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The Risk of Benign Paroxysmal Positional Vertigo After Head Trauma

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Objectives: Head trauma may cause dislodgement of otoconia and development of benign paroxysmal positional vertigo (BPPV). The risk of developing BPPV is expected to be highest shortly after the trauma, then decrease and approach the risk seen in the general population. The aim of this study was to estimate the risk-time curve of BPPV development after head trauma.

Study Design: Prospective observational study.

Methods: Patients with minimal, mild, or moderate head trauma treated at the Department of Neurosurgery or the Department of Orthopedic Emergency at Oslo University Hospital, were interviewed and examined for BPPV using the Dix-Hallpike and supine roll maneuvers. BPPV was diagnosed according to the International diagnostic criteria of the Bárány Society. Telephone interviews were conducted at 2, 6, and 12 weeks after the first examination.

Results: Out of 117 patients, 21% developed traumatic BPPV within 3 months after the trauma. The corresponding numbers were 12% with minimal trauma, 24% with mild, and 40% with moderate trauma. The difference in prevalence between the groups was significant ($P = .018$). During the first 4 weeks after the trauma, it was observed 20, 3, 0, and 1 BPPV onsets, respectively. No BPPV cases were seen for the remainder of the 3-month follow-up.

Conclusion: The risk of developing BPPV after minimal-to-moderate head trauma is considerable and related to trauma severity. Most cases occur within few days after the trauma, but any BPPV occurring within the first 2 weeks after head trauma are likely due to the traumatic event.

Key Words: Benign paroxysmal positional vertigo, head trauma, traumatic benign paroxysmal positional vertigo, time criteria.

Level of Evidence: 3

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INTRODUCTION

Head traumas constitute a fundamental source of death and disability and causes significant suffering for the affected. This can be measured using disability-adjusted life years (DALYs).[1] Dizziness and vertigo are among the most frequent symptoms after head trauma,[2,3] causing distress, deterioration in quality of life, delayed return to work, socioeconomic costs, and in some cases long-term disability up to several years after the injury.[4–6]

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular disorder after head trauma.[7–9] Traumatic BPPV (t-BPPV) may develop if the forces acting upon the skull during the trauma cause otoconia to dislodge from the utricular macula.[10] Other mechanisms may be detachment of otoconia due to tearing or microscopic hemorrhages in the membranous labyrinth.[11] Positional vertigo results from otoconia falling from the utricle into the semicircular canals.[12] Due to the orientation of the semicircular canals in the head, this is most likely to happen during bedrest, and the first attack usually occurs while turning over or sitting up in bed.

BPPV typically causes recurrent, short lasting attacks of vertigo provoked by changes in head position, such as lying down, turning over in supine position, tilting the head backward or forward, and straightening up after bending over.[12,13]

The diagnosis of t-BPPV is based on BPPV being confirmed shortly after a well-documented head trauma. The elapsed time between the trauma and the onset of positional vertigo is critical. Most studies on t-BPPV apply a time criterion, which may range from a few days[11,14,15] up to 3 months.[16] However, there is a lack of clinical studies to inform the choice of such time criteria, and in

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medico-legal cases involving t-BPPV the time criterion is a point of dispute.

The aims of this study were to estimate the prevalence of BPPV after head traumas of different severity and to establish a risk-time curve for developing BPPV the first 3 months after the trauma.

MATERIALS AND METHODS

Design

This was a prospective study of 117 adults (≥18 years) with head trauma, treated at the Department of Neurosurgery or the Department of Orthopedic Emergency at Oslo University Hospital between August 2019 and March 2020.

Ethics

Participation was based on informed consent and the study protocol was approved in advance by the Regional Committee for Medical and Health Research Ethics of Northern Norway (reference number 2019/400) and by the Data Protection Officer of Oslo University Hospital (reference number 19/05879).

