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# LITERATURE REVIEW AND TWO STUDY CASES OF VESTIBULAR PAROXYSMIA FROM AN OTORHINOLARYNGOLOGY CENTRE

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Contributions:
A Study design/planning
B Data collection/entry
C Data analysis/statistics
D Data interpretation
E Preparation of manuscript
F Literature analysis/search
G Funds collection

## Abstract

**Introduction:** Vestibular paroxysmia is a rare disorder of the balance system manifested by recurrent attacks of vertigo, the etiology of which is associated with compression of a blood vessel on the vestibulocochlear nerve. The exact mechanism for triggering the symptoms is not fully understood. The condition occurs in all age groups and without gender predilection. The purpose of the study was to review the literature on vestibular paroxysmia, discuss criteria for the diagnosis of the disease, diagnostic management, proposed therapy, and present clinical cases from the authors' own practice.

**Material and methods:** A review of the scientific literature was conducted via the PubMed database using the keywords "vestibular paroxysmia," "neurovascular conflict cochlear nerve," and "neurovascular conflict vestibular nerve". PRISMA guidelines for systematic reviews were applied.

**Results:** There were 146 publications found; after analysis of titles and abstracts, 56 were included in the review; another 10 manuscripts were identified during reference review. Among them, 11 studies were devoted to diagnostic imaging, 19 described the clinical course of the condition based on retrospective studies, 15 were case reports, and 11 other reports and reviews. Following evaluation of this literature, two clinical cases of vestibular paroxysm from the authors' own practice are described. These cases are presented along with their diagnosis and therapy.

**Conclusions:** Imaging studies are of significant value in diagnosing many vascular–neural conflicts, but for vestibular paroxysmia the diagnosis is made on the basis of clinical symptoms and according to the division proposed by the Bárány Society in 2016. Pharmacological therapy is recommended, and response to therapy is one of the criteria for diagnosing the condition. Surgical treatment is reserved for the most severe cases.

**Key words:** vertigo • microvascular decompression • neurovascular conflict • vestibular paroxysm • vestibulocochlear nerve

# PRZEGLĄD LITERATURY I OPIS DWÓCH PRZYPADKÓW PAROKSYZMU PRZEDSIONKOWEGO Z KLINIKI OTORYNOLARYNGOLOGII

## Streszczenie

**Wstęp:** Paroksyzm przedsionkowy to rzadkie schorzenie układu równowagi objawiające się nawracającymi i napadowymi zawrotami głowy o charakterze wirowania, którego etiologia wiązana jest z uciskiem naczynia krwionośnego na nerw przedsionkowo-ślimakowy. Dokładny mechanizm wyzwalania objawów nie jest do końca poznany. Schorzenie to występuje we wszystkich grupach wiekowych i bez predylekcji płci. Celem tej pracy był przegląd literatury dotyczącej paroksyzmu przedsionkowego, omówienie kryteriów rozpoznania choroby, postępowania diagnostycznego, proponowanych metod terapii oraz prezentacja przypadków klinicznych z praktyki własnej autorów opracowania.

**Materiał i metody:** Przeprowadzono przegląd literatury naukowej za pośrednictwem bazy PubMed, stosując słowa kluczowe „vestibular paroxysmia", „neurovascular conflict cochlear nerve" oraz „neurovascular conflict vestibular nerve". Zastosowano wytyczne PRISMA dla przeglądów systematycznych.

**Wyniki:** Uzyskano 146 wyników, po analizie tytułów i abstraktów do przeglądu włączono 56 publikacji, dodatkowo zidentyfikowano 10 manuskryptów w trakcie przeglądu referencji. Zidentyfikowano 11 badań poświęconych diagnostyce obrazowej, 19 manuskryptów opisujących przebieg kliniczny schorzenia na podstawie badań retrospektywnych oraz prospektywnych, 15 opisów przypadków oraz 11 inne doniesienia, w tym prace przeglądowe, zebrane w niniejszym opracowaniu. Opisano pochodzące z praktyki własnej autorów opracowania dwa przypadki kliniczne paroksyzmu przedsionkowego wraz z przeprowadzoną diagnostyką i wdrożoną terapią.

