

Vestibular neuritis and benign paroxysmal positional vertigo: a case report and review of the literature

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Summary

Background. Benign paroxysmal positional vertigo (BPPV) is the most common subtype of vertigo, that results in self-resolving and paroxysmal episodes of nausea, vomiting, and dizziness. Middle-aged women are most frequently affected. Vestibular neuritis is a viral inner ear disorder characterized by acute vertigo accompanied by nausea and vomiting. It is the second most common cause of secondary BPPV.

Case report. A 21-year-old male patient presented to the emergency room after experiencing severe vertigo after waking up in the morning, accompanied by a walking imbalance. He was diagnosed with vestibular neuritis and after a few months, a Dix-Hallpike and a supine roll test yielded positive results, with right peripheral nystagmus

being present during both. The diagnosis of secondary posterior and horizontal benign paroxysmal positional vertigo was determined. The patient was treated with various repositional maneuvers, which proved to be advantageous for a short period of time, soon after the symptoms got worse. The patient started to face problems with focusing the eyes and temporary vertigo attacks with a slight left nystagmus with a rotary component. As a result of recurring BPPV, therapy was provided. Moreover, vestibular rehabilitation as well as modified repositional maneuvers were consistently done for 12 months. As a result of the treatment, the symptoms have improved, with the complaints not disappearing entirely.

Conclusion. Vestibular neuritis can cause a secondary BPPV, with it usually being treatment-resistant. We suggest a thorough examination of patients with VN due to the possibility of BPPV.

Keywords: vestibular neuritis, benign paroxysmal positional vertigo, repositional maneuvers, nystagmus, treatment-resistant.

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is the most common subtype of vertigo [1]. It presents itself with brief episodes of dizziness, nausea, and vomiting [2]. The second most frequent cause of vertigo is vestibular neuritis (VN) [3]. It is a viral inner ear disorder characterized by acute vertigo with nausea, and vomiting [2, 3]. Secondary BPPV is frequently caused by VN [2].

CASE REPORT

A 21-year-old male patient presented to the emergency room after experiencing severe vertigo after waking up in the morning, accompanied by a walking imbalance. After being diagnosed with vestibular neuritis, the patient received therapy with betahistine, prednisolone, and B vitamins. A few months later a Dix-Hallpike and a positive supine roll test yielded positive results, with right peripheral nystagmus being present during both. The diagnosis of secondary posterior and horizontal benign paroxysmal positional vertigo was determined. The patient was treated with various repositional maneuvers, which proved to be advantageous for a short period of time, soon after the

symptoms got worse. The patient started to face problems with focusing the eyes and temporary vertigo attacks with a slight left nystagmus a rotary component. No pathological findings were detected during the MRI of the head. As a result of recurring BPPV, vestibular dysfunction, persisting vertigo attacks, and issues with focusing the eyes, therapy was provided by administering tab. Cinnarizinum et Dimenhydrinatum. Moreover, vestibular rehabilitation as well as modified repositional maneuvers were consistently done for 12 months. As a result of the treatment, the symptoms have improved, with the complaints not disappearing entirely.

DISCUSSION

Vestibular neuritis is a frequent vestibular illness, that presents itself with sudden vertigo, vomiting, nausea, and disequilibrium [2]. The incidence rate is 3.5 per 100000 people [4]. A common complication of vestibular neuritis is benign paroxysmal positional vertigo [2].

Benign paroxysmal positional vertigo (BPPV) is a vestibular disorder that results in brief yet severe episodes of vertigo that occur when triggered movement relative to

gravity or by specific head positions [5]. This can be described as a feeling of loss of balance, tilt, rotation, or sway [6]. In severe cases, temporary nausea and vomiting lasting up to 1 minute are observed [7]. Dizziness and other associated symptoms are caused by otolith fragments originating from the *macula utriculi*. These fragments move into one or more semicircular canals and make the *ampulla* of the semicircular canals sensitive to gravity [8]. Among adult peripheral vestibular disorders, BPPV is the most common. Its prevalence in the population is 2.4%, with women being more affected [5, 9]. The disease is common in middle-aged and elderly people [7]. Among adult peripheral vestibular disorders, BPPV is the most common. Its prevalence in the population is 2.4%, with women being more affected [5, 9]. However, the increased rate of incidence of BPPV is now being observed more frequently in children and adolescents [5]. As a result of the deficiency of etiological evidence, it is frequently classified as primary or idiopathic [10]. BPPV can be triggered by various factors such as hypertension, migraine, Meniere's disease, vestibular neuritis, ear surgery, head trauma, long-distance travel, upper respiratory tract infections, and prolonged bed rest [11].

The semicircular canals are filled with endolymph and arranged orthogonally, each canal is at right angles to the other two canals, and is referred to as the lateral, anterior, or posterior semicircular canal [12]. The enlarged (ampullary) end *crista ampullaris*, which contains the receptors, is at the end of each of the semicircular canal [6]. It contains hair cells with stereocilia oriented in the same direction, which pass into the *cupula* [12]. The otoconia are crucial for the *utricula* to function, as they innervate the hair cells in the endolymph, which transmit changes in the position of the head, including tilt, rotation and linear acceleration [6]. The *cupula* forms an impermeable barrier to the endolymph as it extends from the surface of the cristae to the top of the membranous labyrinth and the lateral walls, which makes the *cupula* susceptible to changes in the flow of endolymphatic fluid [13]. When the head is turned, the endolymph bounces against the *cupula*, triggering a nerve impulse informing the body of the sensation of movement [6]. During linear head movement, equal forces are generated on both sides of the *cupula* and the stereocilia remain in place without transmitting a signal [12].