Material

Inclusion criterium was head injury categorized according to the Head Injury Severity Score (HISS) as minimal (Glasgow coma scale (GCS) 15 and no loss of consciousness or amnesia), mild (GCS 14 or 15 and brief (<5 minutes) loss of consciousness or amnesia), and moderate (GCS 9–13 and/or loss of consciousness ≥5 minutes or focal neurological deficits). Exclusion criteria were severe head injury (GCS ≤8),[17] dizziness or balance problems within the previous 4 weeks before the trauma, known otovestibular or neurological disorders causing dizziness or balance problems, consent incompetency, dementia, and substance abuse as well as neck injury preventing diagnostic/therapeutic maneuvers.

Inclusion

Patients were included from two different units at Oslo University Hospital. The Orthopedic Emergency Department admits all types of injuries in Oslo, including minimal and mild head traumas. The Department of Neurosurgery admits all degrees of head trauma, but particularly the more severely injured patients where surgery may be necessary. Patients were included regardless of whether vertigo was present or not, and they were examined as soon as practically and medically feasible after the injury. The median time between trauma and the first examination was 5 days (range 0–20 days).

TABLE II.
Univariate Logistic Regression Analyses of t-BPPV Risk Based on Trauma Severity, Age, and Sex.

	P-Value	Odds Ratio	Confidence Interval
Trauma group			
Mild vs. minimal	.125	2.3	0.8–6.7
Moderate vs. minimal	.018	5.0	1.3–19.1
Age ≥ 50 years	.162	0.5	0.2–1.3
Sex (male)	.981	1.0	0.4–2.5

t-BPPV = traumatic benign paroxysmal positional vertigo.

Diagnostic Procedure

The diagnostic procedure started with an interview with particular attention to the presence, onset, and characteristics of vertigo or dizziness; duration of attacks; and relation to head movements of different types. The interview was followed by an examination for nystagmus using infrared video-goggles with the Dix-Hallpike[18] and supine roll maneuvers.[19] Dizziness or vertigo during the maneuvers was noted. Before the examination, participants were instructed to stay in a head-upright position for at least 30 minutes. If the patient had consumed alcohol the previous 12 hours before examination, the examination was postponed. Dix-Hallpike maneuver was performed first by laying the participant supine, with the head turned 45° to the side and the neck extended approximately 20°, if possible. The supine roll maneuver was performed by turning the head maximally to one side, then maximally to the other side, and then back to the first side, with the neck flexed approximately 10 to 20°. In both Dix-Hallpike and supine roll maneuvers, every other participant would alternate between turning the head to the right and left first. If no nystagmus occurred, each position was held for 20 to 30 seconds. If nystagmus persisted, the position was held for approximately 1 minute.

BPPV was diagnosed according to the Bárány Society diagnostic criteria for BPPV.[13] Accordingly, BPPV was classified as BPPV in the posterior, horizontal, or anterior canal; lithiasis of multiple canals; probable BPPV spontaneously resolved; and possible BPPV.[13] The criteria are portrayed in the supplementary table S1. Other categories used in this study were unspecific dizziness, exclusively orthostatic dizziness, and no dizziness.

In confirmed cases, BPPV onset was based on carefully interviewing the patient to determine the first experience of

TABLE I.
Clinical Data From Each Head Trauma Group Separately (Minimal, Mild, and Moderate) and for the Patient Group as a Whole.

	Minimal	Mild	Moderate	Total
BPPV	6 (12%)	12 (24%)	6 (40%)	24 (21%)
Probable BPPV spontaneously resolved	1 (2%)	0 (0%)	2 (13%)	3 (3%)
Possible BPPV	8 (16%)	12 (24%)	1 (7%)	21 (18%)
Unspecific dizziness	18 (35%)	19 (37%)	3 (20%)	40 (34%)
Exclusively orthostatic dizziness	0 (0%)	1 (2%)	2 (13%)	3 (3%)
No dizziness	18 (35%)	7 (14%)	1 (7%)	26 (22%)
Total	51 (100%)	51 (100%)	15 (100%)	117 (100%)

BPPV = benign paroxysmal positional vertigo.