**Wnioski:** Badania obrazowe przedstawiają istotną wartość podczas diagnostyki konfliktu naczyniowo-nerwowego, ale rozpoznanie paroksyzmu przedsionkowego stawiane jest na podstawie objawów klinicznych według podziału zaproponowanego w 2016 r. przez Towarzystwo Bárány'ego. Wskazana jest terapia farmakologiczna, odpowiedź na terapię jest jednym z kryteriów rozpoznania schorzenia. Leczenie chirurgiczne zarezerwowane jest dla najcięższych przypadków.

**Słowa kluczowe:** zawroty głowy • dekompresja mikronaczyniowa • konflikt naczyniowo-nerwowy • paroksyzm przedsionkowy • nerw przedsionkowo-ślimakowy

| Key for abbreviations | |
|---|---|
| AICA | anterior inferior cerebellar artery |
| BMT | betahistine mesylate |
| BPPV | benign paroxysmal positional vertigo |
| CBZ | carbamazepine |
| CISS | constructive interference in steady state |
| CN | cranial nerve |
| CN VIII | vestibulocochlear nerve |
| dVP | definite vestibular paroxysmia |
| ICVD | International Classification of Vestibular Disorders |
| MRI | magnetic resonance imaging |
| MVD | microvascular decompression |
| NVC | neurovascular conflict |
| NVCC | neurovascular cross-compression |
| OXC | oxcarbazepine |
| PICA | posterior inferior cerebellar artery |
| pVP | probable vestibular paroxysmia |
| SCA | superior cerebellar artery |
| VNG | videonystagmography |
| VP | vestibular paroxysmia |

## Introduction

Dizziness is a common problem in the general population. On average, one in five adults [1] and one in eight children [2] experience, at some stage in their lives, symptoms described as unsteadiness, a feeling of swirling motion of the environment (vertigo), or dizziness. A positive correlation between the incidence of dizziness an age has also been described, probably related to the progressive degeneration of the peripheral organs of balance – such as proprioceptive feedback from the limb joints, the eye, and the labyrinth [3,4].

It is still common to divide disorders of the equilibrium system into those of central or peripheral origin; although this facilitates differential diagnosis, it is a major simplification of a complex problem [5,6]. Increasingly, mention is made of the "mixed" nature of dizziness, so that dysfunction of various receptors in the balance system (such as, for example, sensory weakness in the joints, dysfunction of the labyrinth, peripheral neuropathy) and disorders of central information processing (dementia-like changes, degenerative processes of the central nervous system) overlap to form a multifaceted picture [7,8]. Difficulties in the daily life of patients, resulting in a higher incidence of depressive disorders and chronic fatigue syndrome, among others, or adaptive difficulties in learning and work, illustrate how this problem is one that requires researchers to constantly update and improve diagnosis and therapy [9,10].

Benign paroxysmal positional vertigo (BPPV) is the most common cause of dizziness in adults (17.1%), while psychogenic dizziness affects 15% of adults, vestibular syndromes of central origin occur in 12.3%, and migraine-related attacks in 11.4%. Less frequent causes include vestibular neuritis (8.3%), bilateral vestibulopathy (Dandy syndrome) (7.1%) and vestibular paroxysmia (3.7%) [11,12]. There is a different etiology for vertigo in children, with the most common cause being migraine-related attacks (32%), followed by psychogenic causes (20%) and vestibular nerve inflammation (20%) [13,14].

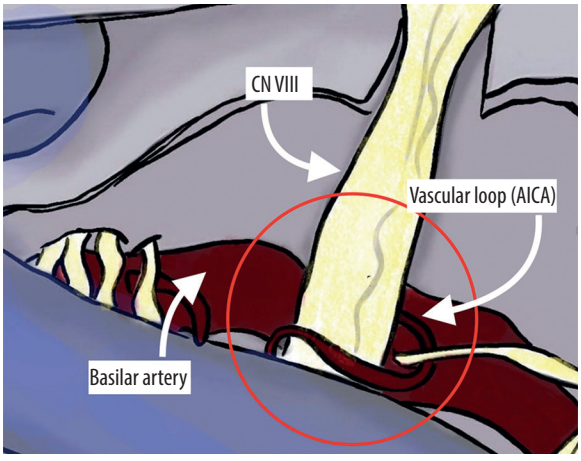


**Figure 1.** Vascular loop of the anterior inferior cerebellar artery wrapped around the vestibulospinal nerve (based on [64])

