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VESTIBULAR PAROXYSMIA (CASE REPORT, DISCUSSION)

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We described the clinical case of vestibular paroxysmia. Male, 58 years old complained of attacks of intense vertigo lasting 15-20 seconds with a feeling of rotating objects counterclockwise. During an attack patient noted the crackling noise in the right ear, a feeling of instability, profuse sweat, nausea. Attacks occur spontaneously, without any triggers, 5-7 times a month during the last 5 months. There are no symptoms between the attacks. Patient consulted a neurologist who diagnosed benign paroxysmal positional vertigo. The prescribed treatment did not change the frequency and intensity of vertigo attacks. On neurological examination no abnormalities were found. MRI brain showed light leukoaraiosis. Audiometry: bilateral high-frequency sensorineural insufficiency. Electroencephalography did not show epileptiform activity. Otolaryngologist: slight bilateral sensorineural hearing loss. Clinical findings were consistent with the diagnosis of vestibular paroxysmia. The patient was prescribed carbamazepine 200 mg twice a day for 1 month. 1 month later at the visit, the patient reported no attacks during the treatment period, so patient recommended to use carbamazepine 100 mg twice a day for the next 3 months. 3 months later, at the follow-up visit, the patient reported only a single attack of mild vertigo during the last 3 months. It was recommended to continue taking carbamazepine 100 mg twice a day for 6 months, followed by a scheduled visit. So, neurologists need to know and actively use in their practice diagnostic criteria of vestibular paroxysmia.

Key words: *vestibular paroxysmia, diagnostic criteria, etiopathogenesis, treatment.*

Connection of the publication with planned research works.
The work is a fragment of the research «Optimization of diagnosis, prognosis and prevention of neuropsychological disorders in organic diseases of the nervous system», state registration number 0120U104165.

Introduction.
Up to now, in Ukraine neurologists do not use enough in their practice international criteria for paroxysmal diseases [1].

A wide range of neurological and otological disorders can be presented as episodic vestibular syndrome [2]. Along with the well-known diseases (benign paroxysmal positional vertigo, Meniere's disease), a number nosologies with episodic vestibular syndrome remain unknown to the majority of neurologists. The latter leads to using of non-informative investigations, misdiagnoses and prescription of ineffective treatment. One of the rare type of episodic vestibular syndrome is vestibular paroxysmia (VP) – debilitating clinical condition characterized by brief recurrent episodes of spontaneous or positional vertigo that respond well to carbamazepine or oxcarbazepine [3, 4].

The aim of the study.
To describe the clinical case of VP and thus demonstrate the importance of knowing the diagnostic criteria of VP.

Object and research methods.
Complaints, medical history, life history, objective and neurological examinations, neuroimaging and neurophysiological investigations in patient with VP.

Research results.
Patient of male gender, 58 years old complained of attacks of intense vertigo lasting 15-20 seconds with a feeling of rotating objects counterclockwise. During an attack patient noted the crackling noise in the right ear, a feeling of instability, profuse sweat, nausea. Within next 10-15 minutes after the end of vertigo attack patient had a feeling of lightness in the head, palpitation, sensation of heat in the body.

Attacks occur spontaneously, without any triggers, 5-7 times a month. The first attack happened 5 months ago while driving in the city, because of which patient almost got into a car accident. Since then, the patient has been afraid to drive.

Patient is very afraid of attacks, because they are accompanied by a very unpleasant feeling of vertigo. In addition, patient worried that the attacks of vertigo could be a symptom of some severe unrecognized disease.

He had no symptoms between the attacks except of *constant high-pitched hissing tinnitus* in both ears that got worse in silence.

Patient consulted a neurologist who diagnosed benign paroxysmal positional vertigo. Patient was taking betahistine 24 mg twice a day for 1 month, phenibut 25 mg three times a day for 1 months. The treatment did not change the frequency and intensity of vertigo attacks.

7 years ago patient had brain concussion due to slipping on the ice. Patient has been suffering from arterial hypertension for the last 3 years, he takes bisoprolol 2,5 mg a day.

On neurological examination no abnormalities were found. Spontaneous or gaze-evoked *nystagmus was not detected*. A bedside *head impulse test was negative*. Equilibrium and coordination were intact. Dix-Hallpike test did not induce positional nystagmus. Hearing evaluation using tuning forks was unremarkable. Patient was asked to complete a hospital anxiety and depression scale: anxiety subscale score – 18 points, depression subscale score – 10 points.

MRI brain showed non-specific white matter T2 hyperintense lesions in white matter around the ventricles of the brain (Fazekas grade 1). Audiometry diagnosed bilateral high-frequency sensorineural insufficiency. Electroencephalography did not show epileptiform activity. Otolaryngologist consultation: slight bilateral sensorineural hearing loss.

