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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

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გამოიცემა თბილისის სახელმწიფო სამედიცინო უნივერსიტეტთან
თანამშრომლობითა და მისი პატრონაჟით

ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

ТБИЛИСИ - НЬЮ-ЙОРК

GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board and The International Academy of Sciences, Education, Industry and Arts (U.S.A.) since 1994. **GMN** carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

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GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией и Международной академией наук, образования, искусств и естествознания (IASEIA) США с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения.

Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

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1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

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При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

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2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

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Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

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In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

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რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

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5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

Содержание:

მნიშვნელოვანი უარყოფითი - საჭმლის მომნელებელი სისტემის დაავადებების მიზეზით სიკვდილიანობასთან (r=-0.577).

მეტაბოლური სტატუსის ასაკოვანი ცვლილებები შეიძლება იყოს მულტიფაქტორული ცვლილებების პრედიქტორები. ლაქტოზას პერსისტენციის პარამეტრები დადებით კავშირშია ბარძაყის მოტეხილობების სიხშირესთან და უარყოფით - კუჭ-ნაწლავის ტრაქტის დაავადებების მიზეზით სიკვდილიანობის მაჩვენებელთან. მულტიფაქტორული პათოლოგიების პროფილაქტიკის მნიშვნელოვან ფაქტორს წარმოადგენს დიეტის კორექცია ლაქტოზას მიმართ შეუთავსებლობის ფენოტიპის და გენოტიპის გათვალისწინებით.

CRANIAL NERVE HYPERFUNCTION SYNDROMES. MODERN APPROACHES TO DIAGNOSIS AND TREATMENT (REVIEW)

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Cranial nerves compression syndromes associated with neurovascular conflict and causing their hyperfunction are pathological conditions that significantly impair patient's quality of life. The most common and well-studied of such syndromes are trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia. In most cases, a clinician is faced with the choice of preferred method of treatment. In this article, we reviewed neurovascular conflicts of most cranial nerves with characteristic clinical syndromes and analyzed preferred treatment strategies, including surgical methods.

Material and methods. We conducted literature review using the Pubmed database, selecting articles on cranial nerves compression syndromes (in particular, trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia), published for 10 years (from 2010 to 2020). The search was performed in English, Russian, and Ukrainian using the following key words and terms: neurovascular conflict, neurovascular compression syndrome, cranial nerve hyperfunction syndrome, facial myokymia, trigeminal neuralgia, hemifacial spasm, glossopharyngeal neuralgia, vestibular nerve compression, vestibular paroxysmia, intermediate nerve neuralgia, vagus nerve compression, and microvascular decompression. The analysis covered all articles with information on the etiology, pathogenesis, clinical findings, diagnosis, differential diagnosis, neuroimaging, and pathomorphological assessment, as well as treatment strategies for such pathology, including surgical methods. After identifying all the articles that met the inclusion criteria and deleting duplicate data, 58 literature sources on cranial nerves compression syndromes associated with neurovascular conflict were selected.

Results and discussion. There is a perception that the cranial nerves compression syndromes associated with neurovascular conflict and leading to nerves hyperfunction are most characteristic of the trigeminal nerve — with the development of typical clinical manifestations in the form of Tic douloureux or idiopathic trigeminal neuralgia. However, this type of lesion may also involve other cranial nerves at the level of the root entry or exit from the brain stem — with the development of a variety of functional disorders and clinical conditions.

The relevance of the problem lies in the fact that due to relatively low prevalence of such syndromes, clinicians — primarily neurologists — often make mistakes in diagnosis and differential diagnosis of this pathology, which causes delay in establishing the correct diagnosis, inadequate therapy, and late neurosurgeon (able, in particular case, to radically eliminate the cause of the disease) involvement in the treatment.

The neurovascular conflict was first described in 1932 by Dandy, an American neurosurgeon who noted that trigeminal nerve compression in the posterior cranial fossa may cause facial trigeminal pain — tic douloureux [11]. A little later, Gardner and Sava described a facial nerve neurovascular conflict in more than half of the patients operated for hemifacial spasm — the motor equivalent of trigeminal neuralgia. However, it was only with the use of the Jannetta`s operating microscope that the frequency of occurrence and prevalence of neurovascular conflict and the efficiency of neurosurgical treatment were truly evaluated. Based on these studies, Jannetta developed and implemented a special surgical technique — microvascular decompression — which allowed the blood vessel and the affected nerve to be separated without detriment to the central nervous system [26]. Until now, this technique is widely used in neurosurgical hospitals worldwide to eliminate and/or alleviate specific syndromes caused by hyperfunction of cranial nerves in neurovascular conflict.