A special case of vertigo of peripheral origin is vestibular paroxysmia (VP), at the root of which is the phenomenon of neurovascular conflict (NVC) [15]. NVC can involve any cranial nerve and arises through pathological contact between a vessel and a nerve, causing characteristic symptoms depending on the function and location of the affected nerve. If there is compression of two or more cranial nerves by a single anomaly, then NVC can lead to overlapping disorders and symptoms [16,17]. Neurovascular pathologies can affect both adults and children [18].

NVC is usually caused by chronic cross-compression of the cranial nerve by an arterial vessel (neurovascular cross-compression, NVCC), since arterial anomalies are relatively more likely to produce symptoms than venous vessels. The most common form of such an anomaly is a vascular loop around the nerve [19,20]. There are also reports of

**Table 1.** Bárány Society diagnostic criteria for the diagnosis of vestibular paroxysmia [24]

| Diagnostic criteria* | |
|---|---|
| Vestibular paroxysm | Probable vestibular paroxysm |
| At least 10 attacks of spontaneous spinning or non-spinning dizziness | At least 5 attacks of spontaneous spinning or non-spinning dizziness |
| Duration < 1 min | Duration < 5 min |
| Stereotyped phenomenology in a particular patient | Stereotyped phenomenology in a particular patient |
| Response to treatment with carbamazepine or oxcarbazepine | – |
| – | Spontaneous or provoked by moving the head |
| Not better accounted for by another diagnosis | Not better accounted for by another diagnosis |

* Each point of the criteria must be met

brainstem tumors masquerading as symptoms of vestibular paroxysmia [21,22], so such a diagnosis should be considered in a differential diagnosis. Other anatomical variations, such as stenosis of the internal auditory canal, can also cause symptoms similar to VP [23]. The most common clinical manifestations of neurovascular conflict include trigeminal neuralgia, glossopharyngeal neuralgia, and tinnitus [23].

VP most often involves compression of the vestibulocochlear nerve (CN VIII) by the anterior inferior cerebellar artery (AICA) in 95% of the cases (**Figure 1**) [24], less frequently (5%) by the posterior inferior cerebellar artery (PICA) [25]. One case of compression by the vertebral artery has also been described [26].

Compression of the vestibulocochlear nerve was first described by Jannetta et al. in 1975, who named the pathology "impaired positional vertigo." [27]. The term "vestibular paroxysmia" was introduced in 1994 by Brandt and Dieterich, who also proposed the first diagnostic criteria [28]. In 2016, the Bárány Society defined new diagnostic criteria, part of the International Classification of Vestibular Disorders (ICVD), for a syndrome they called "vestibular paroxysmia" (VP), distinguishing between definite (dVP) and probable (pVP) forms [24].

Vertigo in VP is most often recurrent, paroxysmal, and of a whirling nature, lasting up to several minutes. BPPV is similar in nature, so a differential diagnosis should also include this disease. The possible ambiguous symptomatology of VP may require an ENT specialist to differentiate this pathology from Ménière's disease or migraine-induced vestibular disorder [29]. Complaints of VP may be accompanied by auditory symptoms – most often tinnitus, less often sensorineural hearing loss, and visual symptoms (nystagmus, often induced by hyperventilation and oscillopsia) [30–35]. Brand et al. also described a correlation, in 56% of the patients they studied, between VP and a fear of heights [36]. Detailed criteria for the diagnosis of VP are contained in the 2016 consensus of the Bárány Society [24], and are listed in **Table 1**.

The purpose of this paper is to present a review of the literature, and to describe the imaging procedures, otoneurological workup, differential diagnosis, and therapeutic differences in this form of NVC. In addition, we present two clinical cases of vestibular paroxysmia diagnosed and treated in the Department of Otolaryngology at the Polish Mother's Memorial Hospital – Research Institute in 2022.