In sum, clinical findings were consistent with the diagnosis of VP according to the criteria proposed by the Barany Society [5]:

A) At least ten attacks of spontaneous spinning or non-spinning vertigo.

B) Duration less than 1 minute.

C) Stereotyped phenomenology in a particular patient.

D) Response to a treatment with carbamazepine/ oxcarbazepine.

E) Not better accounted for by another diagnosis.

The patient was prescribed carbamazepine (200 mg once a day for 3 days, then 200 mg twice a day for 1 month) as pathogenetic treatment for VP and etifoxine hydrochloride (50 mg three times a day for 1 month) as anxiolytic drug.

1 month later at the visit, the patient reported no attacks during the treatment period. Hospital anxiety and depression scale: anxiety subscale score – 10 points, depression subscale score – 8 points. Patient recommended to use carbamazepine 100 mg twice a day for the next 3 months.

3 months later, at the follow-up visit, the patient reported only a single attack of mild vertigo during the last 3 months. It was recommended to continue taking carbamazepine 100 mg twice a day for 6 months, followed by a scheduled visit

Discussion of the research results.

VP is an uncommon episodic peripheral vestibular disease causing acute or sudden brief attacks of vertigo or disequilibrium with or without the presence of auditory and vegetative clinical symptoms [6].

The main symptoms of VP are brief attacks of spinning or non-spinning vertigo which lasts a fraction of a second to a few minutes and occurs with or without ear symptoms (tinnitus and hypo- or hyperacusis). The attacks occur 30 times or more per day may often be triggered by particular head positions or hyperventilation. Unilateral hypoacusis or tinnitus occurs during the attack, occasionally or permanently. In the course of the disease, measurable vestibular and/ or cochlear deficits increase during the attack but are less pronounced during the attack-free interval (neurophysiological function tests used include audiogram, acoustic-evoked potentials, caloric testing, and test for subjective visual vertical). Attacks are improved or lessened by administering carbamazepine (even a low dosage is effective). No central vestibular/ocular motor disorders or brainstem signs are present [7].

As VP is a rare disease (the incidence<1 per 2 000 persons), there is no adequate information regarding the incidence and prevalence of VP documented in the medical literature [8]. The actual incidence of VP is unknown, but one study found it represented 3.9% of vestibular diagnoses at a tertiary outpatient clinic [9].

The mean age of VP patients was 51 years (range 25-67 years) [10].

In 28% of the patients, attacks occurred only at rest, in 22% - attacks were regularly precipitated by a provoking factor, whereas in 50%, attacks occurred spontaneously at rest or were provoked by a certain action [3]. The most frequently reported provoking factors were head (60%) or body (22%) turn. The most common accompanying symptoms were unsteadiness of stance or gait (75%), nausea/vomiting (41%), and tinnitus (28%) [3].

Moreover, VP can generally negatively affect the quality of life of patients and may lead to anxiety [3, 7].

The general difficulty in VP is that it is a purely clinical diagnosis that cannot be clearly proven by any objective parameter. Medical history, systematic clinical neurological examination, and neuro-otological examinations are required for assessment of the patients with VP. Otological examinations show normal external ear and tympanic membrane. Neuro-otological examination reveals no spontaneous or gazes induced nystagmus.

A battery of audiovestibular tests and MRI should be done in all patients of VP. MRI excludes structural brain lesions and could reveal vessel-vestibulocochlear nerve contact. The routine audiological evaluation includes pure tone audiometry for revealing the hearing status of the patients and also for ruling out the middle ear problems.

Identification of the affected side is often not possible [5]. However, approximately 50% of patients undergoing testing of vestibular and audiological function exhibit signs of a mild to moderate unilateral hypofunction during the attack-free intervals [3, 11].

Before making the diagnosis of VP, several diseases should be excluded. The important differential diagnosis of the VP includes benign paroxysmal positional vertigo, central positioning vertigo, Meniere's disease, Tumarkin's otolithic crisis, multiple sclerosis with the involvement of the brainstem, vestibular migraine, panic attacks, somatoform phobic postural vertigo, perilymph fistula, superior canal dehiscence syndrome, superior oblique myokymia, episodic ataxia type 2, orthostatic dysregulation, epileptic vestibular aura and vertebral artery occlusion syndrome [7, 12].

In benign paroxysmal positional vertigo, the attacks are induced by changes of head or body position relative to gravity, and the diagnosis can be proven by the diagnostic positional maneuvers (Dix-Hallpike test etc).

Tumarkin's otolithic crisis ("vestibular drop attacks") are sudden falls that usually not accompanied by vertigo and occur most often in patients with known Meniere's disease, typically while standing, whereas in VP the attacks occur in any body positions.