According to the literature, compression neurovascular syndromes are described for most cranial nerves: optic, oculomotor nerve groups: oculomotor, pathetic and abducent nerves; trigeminal, facial, vestibulocochlear, glossopharyngeal, vagus and accessory nerves, respectively. Such pathologies have different frequency and prevalence, as well as specific clinical picture [24].

Neurovascular conflict of optic and oculomotor nerve groups can be manifested by visual deterioration, optic atrophy, ptosis, or facial myokymia associated with superior oblique eye muscle lesion. Trigeminal-vascular conflict, by a classic trigeminal neuralgia. Facial nerve compression, by hemifacial spasm with secondary facial nerve neuropathy. Vestibulocochlear compression is associated with a variety of dementia syndromes, tinnitus, as well as vestibular paroxysmia, characterized by spontaneous vertigo attacks. Vascular compression of the bulbar group nerves — glossopharyngeal and vagus — is manifested by paroxysms of glossopharyngeal neuralgia, otalgia, and pharmacoresistant arterial hypertension, particularly in young people. Accessory nerve compression due to neurovascular conflict is manifested by dystonic syndromes in the form of spasmodic torticollis.

In most cases, there is a lesion of one cranial nerve, but multifocal compression of several nerve trunks with the development of polymorphic neurological symptoms is not uncommon. The literature describes cases of simultaneous lesions of cranial nerves 5 and 7, causing tic convulsif (trigeminal neuralgia with hemifacial spasm), combined lesions of nerves 7 and 8, and 9 and 10, respectively [2,6,33]. In such cases, sinuous vertebral artery is often the cause of compression.

Walter E. Dandy wrote, "The sensory root is frequently indented, lifted up, or bent at an angle to the artery... This I believe is the cause of tic douloureux" [46]. Since Walter Dandy first described the vascular loop/trigeminal nerve root conflict in trigeminal neuralgia patients, the theory of neurovascular compression has been postulated and widely accepted. It is believed that close contact between a blood vessel — a vein, but more often, an artery — and a nerve trunk that exists for a long period of time may cause local demyelination of the nerve root and induce the development of a phenomenon called ephaptic transmission of nerve impulses [27]. Heart-generated blood vessel pulsation during the cardiac cycle propagates a blood pressure wave up to the capillary bed. Venous blood flow also has the wave nature and leads to cyclical changes in intrathoracic pressure, thereby regulating venous return. Inside the skull, the main arteries and cranial nerve veins and trunks mostly pass in the posterior cranial fossa, which has a complex anatomy and includes a lot of structures: skull bones with holes therein, meninges and cerebrospinal fluid, brain stem, cerebellum, blood vessels and nerves. In fact, each of the three neurovascular complexes of the posterior cranial fossa includes one of the three cerebellar arteries, one of the three parts of the brain stem, one of the three cerebellar stalks, one of the three surfaces of the cerebellum, one of the three slots between the brain stem and the cerebellum, and one of the three groups of cranial nerves exiting the brain stem from top to bottom. Often, both vertebral and basilar arteries act as a compressing agent [22].

Venous neurovascular conflicts may also cause cranial nerve hyperfunction. There could be purely venous compressions or combined arteriovenous compressions. According to Dumot et al. [14], the incidence of trigeminal neuralgia due to venous neurovascular conflict varies from 6.1% to 68% (avg., 25.3%). Another study of 326 patients by Dumot and Sindou [15] found that 124 (38%) people had a venous neurovascular conflict: 29 (8.9%) had isolated conflict and 95 (29.1%) had conflict involving an artery. Clinically, the diagnosis of venous compression is difficult, because in many patients it is manifested by atypical trigeminal neuralgia and background persistent facial pain with episodic pain paroxysms.