## Material and methods

The PubMed database was reviewed using the PRISMA guidelines for systematic reviews (**Figure 2**). The following keywords were used: "vestibular paroxysmia", "neurovascular conflict cochlear nerve", and "neurovascular conflict vestibular nerve". There were 112 publications related to the first term, 13 to the second, and 21 to the third. Duplicates (11 publications) were removed.

The following inclusion criteria were used: 1) case reports, review papers, original papers; 2) subjects of papers on dizziness or vertigo in the course of NVC on vestibulocochlear nerve; 3) papers in Polish or English. The exclusion criteria were: 1) conference reports and letters to the editors; 2) papers older than 20 years. Two authors independently reviewed the titles and abstracts selected in stage one of the review ($n = 135$). There were 56 papers in Polish or English which met the inclusion criteria and 10 papers were also included for further review during the citation review.

## Results

From the database search, there were 146 manuscripts retrieved for initial review, and, after analysis of titles and abstracts, 66 publications were included in the review. There were 11 studies devoted to diagnostic imaging, 19 describing the clinical course of the condition based on retrospective studies, 15 case reports, and 11 other reports and reviews. Additionally, 10 manuscripts were identified during reference cross-check.

The results of our review can be divided into three main categories: diagnostic imaging of VP, pharmacological treatment, and surgical treatment. These categories are discussed below.

### Imaging diagnostics

The gold standard of imaging in NVC is magnetic resonance imaging (MRI) [37]. The test allows evaluation of

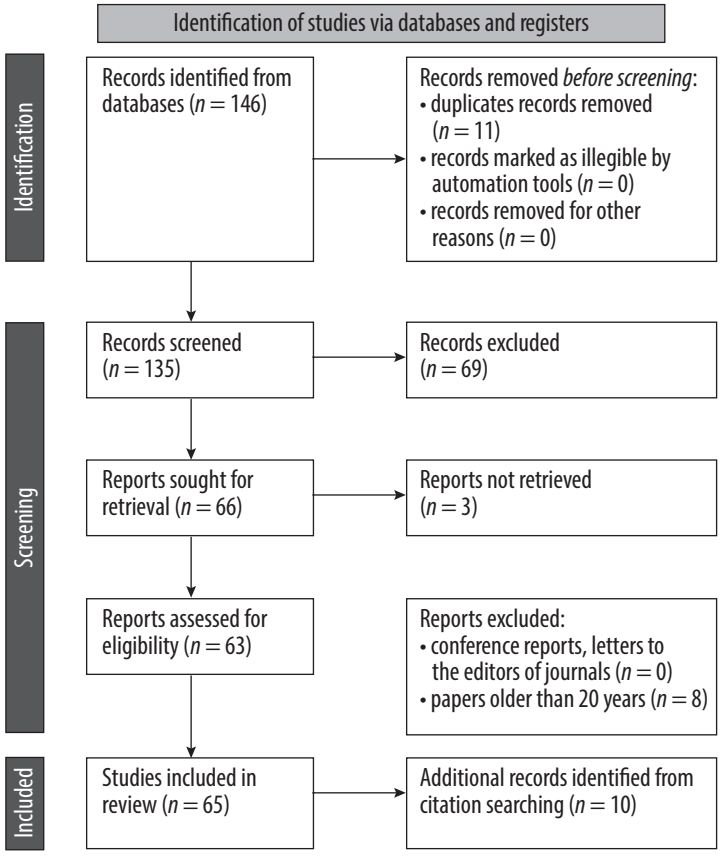


**Figure 2.** PRISMA flowchart for systematic reviews

the location of the NVC and differential diagnosis with other forms of lesions of the cerebello-pontine angle and central nervous system, which can manifest as dizziness or a sense of instability [38,39]. Karamitros et al. [40] found the 3D CISS (constructive interference in steady state) sequence to be the most diagnostic. It has a high signal and high matrix resolution, which allows for the evaluation of fine structures. The diagnostic criterion was defined as the absence of cerebrospinal fluid between the vessel and the affected nerve [40]

A case of VP was presented by Badełek-Izdebska and Zawadzka-Głos [41] in 2015 where the complaints were accompanied by sudden unilateral sensorineural hearing loss. The authors described NVC within the vestibulocochlear nerve caused by compression of the anterior inferior cerebellar artery; pharmacological therapy was introduced and gradual resolution of symptoms was observed over time. In the three case series of VP described by Lehnen et al. [18], posturography and VNG showed no abnormalities (as in the disease course we will describe later). Although the imaging studies in all three patients visualized NVC, the authors emphasise that VP is diagnosed based on clinical symptoms, and the visualisation of NVC on MRI is only one of the subsidiary criteria for diagnosing the disease [18].