TABLE III.
Time of Onset of Definite BPPV Cases (n = 24).

	Instances of Definite BPPV
The same day as trauma	8
1 day after trauma	6
2 days after trauma	2
3 days after trauma	1
4 days after trauma	2
6 days after trauma	1
12 days after trauma	1
14 days after trauma	2
25 days after trauma	1

BPPV = benign paroxysmal positional vertigo.

positional vertigo or dizziness. BPPV cases were classified as unilateral if only one labyrinth was affected, and bilateral if both labyrinths were affected simultaneously. Telephone interviews were conducted at 2, 6, and 12 weeks after the first examination. Patients were also asked to make contact as soon as possible if dizziness happened to occur, change in character, or reoccur. Patients suffering from dizziness at telephone follow-up were offered a new clinical examination, similar to the first. Of these, two-thirds accepted the offer. The most common reasons for declining the offer of a second consultation were insignificant symptoms and practical barriers (e.g., old age, living far away). Confirmed cases were referred to an experienced otoneurologist (one of the coauthors) for treatment and follow-up.

Statistical Methods

Probable and possible BPPV cases were reported, but not included in the statistical analyses. Logistic regression was used to analyze differences in risk of BPPV based on age, gender, and trauma severity. A *P*-value of <.05 was considered statistically significant. The risk-time curve of BPPV was visualized using Kaplan-Meier and smoothed hazard ratio graphs. Data were censored 3 months after the trauma. Statistical analyses were performed using Stata 15.0 (StataCorp, U.S.A.) and R version 4.0.2 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

One hundred fifty-one patients were asked to participate. Twenty-three patients declined participation, while 11 either canceled or did not show up for examination. Although patients were not required to state a cause for nonparticipation, a majority offered an explanation spontaneously, which was noted. The given cause was for the most part practical or health barriers to participation. Health issues included old age or frailty (four cases) or preexisting morbidity (nine cases). Practical barriers were noted in 13 cases. In five cases no explanation was given, and in three cases patients mentioned that they did not feel dizzy at the time.

One hundred seventeen patients were included. Mean age was 50 years (range 18–90 years), and 49 (42%) were women. The trauma severity according to HISS[17] was minimal, mild, and moderate in 51, 51, and 15 cases,