The pathophysiological aspects of neurovascular compression lie in the fact that ephaptic transmission probably leads to abnormal electrical neuronal discharges propagating to the muscle fiber or sensory nerve endings of the corresponding cranial nerve. In the future, this may cause pathological exciting signals in the brain stem nuclei and constant electrical impulse with the formation of a "vicious circle" in the neural transmission. Most accurately, this pathophysiological cascade is reflected in the theory of Rasminsky, who summarized the above events as follows: mechanical or ectopic excitation; impulse reflection: orthodromic and/or antidromic conduction; ephaptic excitation or "cross-linking" between axons after a nerve impulse, which explains the phenomenon of synkinesis (e.g. in hemifacial spasm [8]).

The most pronounced pathophysiological changes are found in the cranial nerve roots in their direct brain stem entry/exit area, in the so-called Obersteiner-Redlich zone (a root entry zone, REZ). This is a 50 μm long transitional section, where depletion and/or complete absence of the myelin sheath is noted, where there is a transition of the "central" myelin synthesized by the central nervous system oligodendrocytes into the Schwann cells' "peripheral myelin". The "central" myelin in this part of the nerve is thinner and more susceptible to traumatic injury by any agent [19]. It is also worth noting that sensitive fibers of any nerve have a more extended "central" myelination section than motor fibers [34]. The relationship between the extent and volume of myelination of the central fragment of the cranial nerve roots has been well studied by Guclu et al. According to them, the largest length of this section is in the facial nerve, less in the trigeminal nerve, and the shortest fragment is in the glossopharyngeal and vagus nerves, which exit the brain stem in the form of several roots, unlike the facial nerve, which is represented by a single stem [20].

It should be noted that a number of authors note differences in the terms: root entry/exit zone (REZ) and transition zone (TZ) [48].In some publications, REZ is used as a synonym for TZ, while in other sources REZ refers to the nerve region that includes TZ, the central part of the myelinated root, and the adjacent surface of the brain stem. But all the same, the "transition zone", the TZ, is a more relevant and vulnerable anatomical structure, which is not always in the same position as the REZ. E.g., in the vestibulocochlear nerve, the TZ is clearly more distal than the REZ. Perfect anatomical knowledge of the TZ position and morphology is fundamental to the interpretation of neuroimaging results in cases of suspected neurovascular conflict [37].

In addition, the transition zone is also an area where the endoneurial microvascular network approaches the anastomosis with extraneural vascular plexus, which makes it poorly vascularized, creating a greater susceptibility to damage [8].

A number of authors describe the structural and morphological nerve roots changes during neurovascular conflict (exemplified by the trigeminal nerve) in the form of structure deformation, course deviation, groove formation on nerve fiber surface, nerve atrophy, axon reduction and demyelination, disrupted structure of the myelin itself, fibrosis, and excess of collagen fibers. [12]

In most cases, neurovascular conflict occurs due to the peculiarities of anatomical blood vessel course, abnormal looping, ectasias of posterior cranial fossa vascular structures that make up the vertebrobasilar system. Often, there is a compression not only of the cranial nerves, but also of the brain stem itself, as, for example, in case of vertebrobasilar dolichoectasia [58].

Small size of the posterior cranial fossa is probably the predisposing factor of neurovascular conflicts. According to some data, neurovascular conflict patients more often than the general population have a combination of small posterior cranial fossa and the Arnold-Chiari malformation; they also have earlier manifestation of symptoms in the nerve of interest. Moreover, after surgical treatment of the Arnold-Chiari malformation, complete regression of clinical manifestations of neurovascular conflict is often observed.

In neurovascular conflicts of any cranial nerve, remissions are possible, which is probably due to spontaneous root remyelination. Table 1 shows the main syndromes associated with cranial nerves compression.