Be that as it may, not all cases of image-confirmed neural–vascular contact result in clinical symptoms. Arteries are thought to be more likely to cause symptomatic NVC than veins, probably due to higher pressure and pulsation. The anatomical location of NVC should also be considered: the most sensitive area to pressure is the transition zone between the central and peripheral myelin. Therefore, accurate knowledge of the location and morphology of the transition zone is essential for interpreting neuroimaging findings. In the case of the vestibulocochlear nerve, this zone is longer than in the case of other cranial nerves [42,43].

In a study by Hufner et al. based on an analysis of 32 symptomatic patients diagnosed with VP, neurovascular conflict at the vestibulocochlear nerve was confirmed by MRI in 95% of patients, of which bilateral NVC was found in 42% [44]. Important data on diagnostic imaging is provided by a retrospective cohort study conducted by Steinmetz et al. [45]. Based on analysis of clinical data of 146 patients meeting VP criteria (73 pVP patients and 73 dVP patients), 10% of patients did not show NVC on MRI scans [45]. The authors hypothesize that there may be another excitatory phenomenon associated with the vestibulocochlear nerve, such as spontaneous membrane discharges or central brainstem paroxysmia, which may mimic the course of VP in patients and without giving evidence of NVC on imaging. A similar disjunct is also observed in other nerve compression syndromes such as trigeminal neuralgia or hemifacial spasm. In addition, studies using 7-tesla MRI on patients who have had clinically confirmed VP, and NVC evident in 1.5 and 3-tesla machines, have shown the absence of structural changes in the nerve [46]. This supports the hypothesis of a different pathophysiological mechanism of the disease than direct compression of the vessel on the nerve [46]. In **Table 2**,

**Table 2.** Summary of literature review of imaging diagnostics associated with VP

| Authors and year | Study population | Results |
|---|---|---|
| Badełek-Izdebska et al. 2015 [41] | 15-year-old woman with vertigo and hearing loss | NVC found in MRI, good response to VP treatment |
| Bae et al. 2017 [39] | 15 CBZ-responding patients with tinnitus and 8 control subjects | NVC was observed in MRI significantly more often in symptomatic group |
| Best et al. 2013 [43] | 20 VP patients and control group of 20 patients with trigeminal neuralgia | NVC observed in MRI in all VP patients and in 7 patients with trigeminal neuralgia |
| Hufner et al. 2008 [44] | Follow-up examination of 32 symptomatic VP patients | NVC observed in MRI in 95% of cases, bilaterally in 42% of cases |
| Lehnen et al. 2015 [18] | 3 pediatric patients with non-specific neuro-otologic symptoms | NVC in MRI in all cases |
| Rommer et al. 2015 [46] | 6 VP patients with confirmed NVCC in MRI | 7T MRI confirmed the pathologic NVC as shown in 1.5T and 3T MRI, able to exclude evident structural lesions of the CN VIII in all patients |
| Serra et al. 2010 [37] | 15 patients with non-specific neuro-otologic symptoms | MRI revealed 14 vascular pathologies of the AICA artery and 1 of the vertebral artery. NVC bilaterally present in 5 patients |
| Sivarasan et al. 2019 [38] | 9 patients with VP and control group with 20 patients with unilateral tinnitus | NVC was observed in MRI in all 9 VP patients; also in 9 patients with unilateral tinnitus |
| Steinmetz et al. 2022 [45] | 146 patients (73 dVP, 73 pVP) | NVC observed in MRI in 90% of cases |

Note: CBZ, carbamazepine; CN VIII, vestibulocochlear nerve; dVP, definite vestibular paroxysmia; MRI, magnetic resonance imaging; pVP, probable vestibular paroxysmia; VP, vestibular paroxysmia

we summarise the results of our review regarding diagnostic imaging. The table includes original research papers as well as case reports.

