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CLINICAL STUDY

Secondary complications of vestibular neuropathy

Pavol SKACIK[1,2], Stefan SIVAK[1,2], Egon KURCA[1,2]

Neurology Department, University Hospital Martin, Martin, Slovakia.
pavolskacik@gmail.com

ABSTRACT
Vestibular neuropathy represents the third most common cause of peripheral vestibular disorders. The clinical picture of the disease is characterized by acute vertigo with vegetative accompaniment. A typical course is monophasic with the resolution of symptoms within a few days to complete adjustment.
In some patients, secondary complications may occur, which we define into three groups: organic, cognitive, and functional. The occurrence of complications and their persistence is often an overlooked issue, which does not receive sufficient attention and can lead to a significant deterioration in quality of life. Therefore, the management of these patients requires a comprehensive approach and interdisciplinary cooperation between neurologists, physiotherapists, psychologists, and psychiatrists.
In the following text, the authors attempt to elucidate this issue further *(Fig. 1, Ref. 49).* Text in PDF www.elis.sk
KEY WORDS: vestibular neuropathy, secondary complications, vertigo, acute vestibular syndrome.

Introduction

Vestibular neuropathy represents the third most common cause of peripheral vestibular disorders, following benign paroxysmal positional vertigo (BPPV) and Ménière's disease (43). It was first described in 1952 (39). The etiology of this condition is presumed to be viral infection (HSV-1) with local inflammation and edema of the vestibular portion of the eighth cranial nerve and Scarpa's ganglion (15).

The clinical picture of the disease is characterized by acute vertigo with vegetative symptoms. Objectively, there is a presence of horizontal-rotational nystagmus with a fast component directed towards the contralateral side of the damaged vestibular apparatus, a positive head impulse test towards the lesion side, asymmetry of vestibulospinal reflexes (Hautant test, Unterberg test, Romberg test) (48).

The therapy of vestibular neuropathy can be divided into pharmacological and non-pharmacological. Pharmacological therapy involves pulse therapy with corticosteroids, antivirotics, anti-vertiginous drugs, and antiemetics. Non-pharmacological therapy mainly includes vestibular rehabilitation (48).

In the following text, the authors address the complications caused by this condition.

Classification of secondary complications of vestibular neuropathy

The precise boundary between individual categories may not always be clearly defined, and there may be overlapping between them. Complications can be defined into three basic categories:

1) Primary organic (chronic unilateral vestibular dysfunction, recurrent vestibular neuritis, secondary BPPV, musculoskeletal disorders, oscillopsia).
2) Cognitive and neurobehavioral (cognitive dysfunction, anxiety-depressive disorder)
3) Functional neurological disorders (PPPD, functional posture and gait disorders).

The overlap of individual categories is a relatively common phenomenon. This can be explained by the following example. A patient overcomes an attack of vestibular neuropathy. Subsequently, they experience symptoms such as dizziness of varying intensity, duration, and character for months to years. Sometimes, these symptoms are provoked by visual stimulation (e.g, moving objects in the visual field). The patient also reports a sensation of imbalance in posture and gait. Psychological examination reveals reactive anxiety disorder. In this patient, clinically diagnosed chronic unilateral vestibular dysfunction (category I) coexists with anxiety disorder (category II) and functional neurological disorder – visual vertigo (currently classified among PPPD) (category III) (Fig. 1).

Chronic unilateral vestibular dysfunction

Chronic unilateral vestibular dysfunction (CUVD) encompasses a broad category of vestibular disorders characterized by partial or complete loss of function of one vestibular apparatus (23).