Table 1. The main syndromes associated with cranial nerves compression

Cranial nerve	Clinical syndrome	Diagnosis criteria	Compressing vessel
III	Oculomotor nerve neuropathy [17]	Sedimentation symptoms in the form of nerve palsy, exotropia, ptosis, mydriasis, and accommodation disorders are more common	PCA SCA
IV	Superior oblique eye muscle myokymia [49]	Diplopia, oscillopsia	SCA
V	Idiopathic trigeminal neuralgia [4] Persistent idiopathic facial pain [40]	Unilateral facial pain in the dermatomes corresponding to the zones of innervation of trigeminal nerve branches 1, 2, or, more common, 3 in the form of lumbago, electric shocks lasting from several seconds to several minutes, a series of repeated pain attacks for several hours and even days, the presence of the so-called trigger areas, alternation of pain and refractory periods	SCA (75% – 88%) AICA (9.6% – 25%) Venous structures (8% – 10%) Multiple compression, 38%
VII	Hemifacial spasm [6,7,16] Intermediate nerve neuralgia [43]	Unintentional, strictly unilateral tonic and/or clonic contractions of the facial muscles, usually including platysma, which are not controlled by the mind and persist when asleep Short-lasting attacks of lancinating intense shooting ear pain, external auditory meatus, may be accompanied by lacrimation and a change in taste/unpleasant taste sensations.	AICA (43%) PICA (31%) VA (23%) Venous structures (3%) Multiple compression, 38%
VII	Vestibular paroxysmia [43,55]	At least 10 vertigo attacks (attack duration of less than 1 minute, dizziness occurs spontaneously or when the head is turned, the attacks are stereotypic; anticonvulsants are effective, other reasons are excluded	AICA (75%) Veins, 10%
IX	Glossopharyngeal neuralgia [3,4]	Paroxysms of unilateral acute pain in the submandibular region with radiation to the neck, tragus, auditory meatus, duration 1–3 seconds, provoked by yawning, swallowing, talking, attack onset from the tongue root with spread to the palatine velum, tonsils, pharynx; possible vegetative symptoms in the form of bradycardia and hypotension up to syncope	AICA (68%) VA (2%) Venous structures (6%) Multiple compression (23%)
X	Vagus nerve neuropathy [2,51] Atypical otalgia [30] Superior laryngeal nerve neuralgia [56] Hemi-laryngopharyngeal spasm (HELPS) [10,25]	Syncopes, bradyarrhythmias, atypical otalgia, dyspeptic symptoms in the form of nausea, vomiting, or diarrhea Intense shooting ear pain. Pharynx and larynx shooting pain Intentional pharynx muscles contractions by hemitype	Posterior inferior cerebellar (90% of all cases), vertebral or basilar arteries, or veins
XI	Spasmodic torticollis [1]	Unintentional tonic or periodic neck muscles spasms, manifested by abnormal head position, as well as intentional movements of cervical muscles.	VA

Currently, success in neurovascular conflicts diagnosis is largely due to application of high-power magnetic resonance scanners able to verify and measure the length of the transition zone, where the nerve root and the vessel are in contact. According to the literature, standard diagnosis involves a combination of three-dimensional T2-weighted high-resolution imaging with three-dimensional span angiography and three-dimensional gadolinium-enhanced T1-weighted imaging.

The combination of these techniques makes it possible to successfully verify the conflict and identify treatment tactics with an emphasis on surgical methods and, to some extent, predict the treatment response. The following methods are used: CISS imaging modes, FIESTA, balanced steady-state free precession MRI, controlled equilibrium and radio frequency discharge pulse, improved optimized contrast sampling using various rotation angle changes (SPACE sequence; Siemens, Germany), which provide

accurate visualization of the cisternal part of the affected cranial nerve. Multiplanar oblique reconstructions and fusion of three-dimensional T2-weighted sequences with corresponding TOF images or three-dimensional gadolinium-enhanced T1-weighted images may be useful, especially for preoperative studies [57].

In recent years, diffusion-tensor imaging with tractography has been increasingly used — mainly for verification of demyelination, especially in trigeminal neuralgia [47].

In addition, high-resolution techniques are useful for differential diagnosis and identifying other causes of neurovascular conflict: multiple sclerosis, ischemia, tumors, vascular pathology, etc.

In case of contraindications to MRI, it is possible to use helical computed tomography (HCT) with angiography. The study is conducted with intravenous contrast and allows evaluating the topographic features of the nerve root location, although does not allow visualizing minimal structural changes in the nerves themselves and in their roots exit zone. HCT is also recommended in suspected subarachnoid hemorrhage of aneurysmal origin.

In some cases, when the neurovascular conflict is combined with the neuropathy in the nerve of interest for 4+ months and the cause could not be verified even by an MRI scan, it is advisable to perform a positron emission brain tomography to clarify the pathological process nature, including differential diagnosis with lymphoproliferative diseases.