Benign paroxysmal positional vertigo is caused by shifting calcium carbonate crystals or otoliths in the fluid-filled semicircular canals of the inner ear [6]. There are several theories for the occurrence of BPPV. One of them is described by Schuknecht, who states that otolith fragments detach from the *macula utriculi* and attach themselves to the semicircular canals in the *cupula*, causing it to become encumbered. The weight of the otoliths causes the *cupula* to become immobile and it does not return to a neutral position when a certain position is achieved [6, 9]. Another theory predicts that the *ampulla* of the posterior semicircular canal accumulates displaced otoconia, which have a higher weight than the endolymph [14]. Due to the movement and gravity the otoconia the endolymph to drift away from the *ampulla*

in turn diverting the *cupula* and producing nystagmus [6]. In the case of secondary BPPV after VN, the later disease causes the otoliths to detach, causing secondary BPPV [2].

BPPV is diagnosed through a variety of maneuvers, that are done by tilting the head in planes of each semicircular canal. The most used one being the Dix Hallpike maneuver [8, 9]. This maneuver provokes nystagmus by rotating a seated patient's head at 45 degrees, afterward, the patient is swiftly lowered to a supine position with the neck being extended below the level of the table [15]. Other diagnostic possibilities include the VBIT test which is a useful tool in diagnosing BPPV when used together with other positional maneuver tests [16].

The treatment options vary on the location of the otolith and are dependent on age, comorbidities, and the disease pathophysiology. The treatment of posterior cupulolithiasis includes the Epley maneuver and, as Guneri et al. in their study demonstrated that, the addition of the betahistine has significantly decreased the symptoms [1, 17]. Semont maneuver, the 3-position maneuver, the Gans, and the Parnes maneuvers have also shown high efficacy in treating BPPV [1, 6]. For the treatment of horizontal cupulolithiasis, the Gufoni maneuver is often a first choice due to its easy execution and overall effectiveness [1]. The superior BPPV reposition includes the Epley and the Vacovino maneuvers, which were both found highly effective [1].

CONCLUSION

In conclusion, secondary BPPV caused by VN is frequently treatment-resistant. Numerous treatment options have been proposed, which only when used together, have been proven somewhat efficacious, without fully reversing the symptoms.

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Santrauka

VESTIBULINIS NEURONITAS IR GERYBINIS PAROKSIZMINIS POZICINIS SVAIGIMAS: KLINIKINIO ATVEJO APRAŠYMAS IR LITERATŪROS APŽVALGA

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Darbo tikslas. Gerybinis paroksizminis pozicinis svaigimas (GPPS) yra dažniausiai pasitaikantis galvos svaigimo potipis, kuris pasireiškia savaime praeinančiais paroksizminiais pykinimo, vėmimo bei galvos svaigimo epizodais. Dažniausiai serga vidutinio amžiaus moterys. Vestibulinis neuronitas yra virusinis vidinės ausies sutrikimas, kuriam būdingas ūmus galvos svaigimas, lydimas pykinimo ir vėmimo. Tai antra pagal dažnumą antrinio GPPS priežastis.

Atvejo aprašymas. Aprašome klinikinį atvejį 21 metų paciento, kuris atvyko į priėmimo skyrių dėl ryte pabudus pasireiškusio stipraus galvos svaigimo, nestabilumo jausmo einant. Jam buvo diagnozuotas vestibulinis neuronitas, o po kelių mėnesių atlikus Dix–Hallpike ir horizontalios padėties pasukimo testus buvo gauti teigiami rezultatai: atliekant abu testus buvo matomas dešinysis periferinis nistagmas. Nustatytas antrinis užpakalinis ir horizontalus gerybinis paroksizminis pozicinis galvos svaigimas. Pacientas buvo gydomas įvairiais repozicininiais manevrais, kurie buvo naudingi trumpą laiką, tačiau simptomai vėl pablogėjo. Pacientas pradėjo susidurti su žvilgsnio fokusavimo problemomis ir laikiniais galvos

svaigimo priepuoliais, kurių metu buvo nedidelis kairysis nistagmas su rotaciniu komponentu. Dėl pasikartojančio GPPS buvo skirtas gydymas. Be to, 12 mėnesių buvo atliekama vestibulinė reabilitacija bei modifikuoti repoziciniai manevrai. Gautas klinikinis pagerėjimas, tačiau nusi-skundimai visiškai neišnyko.

Išvados. Vestibulinis neuronitas gali sukelti antrinį GPPS, kuris dažniausiai yra atsparus gydymui. Siūlome nuodugniai ištirti pacientus, sergančius vestibuliniu neuronitu, dėl galimo GPPS.

Reikšminiai žodžiai: vestibulinis neuronitas, gerybinis paroksizminis pozicinis svaigimas, repoziciniai manevrai, nistagmas, atsparumas gydymui.

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