One such condition may be the aftermath of vestibular neuropathy, where inadequate compensation for the loss of the vestibular apparatus occurs. After an episode of vestibular neuritis, patients may report persistent difficulties for months to years. For example, in a study by Kammerlind (21). 50% of

[1]Neurology Department, University Hospital Martin, Martin, Slovakia, and [2]Neurology Department, University Hospital Martin, Martin, Slovakia

Address for correspondence: Pavol SKACIK, MD, Neurology Department, University Hospital Martin, Kollarova 2, SK-036 01 Martin, Slovakia. Phone: +421 949 705 506

patients reported dizziness-related difficulties 3 to 6 years after an acute vestibular neuritis episode. This is associated with a lower quality of life and increased incidence of anxiety and depressive disorders. Objective signs of vestibular apparatus damage persist in 30% of patients more than 1 year after a vestibular neuritis episode (24).

To diagnose CUVD, signs of unilateral vestibular apparatus damage must be present. This can be determined through clinical examination (head impulse test, head shake nystagmus, Unterberg test) or with the use of caloric testing or video head impulse testing (currently preferred in diagnosis) (23).

Symptoms reported by patients with CUVD are extremely heterogeneous. Among the most common are chronic persistent or recurrent (worsening in attacks) dizziness, balance disturbances, visual dizziness, and oscillopsia. Other difficulties may include an exhaustive syndrome, cognitive function disorders, and autonomic symptoms.

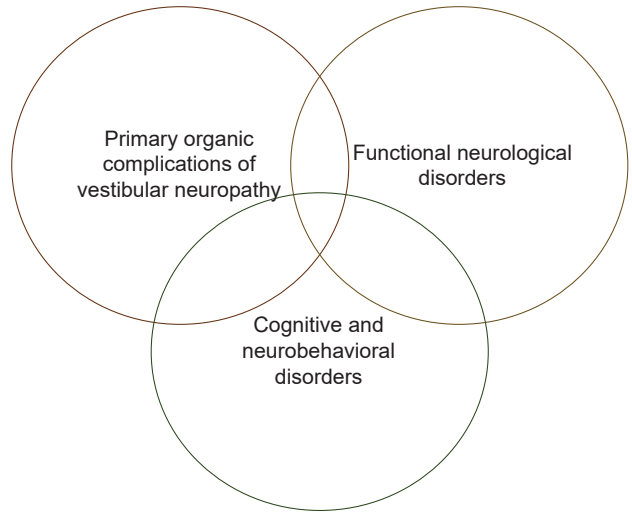

Fig. 1. Basic categories of secondary complications of vestibular neuritis depicting their overlap and possible combinations.

Recurrent vestibular neuropathy

Initially, vestibular neuropathy was considered a disease without recurrences (12). Currently, it is proven that recurrences occur in approximately 2 to 10% of patients (20, 26, 29). In most cases, there is one relapse of the disease, but multiple relapses can occur, either on the ipsilateral or contralateral vestibular apparatus (5, 20).

When a recurrence of acute vestibular syndrome occurs, it is important to exclude other etiologies that could mimic recurrent vestibular neuritis (differential diagnosis of acute vestibular syndrome).

Secondary benign paroxysmal positional vertigo (BPPV)

BPPV represents the most common cause of positionally triggered episodic provoked vestibular syndrome. BPPV is classified into idiopathic, where the cause is unknown and is presumed to be a combination of factors such as age, degenerative changes in the vestibular apparatus, otolith metabolism disorder, vitamin D hypovitaminosis, decreased estrogen levels in women. Secondary causes may include head injuries, Ménière's disease, and the aftermath of vestibular neuropathy (20).

The mechanism by which secondary BPPV arises can be attributed to the anatomical distribution of the vestibular nerve and inner ear. Inflammation more commonly damages the upper branch of the vestibular nerve. During vestibular neuropathy, direct damage to the utricle macula occurs, leading to the release of otoliths, most commonly into the posterior semicircular canal (45). The incidence of secondary BPPV varies in the literature, studies focusing on monitoring BPPV after vestibular neuropathy report approximately 10 to 15% of cases (20, 26, 29). The average onset time of BPPV is approximately 18 days (28). Secondary BPPV has several characteristics. Patients have a lower average age, predominantly damage the posterior semicircular canal, have lower efficacy of repositioning maneuvers, and experience more frequent recurrence of symptoms (3).