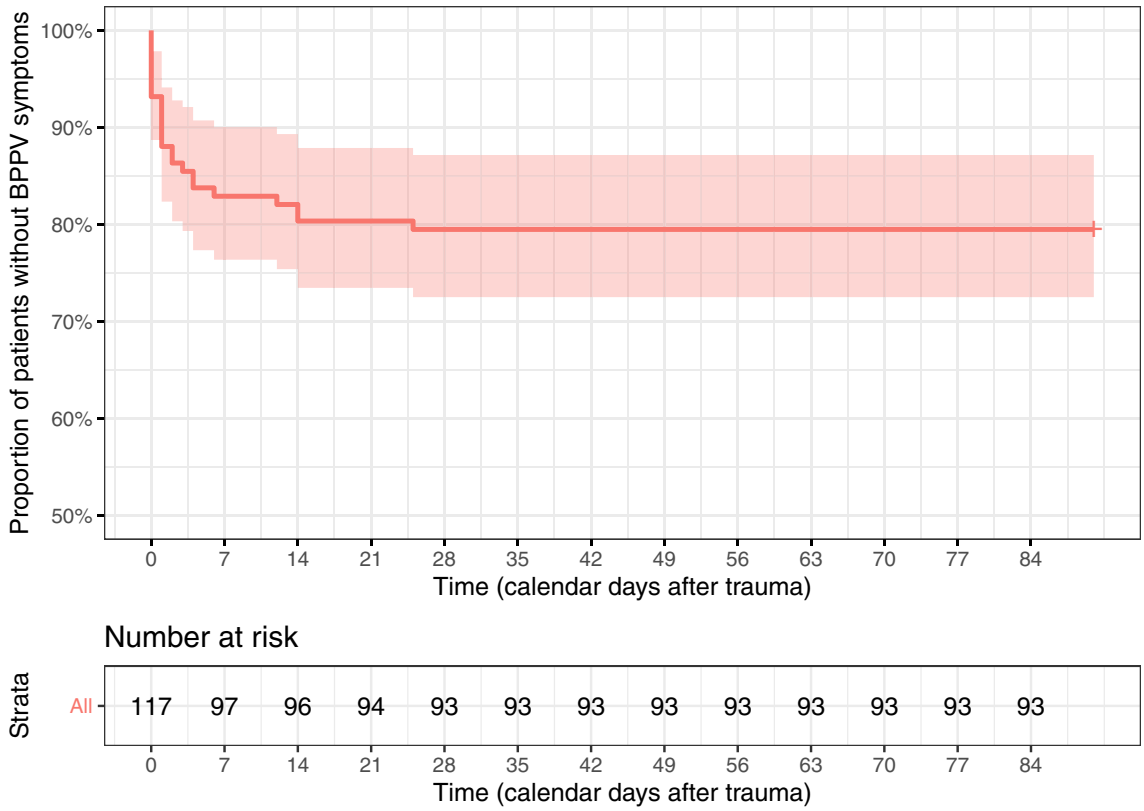

Fig. 1. Kaplan-Meier curve of 117 patients followed for 3 months after head trauma, illustrating the proportion of patients free from definite BPPV. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

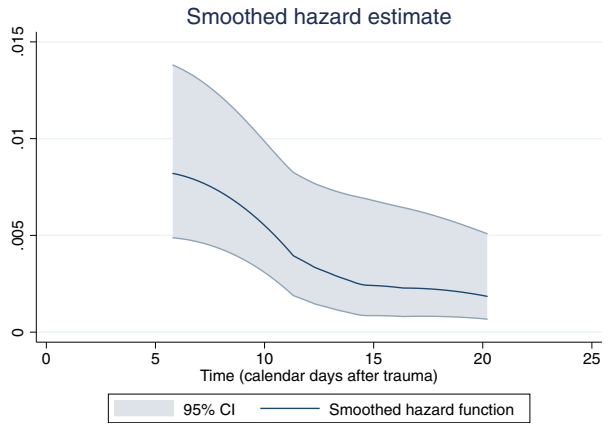

Fig. 2. Smoothed hazard ratio curve including definite BPPV cases from the whole patient group. Illustrating the short-time risk of developing BPPV after head trauma for a patient who has not developed BPPV. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

respectively. The dropout rate was 11% (five patients at 2 weeks, five patients at 6 weeks, and three patients at 12 weeks).

Twenty-four patients (21%, 95% CI: 14%–29%) developed BPPV within 3 months after the trauma. After minimal, mild, and moderate head traumas, BPPV occurred in 6 of 51 (12%, 95% CI 4%–24%), 12 of 51 (24%, 95% CI 13%–37%), and 6 of 15 (40%, 95% CI 16%–68%) cases, respectively. In total, 78% in the entire group experienced some form of dizziness. The prevalence of BPPV and other forms of dizziness are shown in Table I.

The results of the logistic regression analyses are shown in Table II. With minimal head trauma as reference, increased risk of BPPV was associated with moderate head trauma ($P = 0.018$), but not with mild trauma. The risk of BPPV was not associated with age or sex.

The onsets of definite BPPV cases are shown in Table III. The Kaplan-Meier survival curve is shown in Figure 1 and the smoothed hazard ratio curve in Figure 2.

The Kaplan-Meier curve illustrates that the risk of developing t-BPPV is highest shortly after the trauma. All but one of the BPPV cases report onset of characteristic symptoms within 2 weeks after the trauma. The remaining BPPV case had symptom onset 25 days after the trauma.

The smoothed hazard ratio curve shows that the short-time risk of developing t-BPPV is decreasing with time after head trauma and eventually flattens out.

Table IV shows the BPPV subtypes among the definite BPPV cases.