Paroxysmal brainstem attacks with vertigo, dysarthria or ataxia (after stroke or in multiple sclerosis) may be difficult to distinguish, as they also respond to low doses of sodium-channel blockers. It was shown that they may be caused by a brainstem lesion due to multiple sclerosis plaques or lacunar infarctions, which also lead to ephaptic discharges of neighboring fibers of the brainstem paths [13].

Vestibular migraine has the duration of the vertigo attacks from 5 min to 72 hours, current or previous history of migraine, most attacks being accompanied by other migrainous symptoms [14].

Vertigo due to vertebrobasilar transient ischemic attacks occurs as rule together with other neurological sign, most frequently with focal brainstem symptoms [15].

Panic attacks include a discrete period of intense fear or discomfort, in which the following symptoms develop abruptly and reach a peak within minutes: feeling dizzy, unsteady, lightheaded, or faint; nausea or abdominal distress; palpitations, and/or accelerated heart rate;

sweating; trembling or shaking; sensations of shortness of breath or being smothered; feeling of choking; chest pain or discomfort; derealization or depersonalization; fear of losing control or going insane; sense of impending death; paresthesias; chills or hot flashes. Moreover, panic attacks are longer than typical attacks of VP.

The cardinal symptoms of perilymph fistula (and superior canal dehiscence syndrome) are attacks of vertigo caused by changes in pressure, for example, by coughing, pressing, sneezing, lifting, or loud noises and accompanied by illusory movements of the environment (oscillopsia) and instability of posture and gait with or without hearing disorders. The attacks, which can last seconds to days, may also occur during changes in the position of the head (e.g., when bending over) and when experiencing significant changes in altitude (e.g., mountain tours, flights) [16].

In episodic ataxia type 2 duration of the vertigo-like attacks varies from several minutes to hours and more than 90% of the patients have cerebellar signs, in particular gaze-evoked nystagmus and downbeat nystagmus. Also, the onset of manifestations after the age of 20 is unusual [17].

Epileptic vestibular aura is primarily associated with temporal lobe seizures. Epileptic vestibular auras can manifest with short attacks of vertigo and nystagmus. Vestibular aura with additional aura symptoms is much more prevalent than isolated vestibular aura [18].

In orthostatic hypotension the symptoms occur when the patient stands up and may be associated with vertigo and downbeat nystagmus; the key to this diagnosis is measurement of supine and orthostatic blood pressure [19].

An assumed mechanism of VP is a neurovascular cross-compression of the vestibular nerve offended by a vascular loop [20]. The intra-cisternal part of the vestibular nerve, where the nerve is myelinated by oligodendroglia, is assumed to be the most vulnerable segment for VP [21]. A loop of the anterior inferior cerebellar artery seems to be involved most often, seldom the posterior inferior cerebellar artery, the vertebral artery, or a vein. The symptoms are thought to be triggered by direct pulsatile compression and ephaptic pathological paroxysmal interaxonal transmission between neighboring and possibly in part demyelinated axons. Another cause under discussion is central hyperactivity in the vestibular nuclei, which is induced and maintained by the compression [3, 7]. The demyelination in the root entry zone is usually not visible in standard MRI. Only 65.5% of VP patients had the evidence of vessel-nerve contact in MRI [4]. Since approximately 30% of healthy adults also show such a vessel–nerve contact, its specificity is low and therefore imaging is not part of the current diagnostic criteria [22].

Low-dose sodium channel blockers, e.g., carbamazepine/oxcarbazepine, are effective in the vast majority of VP, and are required for the diagnosis of definitive forms of VP [5]. It is important that in VP are often effective just low doses of carbamazepine (200-600 mg/d) or oxcarbazepine (300-900 mg/d) [23]. However, up to now the long-term course of VP as well as risk of the vertigo attacks recurrence are not entirely clear.

Conclusions.

Neurologists need to know and actively use in their practice diagnostic criteria of VP.

Prospects for further research.

Identification and analysis of clinical cases of other rare types of episodic vestibular syndromes.

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ВЕСТИБУЛЯРНА ПАРОКСИЗМІЯ (КЛІНІЧНИЙ ВИПАДОК, ОБГОВОРЕННЯ)

Дельва І. І.

Резюме. *Мета.* Описати клінічний випадок вестибулярної пароксизмії (ВП) і таким чином продемонструвати важливість знання неврологами діагностичних критеріїв цього захворювання.

Результати. Чоловік 58 років звернувся зі скаргами на напади раптового, інтенсивного головокружіння тривалістю 15-20 секунд з відчуттям обертання предметів проти годинникової стрілки. Під час нападів хворий відмічає тріск у правому вусі, відчуття нестійкості, профузний піт, нудоту. У наступні 10-15 хвилин після закінчення нападів хворого турбують відчуття легкості в голові та жару в тілі, серцебиття.