In cases where neuroimaging methods are not able to confirm or disprove the presence of vascular compression, it is possible to use invasive techniques, including endoscopic neurosurgery, which allows visualizing the pathology in real time and determine real possibilities of therapeutic measures.

Treatment strategies in neurovascular conflicts are diverse and include both therapeutic and surgical methods. According to international recommendations, for pain paroxysm relief in trigeminal and glossopharyngeal neuralgia, intravenous fosphenytoin or lidocaine infusions can be used. For long-term treatment, carbamazepine or oxcarbazepine is recommended as the first choice. Lamotrigine, gabapentin, type A botulinum toxin, pregabalin, baclofen, and phenytoin can be used alone or as an additional therapy. Adding ropivacaine injection to any of the above drugs may also have an effect [32]. Combination with antidepressants to control pain is possible.

In vestibular paroxysmia, carbamazepine in a daily dose of 200 to 800 mg is also the drug of choice; in case of poor tolerance to the drug, sodium channel blockers such as valproic acid and phenytoin can be considered as alternatives.

When conducting drug treatment, it is important to monitor pain syndrome intensity as per VAS. The use of specialized scales to evaluate drug therapy efficiency, such as BNI (Barrow Neurological Institute scoring system) and MMS Scale (Modified Marseille Scale) [31], is reasonable.

In the treatment of facial hemispasm, spasmodic torticollis, and other neurovascular compression syndromes accompanied by hyperkineses in addition to anticonvulsants administration, botulinum therapy by botulinum toxin type A injection into the affected muscles is currently preferred. According to the literature, the efficiency of this procedure is 90–100% [41]. In addition, botulinum toxin A has a good tolerance profile, minimum adverse effects and small number of contraindications, which allows to use it in elderly people with comorbidities. Transient facial asymmetry and diplopia, which usually regress within a month, are more often described as adverse effects.

Regarding the efficiency of botulinum toxin in trigeminal neuralgia treatment, large systematic reviews indicate that the use of botulinum toxin type A in dosage from 25 to 100 IU, in combination with other systemic drugs, reduced pain syndrome frequency and intensity to 12 weeks [44]. Research of Li, S et al. indicates significant reduction in pain intensity in 25% of patients within 14 months after botulinum toxin A injection [35]. It is possible that botulinum toxin type A may have an effect as an adjunctive therapy in some individual cases of trigeminal neuralgia, but given very low quality of evidence, a recommendation to use it in respective cases is unreliable.

Nevertheless, practical experience shows that drug treatment is effective, on average, in 60% to 80% of patients — therefore, surgical treatment should be considered in the absence or insufficient clinical effect as early as possible. The results of Heinskou T et al. series of 156 trigeminal neuralgia patients indicate that the majority of patients (88%) preferred surgery to pharmacological treatment [24].

Microvascular decompression (MVD) is believed the operation of choice in posterior cranial fossa neurovascular conflicts. Thus, according to the literature, the MVD efficiency in trigeminal neuralgia (the absence of pain and the need to continue conservative treatment) is up to 90% in the 1st postoperative year and up to 70–73.4% in 10–15 years after the surgery [9]. In glossopharyngeal nerve neuralgia, the MVD efficiency is up to 90%, which means that surgical treatment can be considered the gold standard of the therapy [54]. The MVD efficiency in facial hemispasm is also high and, according to some reports, reaches 85–90% in the short term and 70% in the long term [50]. As for vestibular paroxysmia, there is insufficient data on the MVD as a treatment method. There are minor reports in the literature on partial success of surgical treatment using this technique [39]. Nevertheless, it is recommended to consider it as a treatment option for patients with neurovascular conflict of the 8th pair of cranial nerves and a clinical picture of vestibular paroxysmia in patients who do not respond to or poorly tolerate pharmacological treatment, given that there is a high risk of brain stem infarction due to intra- or postoperative vasospasm.

In most cases, the posterior cranial fossa MVD is carried out by retrosigmoid approach with mandatory intraoperative facial nerve function monitoring to minimize complications. According to various sources, surgical complications include transient (20%) and persistent (3–8%) hearing impairment, vestibular disorders, sensitive facial damages (5–30%), transient (5–6%) and persistent (2%) facial palsy, and cerebellar stroke (1–2% of patients) [53]. According to several authors, relapse risk factors include female sex, 8+ years disease duration, a vein as a source of conflict, and the absence of instant postoperative symptoms regression. Intraoperative detection of focal arachnoiditis reduces the efficiency of microvascular decompression.

