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Bilateral benign paroxysmal positional vertigo following a tooth implantation

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Abstract
Benign paroxysmal positional vertigo (BPPV) is a common cause of vertigo and may occur following recent head trauma. Bilateral involvement in BPPV is considered rare and has received little attention in the medical literature. We describe an unusual case of bilateral BPPV in a middle-aged woman that presented during a dental implantation, performed with the use of osteotomes. We discuss the diagnostic and therapeutic challenge of this entity.

Key words: Vertigo; Dentistry, Operative

Case report

A 52-year-old female nurse presented to an otolaryngology clinic with incapacitating vertigo, accompanied with vomiting that had started 24 hours previously. The previous day the patient had undergone a tooth implant. Dental implants were planned for the first and second left upper premolar teeth (#24 & #25). Due to a narrow alveolar ridge on the bucco-lingual dimension, a ridge expansion osteotomy technique was performed.[1] After elevating a mucoperiosteal flap at this site, a 2 mm Pilot drill was first used, followed by the introduction of a 2.9 mm osteotome. During the removal of bone with the osteotome the patient experienced a severe sensation of spinning. Implantation was completed at the site of tooth #24 and the remainder of the procedure was aborted. The patient's vertigo was continuous and worsened upon raising and lowering her head, and she was overwhelmed with a sense of unsteadiness. The patient was unable to identify a specific side that exacerbated her symptoms and was only able to sleep in the supine position. She denied hearing loss, tinnitus or aural pressure, and a history of central nervous system or otological disease was negative. The rest of her past medical history was unremarkable.

Upon entering the examining room the patient was unable to maintain her balance and required physical support. Her occulomotor examination was normal and there was no spontaneous nystagmus noted. She had guarded head movements with rigidity of the cervical muscles and was unable to undergo the head thrust test (Halmaghi's manoeuvres) or to have her head shaken to examine for the presence of post-head shake nystagmus. On the Dix-Hallpike (DH) positioning test the patient experienced vertigo and a geotropic tortional nystagmus was noted in both head-hanging positions. The amplitude and velocity of the nystagmus as well as the sensation of vertigo were greater when the examination was performed on the right than on the left side and accordingly, Epley's particle reposition procedure was performed on the right side. The patient was instructed to sleep in a semi-sitting position for three nights. The patient stayed vertiginous for another week and then gradually recovered and was able to return to work.

On a follow-up visit at one month she felt better but was afraid to have the DH examination repeated. Six weeks after her initial visit the patient re-experienced bouts of vertigo lasting for approximately 20 seconds, while positioning her head downwards to the left. The head thrust test was normal and there was no post-head shake nystagmus. The DH exam demonstrated geotropic tortional nystagmus in the left head-hanging position, accompanied with a sensation of spinning. Examination of her right side was normal, as was the rest of the neurotological exam. The Epley manoeuvre was performed on her left side, and the patient was re-instructed to sleep in the semi-sitting position for three nights. The patient returned to work a week later and experienced gradual improvement during the next two months.

Discussion

BPPV is a common cause of vertigo and is characterized by episodes lasting for seconds. The diagnosis is established by inducing a rapid change from the sitting position to the left or right head-hanging position – the DH manoeuvre. Typically, a tortional geotropic nystagmus is seen when the affected ear is downmost, accompanied with a sensation of vertigo. Other classic features of the nystagmus in BPPV include a latency of several seconds, fatigability and reversal of the direction of nystagmus when the patient returns to the upright sitting position.[2] Schuknecht explained the pathophysiology to be caused by cupulolithiasis-deposition of utricular otoconia on the cupula of the posterior semicircular canal.[3] The more accepted theory today is canalithiasis; free floating material within the lumen of the posterior semicircular canal that moves with gravity and deflects the cupula.[2]

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Head trauma in general, is a well recognized aetiology, and has been documented in 17 per cent of 240 patients presenting with BPPV.[4] The aetiology of head trauma was considered in this study when it preceded symptoms by no more than three days, however no reference is made to the exact type and mechanism of trauma.