## Pharmacological treatment

The mainstay of VP treatment is the sodium channel blockers carbamazepine (CBZ) (200–800 mg/day) or oxcarbazepine (OXC) (300–900 mg/day) [47]. It is recommended that the drugs be administered at the lowest daily dose possible until the discomfort is relieved. If there is insufficient improvement, the dose can be increased to the target dose, but not more than the maximum recommended daily dose [44,48-52]. A good clinical response to drug treatment with this regimen is one of the criteria for the diagnosis of VP [53]. A study of the course of the disease in 32 patients over 3 years showed a significant and consistent reduction in episode frequency, which fell to 10% of the initial value, as well as a reduction in the intensity and duration of episodes [44]. Xue et al. conducted a clinical study using OXC together with betahistine mesylate (BMT) in patients with hypersensitivity to CBZ; the results showed increased efficacy of this combination compared to monotherapy with OXCs [54]. Yi et al. in a retrospective study on the support of BMT therapy with OXC in patients with paroxysmia also demonstrated the greater efficacy of this regimen [55].

Alternative treatment regimens include sodium channel blockers such as phenytoin (100–300 mg 3 × daily) or valproic acid (100–300 mg 3 × daily) [56]. Strupp et al. also demonstrated the effectiveness of lacosamide (100–200 mg 2 × daily) [57].

In **Table 3**, we summarise the results of our review regarding pharmacological treatment. Again, the table includes original research papers and case reports.

## Surgical treatment

Although there are reports of partial successes and one clinically well-documented case, microvascular decompression (MVD) should only be performed if the diagnosis is confirmed, the affected side can be identified, and treatment with CBZ or OXC was initially effective but produced side-effects [58,59]. A preoperative high-resolution MRI scan is recommended because of the frequent difficulties arising from abnormalities in vascular anatomy in patients with VP [60]. Bernard-Demanze et al. [61] describe the case of an 84-year-old female patient with NVC within the vestibulocochlear nerve, in whom they performed a microvascular decompression procedure. The intervention resulted in complete removal of dizziness and tinnitus, which had a positive effect on the patient's balance and improved quality of life [61]. However, due to the anatomical location and the high risk of damage to the vestibulocochlear nerve or facial nerve, procedures in this area should be considered a last resort therapeutic option [62,63]. In **Table 4** we summarise the results of our review regarding surgical treatment of VP. The table includes original research papers and case reports.

## Presentation of clinical cases

### Case 1

An active 31-year-old male, a personal trainer by profession, presented to our clinic with paroxysmal, short-lasting