TABLE IV.
Subtypes of BPPV in Patients Who Developed Definite BPPV (n = 24) During 3-Month Follow-up After Minimal-to-Moderate Head Trauma (n = 117).

	Instances of definite BPPV
Posterior canal BPPV	5
Horizontal canal BPPV geotropic direction changing nystagmus	7
Horizontal canal BPPV apogeotropic direction changing nystagmus	2
Anterior canal BPPV	5
Bilateral BPPV	5

Diagnosis according to Bárány Society Criteria.[13]
BPPV = benign paroxysmal positional vertigo.

DISCUSSION

Our results show that 21% (95% CI: 14%–29%) of 117 patients with minimal-to-moderate head injury developed BPPV within a 3-month follow-up. All cases reported an onset of characteristic symptoms within 1 month after the trauma. The risk of BPPV was associated with trauma severity, but not with age or sex. To our knowledge, this is the first study to document the risk-time curve of BPPV development after head injury.

A population-based study estimated the 4-week prevalence of BPPV to 0.7% (95% CI: 0.5%–1.1%) and the 1-year prevalence to 1.6% (95% CI: 1.3%–2.1%).[20] Based on these figures, we loosely estimated the expected prevalence of idiopathic BPPV (i-BPPV) in our 3-month follow-up of 117 patients with a mean age of 50 years to 0.5% to 2%. This was outside the 95% confidence interval for the observed prevalence of BPPV in our study after minimal head trauma (4%–24%). In other words, the risk of t-BPPV was significantly increased after all degrees of head trauma compared with the general population.

There is a lack of epidemiologic studies documenting the risk of BPPV after head trauma. However, such studies often report a traumatic etiology in about 10% of the patients. Józefowicz-Korczyńska et al. studied 179 patients with mild head injury, where 19 patients were diagnosed with BPPV (10.6%).[8] Davies et al. studied 100 head injured patients with vestibular complaints, and 15 were diagnosed with BPPV (15%).[7] Dlugaiczyk et al. studied 74 BPPV patients and attributed this to a traumatic event in 14.9%.[21] Balatsouras et al. diagnosed BPPV in 493 patients at a neurotology department over a period of 7 years and attributed this to a head trauma in 38 of the patients (7.7%).[22] Ernst et al. included 63 patients who complained of vertigo after head or neck trauma and found that 36 of these (57%) had developed BPPV within the first 24 hours after the trauma.[9] A factor contributing to such a high BPPV occurrence could be the exclusive inclusion of patients suffering from vertigo or dizziness after their trauma.

There are currently no internationally accepted diagnostic criteria for t-BPPV. Inclusion criteria for studies on t-BPPV in the scientific literature generally include a cutoff value for the time window between trauma and onset of characteristic positional vertigo. However, this cutoff value has ranged from a few days[11,14,15] to 3 months.[16] One experimental study on mice indicated that otoconia may continue to detach from the utricular macula for several months after vibration to the skull.[23] We cannot exclude the possibility that this may also be the case in humans. However, in our study, the risk of developing definite BPPV was highest shortly after the trauma and

subsequently flattened out. All cases but one occurred within 2 weeks after the trauma. The single case that occurred after 25 days could be due to the trauma, but a sporadic case of i-BPPV was also expected during the follow-up. For this reason, our study indicates that 2 weeks may be a reasonable time window for the diagnosis of t-BPPV. After this, the association with head trauma becomes uncertain.

We found significant BPPV occurrence even after minimal head traumas. This indicates that a low impact injury that causes no loss of consciousness or amnesia can cause dislodgment of otoconia and subsequent BPPV. This is in agreement with a number of studies documenting BPPV after sports activities like running, aerobics, swimming, postural gymnastics, and American football,[24–26] as well as surgery involving vibration to the scull including otologic, dental, and maxilla-facial surgery.[24,27–30]

The typical patient with i-BPPV is >50 years old,[31] and women are affected more often than men.[14,15] However, in our study, the risk of t-BPPV was not influenced by age or gender. This is confirmed by other studies,[11,14,15] which substantiates that the pathophysiology of t-BPPV seems to be different from i-BPPV.

