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Benign paroxysmal positional vertigo following whiplash injury: a myth or a reality?

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Abstract

Objective: The aim of the study was to evaluate the true incidence, diagnosis, and treatment of benign paroxysmal positional vertigo (BPPV) arising after whiplash injury and to distinguish this type of posttraumatic vertigo from other types of dizziness complained after trauma.

Methods: This was a retrospective study comprising patients referred to our center after whiplash injury. The patients were evaluated with neurotologic examination including bedside and instrumental tests. A Dizziness Handicap Inventory evaluating the symptoms of patients was submitted before and after treatment and was evaluated. The BPPV patients were separately evaluated from those with cervicogenic vertigo, and a comparison between our data about idiopathic BPPV was done.

Results: Eighteen patients of whiplash who had BPPV were evaluated. The mean age was 38.2 years. BPPV was the cause of vertigo in 33.9% of total whiplash patients. In 16 cases, the posterior semicircular canal was involved; the lateral semicircular canal was involved in 2 cases. The instrumental neurotologic assessment did not show any alteration of either vestibulospinal reflexes or dynamic ocular movements. Duration of symptoms before treatment ranged from 3 to 26 days. A total of 55.5% of patients had relief from their symptoms after first repositioning maneuver. The Dizziness Handicap Inventory score improved in all patients treated with repositioning maneuvers, but no difference emerged with idiopathic BPPV data.

Conclusion: BPPV after whiplash injury could be unveiled with a simple bedside examination of peripheral vestibular system, and a treatment could be done in the same session. The diagnosis of posttraumatic BPPV is not different from the idiopathic form, but the treatment may require more maneuvers to achieve satisfactory results.

1. Introduction

The equilibrium is the result of a perfect integration of input from eyesight, proprioceptive receptors, and labyrinths. The whiplash injury is a traumatic lesion due to rapid flexion-extension movement of the cervical column. This causes disturbance signals reaching from the cervical proprioceptive system to the central vestibular system and thus has an adverse effect on equilibrium. Balance problems affect 5% to 50% of patients of whiplash injury [1,2]. The whiplash injury is generally due to car collision and is the first cause of insurance claims. About 15% to 20% of cases develop the so-called late whiplash syndrome with persistent complaints including headache, vertigo, instability, nausea, and tinnitus [2].

The cervical trauma may increase the discharge of muscles' proprioceptive receptors of the neck [3] and may

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interfere with normal activity of vestibular system, resulting in an alteration of vestibular-ocular reflexes [4,5]. The neck pain, and related balance impairment, is the most common symptom and may be the cause of the symptoms complained by patients that is generally called *cervicogenic vertigo*.

The quantification of vestibular damage is difficult in forensic cases. For these reasons, it is mandatory to detect maligners and distinguishing them from patients with true disequilibrium disturbance [6].

Labyrinthine vertigo and auditory disturbances are present in 25% and 17% of subjects, respectively [7]. The incidence of dizziness with even mild head injury ranges from 15% to 78%, probably because of an unclear definition of the trauma and of the concept of dizziness [8-10].

Benign paroxysmal positional vertigo (BPPV) is the most frequent cause of peripheral vertigo [11]. It accounts for approximately 24% of all cases of peripheral vestibular disorders [12]. This type of vertigo is generally seen in individuals 40 years and older, with the highest incidence between 50 and 70 years [13].

The exact etiology of BPPV is still debatable. More than 50% of all reported cases are idiopathic in nature [14]. Adler [15] was the first to describe manifestations of BPPV in posttraumatic cases. Classic BPPV involves the posterior semicircular canal (PSC) and represents the most common type of BPPV [13,16-18].

Diagnosis of BPPV is based mainly on history of characteristic positional vertigo along with the classic clinical signs. Symptoms are characterized by rotating vertigo with nausea and vomiting, elicited by movements of the head. The nystagmus typically has a latency (of few seconds) and is of limited duration (10–20 seconds), transient, and fatigable [16].

