumas were diverse and included common minor blows to the head from a football or during a fall, as well as relatively moderate-severe head and neck trauma with brief loss of consciousness during MVCs. There were also unusual causes, such as taking a violently shaky speedboat trip, drilling of a burr hole for insertion of a VP shunt, and traumatic dental surgery using a hammer and a chisel. Interestingly, bone surgery with a hammer and a chisel for removal of an occipital osteoma performed in 1 of our patients was previously reported as an unusual cause of BPPV.

There is a general agreement that t-BPPV accounts for 15% to 20% of all BPPV cases. There may be several reasons why there was a lower frequency of t-BPPV in our series (8.5%). Our patient population is composed of a highly selective group referred to a tertiary referral center, while patients sustaining trauma during MVCs or occupation-related trauma are routinely referred directly to the emergency department. Moreover, choosing an inclusion criterion of 3 days posttrauma for t-BPPV may explain in part its infrequent occurrence among our patients with BPPV. The exclusion of patients with a history of inner ear disease, migraine, and cerebrovascular disease may also affect our results. In several studies, inner ear diseases accounted for 8% to 39% of cases with BPPV. However in a more recent study of 2847 patients with BPPV, only 81 (3%) had definite BPPV secondary to a documented ipsilateral inner ear disease. Interestingly, in this particular study, BPPV that followed trauma without objective evidence of inner ear damage was classified as idiopathic, or primary.

Only 2 of our 21 patients were diagnosed with BPPV prior to referral. The most common diagnoses prior to referral were unspecified traumatic dizziness, whiplash-associated dizziness, and cervical vertigo. In fact, there is no clinical evidence that cervical injuries alone can elicit nystagmus with consequent vertigo. Interestingly, in this particular study, BPPV that followed trauma without objective evidence of inner ear damage was classified as idiopathic, or primary.

Two of our patients had horizontal apogeotropic nystagmus converted to typical posterior BPPV after a single “barbecue maneuver.” Horizontal BPPV following trauma has only been mentioned and not specifically characterized. Conversion from horizontal to posterior canal BPPV has also been described, especially following treatment with the “forced prolonged position.” Lying down for many hours with the healthy ear down.

Similar to previous publications, a considerable number of our t-BPPV patients (19%) had bilateral BPPV (both ears simultaneously affected) while all our i-BPPV patients had unilateral BPPV. The frequency of bilateral i-BPPV recorded in our clinic is approximately 2.5%, and the response to a single PRM per ear is similar to unilateral cases. Katsarkas found bilateral involvement in 14.3% of t-BPPV cases compared with 6.3% in the idiopathic group. In his traumatic group the number of women was equal to the number of men, as was the case in our patients. The higher proportion of bilateral involvement and the lack of female patients prevalent in the traumatic group are in favor of the assumption that t-BPPV is different from i-BPPV.

A suggestion that the clinical course of t-BPPV may differ from the idiopathic form was previously made but not thoroughly evaluated. Our data indicate that t-BPPV is significantly more difficult to treat than i-BPPV. Sixty-
seven percent of cases needed repeated physical treatment until complete BPPV resolution, in comparison with 14% of patients with i-BPPV. Moreover, during a mean follow-up of 22 months, recurrence was significantly more common in t-BPPV (57%) than in idiopathic cases (19%). Patients who required multiple PRMs seemed to have more recurrences (64%) than those who were successfully treated with one maneuver (42%); however, this difference was not statistically significant.

Epley8 reported an overall 30% recurrence of BPPV during a 30-month follow-up, while Fung and Hall26 found that following a successful physical treatment, 34% of cases had recurrence after a mean follow-up of 19 months. Recently, Nunez et al27 recorded a recurrence of 26.8% during a mean follow-up of 26 months and calculated a yearly recurrent rate of 1.5%. For our entire group of 226 unselected non–t-BPPV patients, we have calculated a 13% yearly recurrence following a successful treatment.

It is conceivable that resistance to treatment and the frequent recurrence of t-BPPV is due to trauma. Otoconia are detached by trauma, and microscopic hemorrhages, or “tissue shearing,” result in biochemical changes that enhance the formation of otoconial clots. Following a successful maneuver, these microscopic changes may reactivate the production of new clots, accounting for the recurrence of BPPV.

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