The presumed aetiology of BPPV in our case is blunt head trauma caused by osteotomy. The ridge expansion osteotomy technique is indicated for tooth implantation with a narrow alveolar ridge.[1] In this way bone is conserved rather than removed, resulting in increased support of the implant. It would seem likely that the use of an osteotome in this case was sufficient to cause dislodging of otoconia and producing the patient's bilateral BPPV. To the best of our knowledge BPPV has not been reported previously, following tooth implantation in general, nor after use of ridge expansion osteotomy, specifically. In a review of the literature there has been a publication on a 25-year-old patient who developed unilateral BPPV three days after a parietal osteoma had been removed with a hammer and chisel.[5] The patient gradually recovered over the next year without a particle reposition procedure being performed. Although the advent of high-powered surgical drills has reduced their overall use, oseotomes are still used in surgery of the head and the neck, especially in septoplasty and rhinoplasty. Since BPPV has not been reported following these common procedures, the complication we have reported should be considered very rare.

Surprisingly, there is scant literature on bilateral BPPV (BiBPPV). Katsarkas[6] studied over 2500 individuals with BPPV and defined bilateral disease as occurring only when both ears are simultaneously affected. The incidence of BiBPPV was significantly higher among post-trauma patients (14.3 per cent) compared to those with idiopathic BPPV (6.3 per cent). Longbridge and Barber[7] presented a comprehensive series of 15 patients with BiBPPV out of a total of 114 patients with BPPV. This subgroup of patients had a significantly higher rate of a recognized aetiology (head trauma and central disease), when compared to a group of patients with unilateral BPPV (51 per cent, *vs* 13 per cent).

Correct diagnosis of unilateral versus BiBPPV may be important when surgical intervention such as a posterior canal occlusion is considered. Stellin and Bandt[8] have postulated that quite often unilateral BPPV may mimic BiBPPV. Their observations are based on performing a body tilt from the sitting position similar to Semont's manoeuvre and not the DH manoeuvre. In their testing, when the head of the patient is parallel to the plane of the body tilt, a tortional geotropic nystagmus may be elicited with the unaffected ear downmost. According to their theory, when the patient is positioned with the unaffected side downmost, without tilting the head on the affected side, the posterior canal becomes uppermost and may also be stimulated be the debris, as it settles on the cupula, ampulopetally. This mechanism of cupulolithiasis on the unaffected side mimics a contralateral BPPV, but the nystagmus is characterized by low amplitude and low frequency. This study raises the question of whether our patient had indeed experienced BiBPPV. When examining Stellin and Bandt's illustrations of their manoeuvre to avoid a false positive BiBPPV it is evident that in the DH position with the head at 45° in relation to the axis of the body, a similar position of the posterior semicircular canals is achieved. Furthermore, the sequence of events in our patient support the assumption that the patient had bilateral disease.

Management of BiBPPV also remains unclear. Parnes and McClure[9] performed a posterior semicircular canal occlusion on an 82-year-old woman diagnosed with BiBPPV. Surgery was performed unilaterally, on the more symptomatic side and the patient was free of symptoms on long-term follow-up. The other studies reviewed above by Katsarkas[6] and Longbridge and Barber[7] do not report on management or outcome of their patients.

In keeping with the two theories of BPPV (cupulolithiasis and canalithiasis), single treatment approaches such as the Epley's and Semont's manoeuvre are aimed at moving debris into the vestibule or into the crus commune by the force of gravity. Performing either one of the manoeuvres on both sides at the same session may be counterproductive as the side first treated may become re-stimulated while the libratory manoeuvre is being performed on the contralateral side. We have therefore adopted a staged strategy of initially treating the side with more symptoms and with a higher magnitude of nystagmus. Only when an improvement in the patient's symptoms and on the DH manoeuvre are seen, does the patient undergo treatment on the contralateral side. Semont's manoeuvre, which does not re-stimulate the side treated first, could be used as an alternative to Epley's manoeuvre, and may be more suitable for a single stage bilateral treatment of BiBPPV.

We conclude that BiBPPV is a rare condition and similarly to unilateral BPPV, it may be caused by trauma to the skull. Management principles are similar to those employed in unilateral BPPV, however convalescence may take longer, especially as a staged canal repositioning procedure may be necessary.

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