In patients of BPPV, correct diagnostic evaluation and appropriate management, in the great number of cases, solve the problem quickly, without the need for any medical treatment [19].

Our work's aims were to analyze the patients affected by BPPV after whiplash injury and to evaluate differences between idiopathic BPPV and so-called cervicogenic vertigo.

2. Materials and methods

A retrospective study of patients of whiplash injury who visited our department between January 2008 and September 2009 was conducted. Patients with a diagnosis of BPPV following the trauma, occurring within 1 week before presentation, were evaluated. The exclusion criteria were history of vertigo before the whiplash injury, history of ear diseases or hearing loss, central nervous system pathology, psychiatric diseases, and history of vascular diseases. The evaluation included clinical history, complete head and neck examination, clinical vestibular tests, pure tone audiometry, and videoculography/videonystagmography with infrared system (ULMER, Synapsis Inc, Marseille, France). The smooth-pursuit function was evaluated by following an oscillating lighted target on a 29-in screen. The target velocity was regular (18°/s); and the oscillating movements were from center to right, back to the center, and center to left. Each trial was of 20 cycles of 57 seconds each. The saccadic test was performed by watching the target on a horizontal plane; the frequency was 0.4 Hz, and the amplitude was ±20°. The parameters analyzed in saccadic movement evaluation were latency, velocity, and accuracy. Our cutoff parameters to consider results as pathologic were as follows: value more than 200 milliseconds for latency, a score less than 77% for accuracy, and a highest velocity less than 530°/s. The smooth-pursuit movement analysis was evaluated measuring the gain (eye velocity/target velocity), and the parameter of normality was a value of 0.88 ± 0.2.

The same operator performed all instrumental tests.

The Dizziness Handicap Inventory (DHI) was used as reference for clinical improvement and was submitted at first contact and during the follow-up.

The clinical vestibular tests used to investigate the positioning nystagmus were the Dix-Hallpike and McClure-Pagnini tests (nystagmus observed in a supine position while turning the head to the left and right). The vestibulospinal function was evaluated by the Romberg test, index deviation test, and Unterberger test [20]. The dynamic ocular bedside examination included the head-shaking test and Halmagyi test. The evaluation of nystagmus and vestibular reflexes was done after a period of at least 5 days from last dosage of vestibular suppressant drugs.

Imaging is generally included in those patients with uncommon clinical presentation or those with lack of response to treatment [21].

After diagnosing BPPV, all patients were promptly treated with canalith repositioning maneuver (CRM) according to the affected semicircular canal. The Semont maneuver or the Epley maneuver was used to treat a PSC BPPV; the Gufoni maneuver was used to manage the lateral semicircular canal (LSC) BPPV [19] The persistence of nystagmus and vertigo spells after CRM was indicative of treatment failure, and repetition of maneuver was done in the same session or in the following 3 days. No medical treatment was prescribed to patients after CRM. The follow-up was done at 1 week, 3 weeks, and 1 month; the patients were examined, and a DHI questionnaire was submitted at the second and sixth months after the treatment.

The patients with cervicogenic vertigo were treated with a combination of physiotherapy, habituation exercises, and analgesics.

Finally, the data where matched with our result in treating idiopathic BPPV [19,22] and with patients complaining a "cervicogenic vertigo" following a whiplash injury to evaluate the differences.

Our Institutional Board reviewed and approved the study.

3. Results

Out of 53 patients of whiplash injury who were referred to our department, 33 were male. The mean age was 40.77 (range, 18–66) years. In all patients, the head trauma occurred within 1 week before presentation.

In 20 patients, the history revealed the presence of rotating vertigo after trauma. An unspecified imbalance or dizziness was the chief complaint in 33 patients, which were labeled as having cervicogenic vertigo. These patients did not show any alteration on neurotologic examination. The hearing was normal in all patients.