Percutaneous puncture techniques (used primarily for the treatment of trigeminal neuralgia) involve bringing the conductor needle to the oval foramen and entering the Meckel's cave with targeted nociceptive trigeminal fibers destruction. The most common techniques are glycerol rhizotomy, microballoon compression, and radiofrequency trigeminal rhizotomy. According to the literature, the efficiency of the latter reaches 76% after one session and 24% if the procedure is repeated 2 to 6 times [29]. Among the complications, the authors describe masticatory muscle disorder, painful facial dysesthesia, decreased corneal reflex, abducent nerve lesion, etc. Despite the lower efficiency of these techniques compared to the microvascular decompression, they can be repeated at varying intervals for pain control, which is especially important in elderly patients subject to high surgical risks.

Radiosurgical treatment. Gamma knife is a method of stereotactic radiosurgery, where Cobalt-60 (70-90 Gy) is the source of ionizing radiation. The method is widely used in trigeminal and glossopharyngeal nerves neurovascular conflicts treatment [52]. The retrogasserian trigeminal segment is its target in trigeminal neuralgia. Akdag et al. [21] presented the outcomes of 250 trigeminal neuralgia patients 5 years after radiosurgical treatment. 70% of patients had no pain, in 20% the pain decreased by more than 1/2, in 5% it was less than 1/2, in 5% it became more intense. The complications included scleral dryness (22%), memory impairment (20%), hearing loss (16%), hypogeusia (16%), dysphagia (13%), and masticatory muscle weakness (11%). Nevertheless, data of larger reviews indicate lower efficiency of radiosurgery compared to the MVD in terms of short- and long-term analgesic effects [42].

According to Kano H et al., stereotactic radiosurgery can be an efficient pharmacoresistant glossopharyngeal neuralgia treatment with a long-term pain syndrome reduction in 55% of patients after 1 or 2 procedures. No patient in their study had vocal cords function changes or swallowing disorders after the procedure [28].

Partial rhizotomy, a transection of 1/3–1/4 of sensory fibers of the trigeminal nerve is more often used if the trigeminovascular conflict is either not verified as the cause of neuralgia, or it is impossible to adequately separate the vessel and the nerve in the microvascular decompression. According to J. Young and R. Wilkins, 6 years after surgery 40 (48%) patients had excellent outcome, 18 (22%) had good outcome, 25 (30%) had mediocre or unsatisfactory outcome, but the number of patients who had pain relapse increased by 2.6% every follow-up year [36]. According to Ruiquan Liu, Zhu Deng et al. the use of a combination of microvascular decompression and partial rhizotomy can also be considered as an efficient surgical alternative [38].

Thus, we can say that compression syndromes in cranial nerves associated with neurovascular conflict are a complex interdisciplinary problem, often poorly understood and undetectable by neurologists. The literature describes cases of neurovascular conflicts involving all cranial nerves except for the olfactory nerve. A blood vessel (an artery, less often a vein), typically in the area where a nerve root exits the brain stem acts as a compressing agent. Clinical findings are diverse and associated with nerve hyperfunction. The most common manifestations are trigeminal neuralgia, glossopharyngeal neuralgia, and hemifacial spasm. Currently, implementation of standard cranial nerve neuroimaging protocol using high-field high-power MR scanners, MR angiography, and DVI tractography into the diagnosis allows to timely detect neurovascular conflict and determine treatment tactics. Neurosurgical intervention should be considered in case of failure or poor tolerance of conservative treatment. In most cases, the operation of choice is microvascular decompression, the essence of which is to separate the compressing vessel from the nerve and insert a sponge between the nerve and the vessel.

Conclusion. Neurosurgical intervention should be considered in case of failure or poor tolerance of conservative treatment. In most cases, the operation of choice is microvascular decompression, the essence of which is to separate the compressing vessel from the nerve and insert a sponge between the nerve and the vessel.

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SUMMARY

CRANIAL NERVE HYPERFUNCTION SYNDROMES. MODERN APPROACHES TO DIAGNOSIS AND TREATMENT (REVIEW)

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In this article, we reviewed neurovascular conflicts of most cranial nerves with characteristic clinical syndromes and analyzed preferred treatment strategies, including surgical methods.