The distribution of subtypes of BPPV in our study differed from the reported distribution in idiopathic cases.[13] In particular, there was a higher prevalence of lateral and anterior canal involvement as well as bilateral cases. The frequent affection of the lateral canal emphasizes the need to perform not only the Dix-Hallpike test but also the supine roll test when examining dizzy head injured patients. Our results are in agreement with previous studies on differences between t-BPPV and i-BPPV.[11,16,22,32]

The main strengths of our study are the prospective study design, low dropout rate, and the inclusion of not only patients who experienced attacks of positional vertigo/dizziness, but also patients with no evident vestibular symptoms. Some BPPV cases caused by head trauma might initially be easily overlooked due to complex and vague symptoms, unless the patient is examined specifically for this diagnosis. Inclusion of patients from two different units of Oslo University Hospital increases the generalizability of the study. The Dix-Hallpike and supine roll maneuvers were video-recorded, which made it possible for the authors to re-examine and discuss cases. We strictly followed the Bárány Society criteria[13] when diagnosing BPPV and classifying BPPV as "probable BPPV spontaneously resolved" and "possible BPPV."

Our study had some limitations. Although probable and possible BPPV are recognized entities in the Bárány Society criteria for BPPV,[13] we chose only to include definite cases in our analyses. Since dizziness after head trauma could be due to other reasons such as central causes, musculoskeletal cervical problems, visual dysfunction, or other vestibular problems, it is in our opinion necessary to use criteria with high specificity when diagnosing BPPV in this setting. Significant underdiagnosis of BPPV was unlikely due to the close follow-up of the patients with regard to vertigo symptoms as well as video documentation of positional nystagmus. Twenty-three patients declined participation, while 11 either canceled or did not show up for examination. The risk of attrition bias was assessed by the authors and found to be low since the reasons given by the patients for nonparticipation were mostly related to practical or health barriers. It should be noted that our results first and foremost can be generalized to minimal, mild, and moderate head traumas, but it is reasonable to assume that serious head traumas may have an even higher occurrence of BPPV. It was not practically and medically feasible to examine this patient group in the acute phase after the trauma. A study investigating the occurrence of BPPV after severe head trauma found that approximately half of the patients with positional vertigo suffered from BPPV.[2]

Because BPPV is such a common finding after head trauma,[7–9,21,22] especially among the more severe head traumas,[2] BPPV should be excluded before attributing vertigo or dizziness to concussion. Gordon et al. concluded that "traumatic BPPV is probably under-recognized or misdiagnosed in clinical practice" and stated that "The Dix-Hallpike maneuver is mandatory in all patients with dizziness and vertigo following head trauma."[33] According to de Clercq et al. and Eric G. Johnson, there is growing support for including a vestibular examination for t-BPPV in the diagnostic workup after all head traumas.[34,35]

Our findings underline the importance of informing, examining, and treating patients for t-BPPV even after minimal head injuries. Routines should be implemented for BPPV testing of all head injured patients with complaints of dizziness. The diagnostic tests for BPPV are simple, noninvasive, and quick to perform. Early detection and treatment can improve quality of life for the patients who develop t-BPPV and decrease the socioeconomic impact of this disorder.

CONCLUSION

Our study found BPPV in approximately one-fifth of patients after minimal-to-moderate head trauma, with the risk increasing with the severity of the injury. The risk of developing t-BPPV was highest immediately after the trauma and subsequently flattened out. Characteristic symptoms occurred within 2 weeks after the trauma in the vast majority of cases. We propose that onset of BPPV symptoms within 2 weeks after an adequate head trauma indicates traumatic etiology, whereas later onset suggests uncertain etiology. Patients should be informed about this risk, and routines should be implemented for detection and treatment of t-BPPV in patients with minimal to moderate head trauma.

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