In 18 of 20 patients, the vertigo was related to head position and movement on the bed. In the remaining 2 cases, a labyrinthine concussion was diagnosed; and these 2 patients were excluded from present analysis. No patient reported history of vertigo before the head trauma.

The result of the Dix-Hallpike diagnostic maneuver was positive in 16 of 18 patients, whereas the result of the McClure-Pagnini maneuver for the LSC was positive in 2 cases.

Out of 18 whiplash patients with BPPV, 11 were male. The mean age of the group was 38.2 (range, 25–66) years. The BPPV was the cause of vertigo in 33.9% of whiplash patients. In 16 cases, PSC was involved, the right side in 9 patients and left side in 7 cases. The left LSC was involved in the remaining 2 cases. The neurotologic assessment did not show any alteration of either vestibulospinal reflexes or dynamic ocular movement (Table 1).

Symptoms duration before treatment ranged from 3 to 26 days. A symptoms-free period was present in all cases, with recurrence of vertigo spells after a period ranging from 1 week to 1 month.

The DHI score improved in all patients treated with CRM, as shown in Table 2. Although the improvement was remarkable if compared with cervicogenic vertigo results, no difference emerged with DHI after treatment of idiopathic BPPV.

Table 1
Analysis of eye movement results of the patients

	Idiopathic BPPV (110 cases)	Cervicogenic vertigo (33 cases)	Posttraumatic BPPV (18 cases)
Smooth-pursuit gain (SD)	0.87 (±0.04)	0.86 (±0.03)	0.87 (±0.04)
Saccadic velocity, °/s (SD)	571 (±34.1)	572.05 (±24.35)	565.9 (±27.47)
Saccadic latency, °/s (SD)	155.3 (±13)	161.73 (±13.07)	155.4 (±14.35)
Saccadic accuracy, % (SD)	0.83 (±0.06)	0.83 (±0.06)	0.84 (±0.06)

The patients with cervicogenic vertigo and posttraumatic BPPV due to whiplash injury.

Table 2
Dizziness Handicap Inventory in the patients pre- and posttreatment

	DHI pretreatment	DHI posttreatment (last)
Idiopathic BPPV	Severe: 75	Severe: 0
	Moderate: 33	Moderate: 1
	Low: 2	Low: 109
Posttraumatic BPPV	Severe: 15	Severe: 0
	Moderate: 3	Moderate: 0
	Low: 0	Low: 18
Cervicogenic vertigo	Severe: 9	Severe: 3
	Moderate: 18	Moderate: 15
	Low: 6	Low: 11

The patients with cervicogenic vertigo and posttraumatic BPPV due to whiplash injury. The score of DHI is as follows: severe (100–70 points), moderate (69–40 points), and low (39–0 point).

A total of 55.5% (10 cases) of patients had relief from their symptoms after first CRM. Two CRMs were necessary in 6 (33.3%) patients, including the 2 patients with LSC BPPV. The remaining 2 (11.1%) patients required 3 sessions of CRM to achieve satisfactory results. Comparing these results with the treatment of idiopathic BPPV, we noted that the posttraumatic variant requires more maneuvers to reach curative repositioning of otoliths (Table 3).

4. Discussion

The effect of trauma on cervical column was first described by Crowe [23] in 1928 and followed by Gray and Abbott [24] in 1953. Significant injuries can occur following even low-speed collision, but simulated accidents have shown that a 5-mph rear-end car crash can result in a positive acceleration of 8.2 G of the head [25].

The whiplash injury is a biomechanical event that produces a distortion of the cervical column causing lesions of several cervical sites, such as the muscles, ligaments, vertebral joints, vessels, and nerves. The typical acute symptoms after whiplash injury includes neck pain, headache, paraesthesia of upper cervical dermatomes, dizziness or imbalance, and tinnitus [26,27]. The majority of patients recover spontaneously after few months of symptomatic treatment. In some patients, the symptoms may persist.