We conducted literature review using the Pubmed database, selecting articles on cranial nerves compression syndromes, published for 10 years (from 2010 to 2020). The analysis covered all articles with information on the etiology, pathogenesis, clinical findings, diagnosis, differential diagnosis, neuroimaging, and pathomorphological assessment, as well as treatment strategies for such pathology, including surgical methods. After identifying all the articles that met the inclusion criteria and deleting duplicate data, 58 literature sources on cranial nerves compression syndromes associated with neurovascular conflict were selected.

Cranial nerves compression syndromes are a complex interdisciplinary problem, often poorly understood and undetectable by neurologists. Literature sources describe cases of neurovascular conflicts with all cranial nerves except for the olfactory one. A blood vessel (an artery, less often a vein), typically in the area where a nerve root exits the brain stem acts as a compressing agent. Clinical findings are diverse and associated with nerve hyperfunction. The most common manifestations are trigeminal neuralgia, glossopharyngeal neuralgia, and hemifacial spasm. Currently, implementation of standard cranial nerve neuroimaging protocol using high-field high-power MR scanners, MR angiography, and DVI tractography into the diagnosis allows to timely detect neurovascular conflict and determine treatment tactics.

Neurosurgical intervention should be considered in case of failure or poor tolerance of conservative treatment. In most cases, the operation of choice is microvascular decompression, the essence of which is to separate the compressing vessel from the nerve and insert a sponge between the nerve and the vessel.

Keywords: neurovascular conflict, neurovascular compression syndrome, cranial nerve hyperfunction syndrome, facial myokymia, trigeminal neuralgia, hemifacial spasm, glossopharyngeal neuralgia, vestibular nerve compression, vestibular paroxysmia, intermediate nerve neuralgia, vagus nerve compression, microvascular decompression, rhizotomy, gamma surgery.

РЕЗЮМЕ

СИНДРОМЫ ГИПЕРФУНКЦИИ ЧЕРЕПНЫХ НЕРВОВ. СОВРЕМЕННЫЕ ПОДХОДЫ К ДИАГНОСТИКЕ И ЛЕЧЕНИЮ (ОБЗОР)

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В обзоре представлен анализ ретроспективных и современных научных источников информаии по вопросам нейро-васкулярных конфликтов большинства черепно-мозговых нервов с характерными клиническими синдромами; рассмотрены наиболее предпочтительные стратегии лечения, в том числе хирургические методы с глубиной поиска 10 лет (2010-2020 гг.). Проанализировано 58 выявленных релевантных источников об этиологии, патогенезе, клинике, диагностике, дифференциальной диагностике, патоморфологической оценке и стратегиях лечения данной патологии.

На основании анализа и синтеза выявленной литературы определено современное состояние изученной патологии, в частности компрессионные синдромы черепно-мозговых

нервов являются сложной междисциплинарной проблемой, в большинстве случаев плохо понимаемой и недодиагностируемой неврологами. Клиника многообразна и связана с гиперфункцией нервов. Наиболее частыми проявлениями являются тригеминальная невралгия, гемифациальный спазм, языкоглоточная невралгия. В настоящее время внедрение в диагностику стандартного протокола нейровизуализации черепных нервов при помощи высокопольных МР-сканеров высокой мощности, МР-ангиографии и DVI-трактографии позволяет своевременно диагностировать нейроваскулярный конфликт и определить тактику лечения. Операцией выбора в большинстве случаев считается микроваскулярная декомпрессия, суть которой заключается в отделении компримирующего сосуда от нерва и установке прокладки между нервом и сосудом.