Management and therapy do not differ from primary BPPV, and the gold standard is the performance of repositioning maneuvers according to the affected semicircular canal. In cases of refractory BPPV, repeated visits to the doctor with the need for repositioning maneuvers may be necessary, and treatment may last for several weeks. Surgical therapy is rarely approached.

Visual symptoms of unilateral vestibular dysfunction

Among the two main symptoms in this group are oscillopsia and visual vertigo, which are currently classified under the diagnosis of PPPD (see below). Oscillopsia represents an illusion of movement of the visual image due to a disturbance in the stabilization mechanisms of the observed image on the retina. It is further divided into oscillopsia present at rest and during head (or body) movements.

In patients with unilateral vestibular dysfunction, it is typically present during head movement. This is common in the subacute stage after vestibular neuritis when the subjective feeling of dizziness is not present, but patients report sensations of the surroundings spinning, instability of the image, which are exacerbated during head movements. These symptoms arise from a disturbance in the vestibulo-ocular reflex (44).

Segmental syndrome of the cervical spine and musculoskeletal disorders

The sudden loss of vestibular function is associated with a behavioural reaction in individuals, wherein they attempt to minimize the subjective feeling of dizziness by reducing visual input (closing eyes). assuming a lying position with the healthy vestibular apparatus closer to the ground. Upon standing and walking, there is greater caution with a heightened awareness of concentrating on maintaining stability and minimizing spontaneous head movements.

This change in vestibular system function leads to an influence on the musculoskeletal system (alteration of muscle tone, affecting postural mechanisms) through vestibulospinal, cervicospinal, and vestibulocervical reflexes. Patients who have experienced an attack of vestibular neuropathy often have associated neck pain, and the incidence of pain increases over time since overcoming unilateral vestibular apparatus damage. On the other hand, the presence of neck pain is a strong predictor of persistent dizziness in this patient population (49). Other frequently described musculoskeletal disorders include postural abnormalities (protraction of the head and shoulders). restriction of respiratory movements, increased muscle tone of the trunk and lower limbs. Vestibular rehabilitation (training of static and dynamic balance, gaze stabilization) represents the mainstay of non-pharmacological therapy for these patients. In addition, physiotherapeutic management should also focus on compensating for other musculoskeletal disorders. This comprehensive approach has a positive impact on persistent dizziness and balance disorders (50).

Therefore, clinical examination of patients, in addition to basic neurological and otoneurological examination, should focus on screening for musculoskeletal system disorders with subsequent appropriate management.

Cognitive disorders

Bilateral vestibulopathy is traditionally associated with impairments in cognitive functions. These predominantly involve visuospatial disturbances as a result of damage to vestibulo-cortical projections responsible for orientation and navigation in space. Structurally, patients with bilateral vestibulopathy often exhibit more pronounced hippocampal atrophy (7).

In the case of unilateral vestibular dysfunction, the situation is somewhat more complex. Some authors do not describe significant changes compared to healthy controls (17). According to others, spatial memory and navigation impairments are also present in this group of patients. In addition to visuospatial function disorders, impairments in short-term memory, executive functions, or attention may be present and may be influenced by lateral damage to the vestibular apparatus (1, 34).

Anxiety and depression

Patients with vestibular system disorders have a higher risk of developing anxiety and depressive disorders (14). The structures responsible for processing vestibular signals, regulating emotions, and mood have close anatomical and functional relationships at various levels of the central nervous system. They are divided into three subgroups: cognitive-behavioural, sensorimotor, and interoceptive, and contribute to the development of anxiety disorders, vertiginous states, or even migraines (2).

Therefore, in patients after an attack of vestibular neuropathy and with persistent chronic vestibular symptoms, it is important to conduct examinations focused on anxiety or depressive disorders, which can be detected using scales such as the Hospital Anxiety and Depression Scale and the Dizziness Handicap Inventory (5).