**Table 3.** Summary of literature review of pharmacological treatment associated with VP

| Authors and year | Study population | Results | Conclusions |
|---|---|---|---|
| Bayer et al. 2018 [47] | 43 VP patients treated with OXC (300 mg) or placebo | 18 patients completed trial; risk of experiencing a day with at least one attack was 0.41 under OXC treatment, and 0.62 under placebo | Trial showed significant reduction of VP attacks under OXC treatment compared to placebo |
| Chen et al. 2022 [52] | 29 VP patients treated with low-dose OXC (300–600 mg) | 22 patients treated with OXC reported a long-term good therapeutic effect; 19 patients had NVC confirmed in MRI | Pharmacotherapy with OXC is effective long-term treatment in VP patients; 20% patients had long-term remission off medication; presence or absence of MRI evidence of NVC does not predict treatment response or remission |
| Hanskamp et al. 2022 [49] | Follow-up examination of 61 VP patients, 12 treated with CBZ (av. 200 mg) | 7 patients treated with CBZ reported a positive effect; 31 of all patients reported limitations in Health-related Quality of Life | Pharmacotherapy with CBZ is effective in VP patients; VP symptoms are associated with decrease in quality of life of those affected |
| Hufner et al. 2008 [44] | Follow-up examination of 32 symptomatic VP patients provided with CBZ or OXC | Treatment with CBZ led to a significant reduction in the attack frequency, intensity, and duration | Response to pharmacotherapy is crucial to diagnosis confirmation |
| Strupp et al. 2019 [57] | 7 VP patients provided with lacosamide (200–400 mg) | 2 patients reported reduction in the frequency of vertigo duration; 3 patients reported lower intensity of attacks | Consider lacosamide as well-tolerated alternative to CBZ or OXC for treatment in VP |
| Xue et al. 2018 [54] | 92 VP patients treated with CBZ (200–300 mg) + BMT (12–18 mg) tablets and 93 VP patients treated with OXC (200–300 mg) + BMT (12–18 mg) tablets | Both groups had similar average vertigo frequency, vertigo score, vertigo duration, and response rate; incidence of side effects was significantly higher in the CBZ + BMT group than in the OXC + BMT group | OXC + BMT may be suitable as an alternative method in VP patients with CBZ hypersensitivity |
| Yi et al. 2016 [55] | 196 VP patients; 73 patients treated with CBZ (200–600 mg), 65 with CBZ (200–600 mg) + BMT (20–40 mg); 58 patients treated with OXC (600–900 mg) + BMT (20–40 mg) | CBZ + BMT group had a greater reduction in the frequency of vertigo; the incidence of side-effects was highest in the CBZ group, second in the CBZ + BMT group, and lowest in the OXC + BMT group | BMT as an augmentation for CBZ or OXC might be an effective and well-tolerated treatment option in VP |

Note: BMT, betahistine mesilate; CBZ, carbamazepine; NVC, neurovascular conflict; OXC, oxcarbamazepine; VP, vestibular paroxysmia

vertigo. The episodes were not accompanied by hearing disorders. He denies any chronic diseases. Episodes have occurred more than 5 times this year, and similar episodes have recurred for the last 3 years. In the past, he has been treated with oral betahistidine preparations and cycles of vestibular rehabilitation, with short-term results. In addition, he suffers from significant anxiety due to vertigo attacks. Diagnostic imaging (RM of the cerebello-pontine area) performed outside our department did not reveal pathological changes in the cerebello-pontine area. The otolaryngological examination showed no pathological changes in the auditory organ. At admission, audiological diagnostics were performed: threshold tonal audiometry and impedance audiometry (PTA 10 dB bilaterally), type A tympanogram bilaterally, and stapedial muscle reflex present at 80 dB. In addition, otoneurological diagnostics were performed: Dix–Hallpike maneuver – nystagmus not visible (evaluation according to the HINTS protocol); head impulse test (HIT) – normal result; evaluation of spontaneous nystagmus (absent); test for skew (absent). Unterberger tests were performed three times – stable for 30 seconds; Romberg – stable for 30 seconds. Diagnostic videonystagmography (rotating chair test, caloric test, spontaneous and gaze-evoked nystagmus tests, saccadic movement tests) and vestibular evoked myogenic potentials (VEMP) were performed – no evidence of damage to the peripheral balance system were found. Using ICVD criteria, a diagnosis of probable vestibular paroxysmia was made, treatment with carbamazepine 200 mg 1 × daily was started, and a psychiatric consultation was ordered to rule out psychogenic dizziness. At a follow-up visit one month later, the patient reported complete resolution of symptoms;

**Table 4.** Summary of literature review of surgical treatment associated with VP

| Authors and year | Number of subjects | Results | Conclusions |
|---|---|---|---|
| Bernard-Demanze et al. 2015 [61] | 84-year-old woman with NVC | Positional vertigo and tinnitus disappeared after MVD; speech intelligibility and postural control also improved | MVD was effective in reducing postural deficits in this patient |
| Liu et al. 2020 [58] | Observation after MVD of 54 patients with trigeminal neuralgia, hemifacial spasm and glossopharyngeal neuralgia, 12 with coexistent VP | 11 patients reported no recurrence of VP symptoms after MVD | MVD is an effective method of treatment in VP with NVC in patients who are resistant to or poorly tolerate pharmacotherapy |
| Møller et al. 1986 [59] | 21 patients with disabling positional vertigo | 16 improved after MVD, 2 patients had no improvement, and 1 patient suffered a cerebellar hematoma | MVD is an effective method of treatment in NVC, but significant complications may occur |
| Strupp et al. 2013 [62] | 55-year-old man with NVC and VP with poor tolerance of CBZ therapy | No further symptoms after MVD | MVD is an effective method of treatment in VP with NVC in patients who poorly tolerate pharmacotherapy |
| Tanrikulu et al. 2014 [60] | 8 patients with NVC syndromes treated with MVD | 5 cases of SCA, 2 cases of AICA, and 1 case venous NVC | All patients relieved of symptoms after MVD |