Pathophysiologically, there is central nervous system weakness following a whiplash injury. With respect to inner ear pathology due to whiplash, the exact nature of the lesion

Table 3
Canalith repositioning maneuver needed to reach the curative repositioning of otoliths

	Idiopathic BPPV	Posttraumatic BPPV
1 CRM	81%	55.5%
2 CRMs	17%	33.3%
>2 CRMs	2%	11.1%

is not known; but some possible explanations are transient ischemia by vertebral artery compression, hemorrhage into labyrinth, direct labyrinthine concussion, and noise of the collision.

A neurotologic evaluation should be performed to find a possible cause of symptoms. The examination includes the assessment of peripheral labyrinth and vestibulo-ocular reflex system by means of clinical vestibular tests, caloric test, and videoculography/videonystagmography.

Previous studies have scarcely demonstrated any relationship between peripheral dysfunction and trauma. Some studies present in the literature have shown positional nystagmus and unilateral hyporeflexia in patients of whiplash injury [9,28-33]. Ettlin et al [34] reported peripheral vestibular deficits in only 2 cases among 18 cases after whiplash injury. However, all these reports have not used the standard value as benchmark or any proper control group. In a previous study, we did not find any correlation between trauma and eye movements in patients with recent trauma.

However, the whiplash injury is considered to be a direct cause of the BPPV [35,36] especially when head trauma is associated. The pathophysiology of the classic BPPV as a disorder of otoliths was suspected first by Barany and later supported by Schuknecht [37,38]. Following the trauma, the otoliths are detached from the utricle and displaced within the labyrinth. Generally, patients affected by idiopathic BPPV are older than those due to trauma.

The patients experience severe vertigo when rolling in one particular direction in bed and, less frequently, may also report dizziness with head motion. For dizziness occurring at times other than in bed, cervicogenic vertigo must be considered after a whiplash trauma. Head injury may equally affect both labyrinths; thus, bilateral BPPV is expected to occur more frequently in the posttraumatic cases [35,39], although we have not noted a high incidence of bilateral cases in our series.

There is consensus on the incidence of posttraumatic BPPV that accounts for 15% to 20% of all cases [40,41].

The diagnosis is confirmed by positional tests (Dix-Hallpike [38], McClure-Pagnini [42]). Establishing a diagnosis of BPPV is beneficial because it is treated by relatively simple physical maneuvers without the need for additional investigations or drug therapy. About 80% of patients with posterior canal idiopathic BPPV become free of symptoms and signs following a single maneuver [43-46]. The percentage of successful repositioning after the first CRM decreases in posttraumatic cases. Our hypothesis is that it may be due to the increased number of otoliths displaced as consequence of trauma.

Although the improvement of patients affected by BPPV is remarkable after treatment, if compared with cervicogenic vertigo patients as shown in our results, any difference emerged comparing DHI after treatment of idiopathic BPPV vs posttraumatic cases. This may be explicable considering that the pathophysiology of BPPV, even if posttraumatic, is related to the same mechanism of otolith movements into labyrinth.

It is widely believed that a whiplash injury may induce a disorder of neck proprioceptors caused by forces applied to the neck in the course of the accident. We concur with Fischer et al [47] in distinguishing either spontaneous or positional nystagmus arising in certain static head positions from the previously reported “cervical nystagmus” because static labyrinthine stimulation may exist. Several authors have failed to demonstrate a relationship between nystagmus and neck proprioceptors’ stimulation [30,31,48].

5. Conclusions

A syndrome in which the dizziness is the main complaint often follows a whiplash trauma. With simple bedside examination of peripheral vestibular system, it is possible to find out the patients with true vertigo; and a treatment could be done in the same session. The diagnosis of posttraumatic BPPV is not different from the idiopathic form, but the treatment may require more CRMs to achieve satisfactory results. In addition, the complete neurotologic examination of vestibular system allows us to recognize maligners who complain of vertigo or dizziness with the aim of insurance claim.

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