Напади виникають спонтанно, без будь-яких тригерів, 5-7 разів на місяць. Перший напад стався 5 місяців тому під час їзди автомобілем в місті, через що хворий ледь не потрапив у дорожньо-транспортну пригоду. Відтоді хворий боїться сідати за кермо.

Пацієнт панічно боїться нападів, оскільки вони супроводжуються вкрай неприємним відчуттям головокружіння. Крім того, хворий хвилюється, що ці напади можуть бути симптомом якогось важкого, досі нерозпізнаного захворювання. Окрім вищенаведених скарг пацієнт відмічає постійний шум в обох вухах, що посилюється в тиші.

Хворий звертався до невролога, було діагностоване доброякісне пароксизмальне позиційне запаморочення. За призначенням невролога пацієнт приймав бетагістин по 24 мг 2 рази на добу протягом 1 місяця, фенібут по 25 мг 3 рази на добу протягом 1 місяця. Лікування ніяк не вплинуло на частоту та інтенсивність нападів головокружіння.

7 років тому був струс головного мозку внаслідок падіння на льоді. Останні 3 роки страждає на артеріальну гіпертензію, приймає бісопролол по 2,5 мг на добу.

При неврологічному огляді відхилень не виявлено. Спонтанний та індукований ністагм відсутній. Тест поштовху голови – негативний. Рівновага і координація – непорушені. Тест Дікса-Холпайка не викликав позиційного ністагму. Пацієнт заповнив госпітальну шкалу тривоги та депресії: субшкала тривоги – 18 балів, субшкала депресії – 10 балів.

МРТ головного мозку – перивентрикулярний лейкоареоз 1 ступіня за шкалою Фазекас. При аудіометрії діагностована двобічна нейросенсорна недостатність. Електроенцефалографія: епілептиформної активності не виявлено. Консультація отоларинголога: незначна двобічна сенсоневральна приглухуватість.

Отримані клінічні дані повністю відповідають діагнозу ВП:

A) Принаймні 10 нападів спонтанного системного або несистемного головокружіння.

B) Тривалість нападів менше 1 хвилини.

B) Стереотипна феноменологія нападів.

D) Ефективність лікування карбамазепіном/оксарбазепіном.

E) Стан пацієнта не може бути пояснений будь-яким іншим діагнозом.

Пацієнту було призначено карбамазепін (200 мг 1 раз на добу протягом 3 днів, потім – 200 мг 2 рази на добу протягом 1 місяця) як патогенетичне лікування ВП та етіфоксину гідрохлорид (50 мг 3 рази на добу протягом 1 місяця) як анксіолітичний препарат.

Через 1 місяць, під час візиту пацієнт повідомив про відсутність нападів головокружіння протягом усього періоду приймання медикаментів. Госпітальна шкала тривоги та депресії: субшкала тривоги – 10 балів, субшкала депресії – 8 балів. Пацієнту рекомендовано приймати карбамазепін по 100 мг двічі на добу протягом наступних 3 місяців.

Через 3 місяці, під час контрольного огляду пацієнт повідомив лише про один напад легкого головокружіння з моменту попереднього візиту. Було рекомендовано продовжувати прийом карбамазепіну по 100 мг двічі на день протягом 6 місяців з подальшим плановим візитом.

Висновки. Неврологам необхідно знати та активно використовувати у своїй практиці діагностичні критерії ВП.

Ключові слова: вестибулярна пароксизмія, діагностичні критерії, етіопатогенез, лікування.

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Abstract. *Objective.* To describe the clinical case of vestibular paroxysmia and thus demonstrate the importance of knowing the diagnostic criteria of VP.

Results. Patient of male gender, 58 years old complained of attacks of intense vertigo lasting 15-20 seconds with a feeling of rotating objects counterclockwise. During an attack patient noted the crackling noise in the right ear, a feeling of instability, profuse sweat, nausea. Within next 10-15 minutes after the end of vertigo attack patient had a feeling of lightness in the head, palpitation, sensation of heat in the body.

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B) Duration less than 1 minute.

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1 month later at the visit, the patient reported no attacks during the treatment period. Hospital anxiety and depression scale: anxiety subscale score – 10 points, depression subscale score – 8 points. Patient recommended to use carbamazepine 100 mg twice a day for the next 3 months.

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Conclusions. Neurologists need to know and actively use in their practice diagnostic criteria of VP.

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A – Work concept and design, **B** – Data collection and analysis, **C** – Responsibility for statistical analysis, **D** – Writing the article, **E** – Critical review, **F** – Final approval of the article.

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