Persistent Postural-Perceptual Dizziness (PPPD)

PPPD represents a complex functional neuro-vestibular disorder that arises from the incorrect processing of vestibular and proprioceptive stimuli compared to the expected position of body parts from higher cortical centers (38).

It is a chronic condition characterized by the presence of persistent (chronic or paroxysmal) vestibular symptoms, which are exacerbated by movement, upright posture, and complex visual stimuli. Etiologically, there may not be a clear precipitating cause, but it can often be triggered by an organic cause – in this case, an acute vestibular syndrome due to vestibular neuropathy. This is described in 2% of patients diagnosed with PPPD (47). Other causes that may trigger functional balance disorders include BPPV, vestibular migraine, psychological stress, panic attacks, injuries, and so on. It is also worth mentioning that PPPD can occur in a population without any vestibular (or other) insult (16).

An interesting fact is that patients with PPPD may have secondary functional posture and gait disorders and psychiatric comorbidities (33). When vestibular symptoms persist after an attack of vestibular neuropathy, an organic and functional basis for persistent difficulties may be combined (32).

Visual vertigo represents an inadequate response to moving objects in space as a result of misinterpreting visual and sensory signals (vestibular, proprioceptive). These symptoms may be present in patients in the acute as well as chronic stage of vestibular neuropathy and other vestibular disorders. Difficulties may be exacerbated when observing moving objects, increasing the risk of developing anxiety and depressive disorders. The exact mechanism of visual vertigo is not described, but it may involve the influence of misinterpreting visual, sensory, and vestibular signals (9, 10).

Functional posture and gait disorders

Functional neurological disorders represent a heterogeneous group of motor, sensory, or cognitive symptoms attributed to a functional disorder of the nervous system and are a common cause of disability with a co-incidence of psychiatric comorbidities (11, 40).

Functional disorders represent the second most common reason for visiting a neurologist after headache (4). They are typically characterized by inconsistency and incongruence of symptoms (31). In some patients, a triggering event can be identified, which may be correlated with the patient's difficulties (the so-called triggering event). Such a cause may include an acute vestibular syndrome due to vestibular neuropathy, which leads to abnormal afferentation (or deafferentation) from the vestibular system, is associated with activation of the sympathetic flight or fight response, and leads to a change in the processing of afferent stimuli in higher cortical analyzers. There is increased conscious focus on a particular movement (in this case, during standing or walking) and the assumption that the movement is performed incorrectly, leading to a change in the movement program and subsequently to its disruption (35).

In some patients after overcoming the acute stage of vestibular neuropathy, significant postural and gait ataxia may persist, making them almost unable to stand and walk independently. This may often appear as a suspicious central lesion of the vestibular system, but additional examinations such as brain MRI are negative, as are the results of other paraclinical otoneurological examination methods, which would suggest a central cause. The clinical picture of vestibular ataxia is modified by the variability, distractibility, and inconsistency of the clinical presentation of posture and gait disorders.

Conclusion

Vestibular neuropathy in a large portion of patients runs as a monophasic condition with the resolution of difficulties within a few days to complete adjustment. The occurrence of complications and their persistence is often an overlooked issue, which does not receive sufficient attention and can lead to a significant deterioration in quality of life. Therefore, the management of these patients requires a comprehensive approach and interdisciplinary cooperation between neurologists, physiotherapists, psychologists, and psychiatrists.

Learning points

- Vestibular neuropathy presents with acute vertigo and vegetative symptoms, objective signs such as horizontal-rotational nystagmus and positive head impulse test.
- Complications of vestibular neuropathy can be categorised into three main categories: organic, cognitive/neurobehavioral and functional with significant overlap between categories.
- Complications and their persistence can significantly impact patients quality of life.
- Thorough assessment and management of complications are essential to mitigate their impact on patients' well-being and overall functioning.
- Managing complications of vestibular neuropathy requires a multidisciplinary approach involving neurologist, physiotherapist, psychologist and psychiatrist.

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