Note: AICA, anterior inferior cerebellar artery; CBZ, carbamazepine; MVD, microvascular decompression, NVC, neurovascular conflict; SCA, superior cerebellar artery; VP, vestibular paroxysmia

in addition, a psychiatric consultation revealed anxiety attacks secondary to the dizziness. The patient remains under observation at our clinic.

### Case 2

A 17-year-old woman presented to our clinic for diagnosis due to paroxysmal vertigo which had been occurring for about a year (more than 10 episodes in the last month). The episodes were short, vertiginous in nature, and are not provoked by a change in body position. They subsided within a few minutes. She reported no hearing abnormalities or chronic conditions. She has so far been treated with betahistidine preparations without significant effect. Otoscopic examination revealed no pathological changes. Romberg and Unterberger tests were performed three times – stable for 30 seconds. Dix–Hallpike maneuver was performed – no test-induced nystagmus was observed. HIT test was unremarkable. Spontaneous nystagmus was not observed. Audiological diagnosis was performed – threshold tonal audiometry and impedance audiometry – bilateral PTA 10 dB, type A tympanogram, stapedial muscle reflex present at 90 dB. A VNG examination was performed according to the protocol described above, no features of peripheral dysfunction were noted, and a symmetrical response was obtained in caloric testing. Magnetic resonance imaging performed in an outpatient clinic and provided by the patient showed no lesions of the cerebello-pontine region.

Using ICVD criteria, a diagnosis of probable vestibular paroxysmia was made, and treatment with carbamazepine 200 mg was started. In a follow-up examination after 1 month of therapy, the patient reported a reduction in the frequency of episodes, and the dose was increased to 400 mg per day. In another follow-up examination one month later, the patient no longer reported vertigo. According to the criteria listed in **Table 1**, the diagnosis was reclassified as vestibular paroxysmia. The patient remains under observation at our clinic.

In both clinical cases presented above, no pathological changes were identified, as in the MRI study of Steinmetz et al. [45]. Estimates are that about 10% of VP cases do not involve clear manifestation of vascular conflict at the vestibulocochlear nerve. We have introduced pharmacological treatment as suggested in the findings of this review and the Bárány Society criteria – the first line of treatment is carbamazepine. Resolution of symptoms was observed which re-classified these cases from probable VP to vestibular paroxysmia.

## Conclusions

We have reviewed recommendations, diagnostic imaging, and suggested therapies for vestibular paroxysmia. Studies covered in the review included the consensus currently proposed by the Bárány Society, clinical studies, case reports, and systematic reviews of the literature. According to the reports presented, MRI examination is the basis for imaging neurovascular conflict. However, in a small number of patients with VP, neurovascular conflict cannot be seen on MR imaging. The cases we presented showed no pathology on MRI imaging. The applied treatment with carbamazepine, according to the recommendations from ICVD, resulted in the disappearance of the patients' vertigo. This correlates with many reports regarding the effectiveness of pharmacotherapy with dibenzoazepine

derivatives. Most often, patients require long-term treatment with these preparations. Microvascular decompression surgery is a complex procedure used for patients intolerant to pharmacological regimens who have severe symptoms and neurovascular conflict confirmed by MRI. This procedure can be considered for selected cases.

In summary, a diagnosis of vestibular paroxysmia is primarily based on a thorough clinical examination, otoneurological testing, and diagnostic imaging. These help exclude other causes of these symptoms, which are more common in those with vestibular disorders. The exact pathomechanism of the disease is unclear, as in some cases vascular conflict with the vestibulocochlear nerve cannot be seen on MRI.

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