რეზიუმე

თავის ტვინის ნერვების ჰიპერფუნქციის სინდრომები. თანამედროვე მიდგომები დიაგნოსტიკასა და მკურნალობაში (მიმოხილვა)

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[1]დნეპროპეტროვსკის სამედიცინო აკადემია, ნერვული სნეულებების და ნეიროქირურგიის კათედრა; [2]დნეპროპეტროვსკის ი.მეჩნიკოვის სახელობის რეგიონული ჰოსპიტალი, უკრაინა

მიმოხილვაში წარმოდგენილია რეტროსპექტული და თანამედროვე სამეცნიერო წყაროების ანალიზი თავის ტვინის ნერვების ნეიროვასკულური კონფლიქტების საკითხებზე, დამახასიათებელი კლინიკური სინდრომებით; განხილულია მკურნალობის უპირატესი სტრატეგიები, მათ შორის – ქირურგიული მეთოდები, ძიების სიღრმით - 10 წელი (2010-2020 წწ.). გაანალიზებულია გამოვლენილი 58 რელევანტური წყარო ამ პათოლოგიის ეტიოლოგიის, პათოგენეზის, კლინიკის, დიაგნოსტიკის, დიფერენციული დიაგნოსტიკის, პათომორფოლოგიური შეფასების და მკურნალობის სტრატეგიების შესახებ.

გამოვლენილი ლიტერატურის ანალიზის და სინთეზის საფუძველზე განსაზღვრულია შესწავლილი პათოლოგიის თანამედროვე მდგომარეობა, კერძოდ, ის, რომ თავის ტვინის ნერვების კომპრესიული სინდრომები წარმოადგენს დისციპლინათშორის პრობლემას, ხშირად – ცუდად გაგებულს და ბოლომდე არადიაგნოსტირებულს ნევროლოგების მიერ. კლინიკა მრავალფეროვანია და დაკავშირებულია ნერვების ჰიპერფუნქციასთან. ყველაზე ხშირ გამოვლინებას წარმოადგენს ტრიგემინური ნევრალგია, ჰემიფაციალური სპაზმი, ენა-ხახის ნევრალგია. დღეს დიაგნოსტიკაში თავის ტვინის ნერვების ნეიროვიზუალიზაციის სტანდარტული პროტოკოლის დანერგვა მაღალი სიმძლავრის მაგნიტურ-რეზონანსული სკანერების, მაგნიტურ-რეზონანსული ანგიოგრაფიის და DVI-ტრაქტოგრაფიის საშუალებით იძლევა ნეიროვასკულური კონფლიქტის დროული დიაგნოსტიკის და მკურნალობის ტაქტიკის განსაზღვრის საშუალებას. შემთხვევათა უმეტესობაში არჩევანის ოპერაციად ითვლება მიკროვასკულური დეკომპრესია, რომლის არსი მდგომარეობს კომპრიმირებული სისხლძარღვის მოცილებაში ნერვიდან და საფენის განთავსებაში ნერვსა და სისხლძარღვს შორის.

TRAUMATIC BRAIN INJURIES IN CHILDREN IN PRACTICE OF PEDIATRIC HOSPITAL IN GEORGIA

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Traumatic brain injury (TBI) is a leading cause of death and disability worldwide and one of the major problems of Public Health [1-6]. Mortality from child traumatic injuries is 3-4 times higher in middle-income countries than in high-income countries [7,8]. Unfortunately, TBI is under-recognized and under-studied, particularly in many low- and middle- income countries [9-11]. Study of TBI reliable and high-quality data represents the basis for elaborating effective strategies for injury prevention [12].

Since 2014 the traumatic brain injury national reporting format for the hospitalized patients is in line with the International E-health standards (ICD-10 codes for each case, including special codes for external causes of the injury, NCSP codes for health intervention etc.). Since 2019 the country started introducing (piloting) Electronic Health Record (EHR) system, which potentially can collect all TBI related necessary information.

In 2018, the total number of hospitalized 0-17 age patients with TBI diagnosis (ICD-10 - S06) was 2314 in all medical facilities of Georgia. Patients were hospitalized in different hospitals in Tbilisi and regions, and the biggest number (12,8%) was hospitalized in Pediatric hospital. In 33 medical facilities was hospitalized up to 5 patients and in 23 medical facilities 5-10 TBI patients during the year. The aim of the research is to study epidemiological features of pediatric TBI based on the example of data from the biggest pediatric hospital in Georgia.

Material and methods. In this study we used descriptive statistics of NCDC official data.

Results and discussion. The total number of pediatric TBI patients during one year (2018) was 296, among them majority were male patients (61,1%), minority of patients were in 15-17 age group (12,5%) while in other three age group (10-14; 5-9 and 0-4) patients were distributed approximately equally (31,8%, 28,7% and 27,0%).