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THERAPEUTICAL APPROACH IN THE TREATMENT OF TRIGEMINAL NEURALGIA

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Abstract: The variety of treatment options for trigeminal neuralgia (TN) reported in the literature reflects clinicians' dissatisfaction with the results of treatment of this severe disease. The search for new methods of treatment is going in two directions: by improving drug therapy through the introduction of new drugs and their combinations and through the development of new surgical techniques affecting the trigeminal nerve (TN) system. Each of the therapeutic methods of treatment is mainly aimed at only one of the links in the pathogenetic chain of trigeminal neuralgia development: either at increasing the threshold of excitability of sensitized neurons of the brain stem and cortex, or at switching off the trigger zone. The main link, where the pathological focus of demyelination (the site of neurovascular conflict) is formed, remains outside the sphere of influence of existing therapeutic methods. The need to improve therapeutic methods is also due to the fact that, in addition to primary TN patients, patients who have no conflict detected during microvascular decompression (MVD), in whom pain relapse occurred after successful MVD, in those cases when root compression by a vessel is detected, but for technical reasons it is impossible to perform safe repositioning of the vessel, when there are medical contraindications to general anesthesia, etc., need treatment.

Key words: Trigeminal neuralgia, treatment, paroxysm, corticosteroid, microvascular decompression, neurovascular conflict, brainstem, cortex.

Research objective: Adequate approach to the conservative treatment of trigeminal neuralgia.

Material and methods. The group of patients receiving conservative therapy included 78 patients, of whom 64 patients did not tolerate surgical interventions, and 14 patients underwent microvascular decompression of the trigeminal nerve root at various times before enrollment for conservative treatment, and they relapsed at various times after surgery. These patients refrained from reoperation and elected conservative therapy. Men were 28%, women - 72%, with right-sided (65%) and left-sided (35%) localization of pain. Duration of exacerbation before admission to the clinic for conservative treatment was different: from 1 to 3 months - in 56 patients, from 4 to 6 months - in 13 patients, from 7 months to 1 year - in 8 patients, and in 1 patient the duration of exacerbation was 2 years. Pain intensity according to VAS scale (VAS) was mainly severe (within 7-9 points) in 55 patients, very severe (10 points) - in 19 patients and moderate (from 4 to 6 points) - in 4 patients. The duration of pain

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paroxysms also varied widely: from 1 to 10 seconds (53 patients), up to 1 minute (18 patients), up to 3 minutes (4 patients) and a series of attacks over 3 minutes (3 patients). The frequency of attacks per day also varied: up to 30 attacks were observed in 54 patients, 31 to 50 in 14 patients, 51 to 100 in 9 patients, and over 100 in one patient. Consequently, the main factors disturbing the patient's daily activity are the intensity and number of pain paroxysms. Up to 2024, 64 patients were treated with the treatment method developed by us, the effect of drugs in which was directed at two links of pathogenesis. On admission to the clinic, the patient continued to take carbamazepine in the same dose, despite the fact that monotherapy with this drug before admission to the clinic did not bring relief. Carbamazepine affected the central mechanisms of pain by increasing the threshold of excitability of sensitized neurons of the brainstem and cortex, but this was not enough to achieve remission. The focus of demyelination at the site of neurovascular conflict was treated with drugs that promote the process of remyelination: corticosteroids, B vitamins (cyankoblamin, thiamine, pyridoxine), -lipoic acid (thioktacid, berliton). Berliton (or thioktacid) was administered by intravenous drip at 600 mg per day. The course of treatment was 10 days. Milgamma (complex of vitamins B1, B6) was administered intramuscularly every other day, alternating with blockades. If possible, the drugs were administered closer to the focus of demyelination. Thus, blockades were performed in the area of the round and oval foramen. The composition of blockades: corticosteroids (Kenolog 40, later DepoMedrol) in combination with vitamin B12 and lidocaine. Since 2024, 14 patients were added to the current treatment regimen with applications to the trigger zone area in order to disable them. The composition of applications: 98% dimethyl sulfoxide solution - 20 parts, 2% lidocaine solution - 40 parts, 2% alcohol solution of anesthesin - 40 parts. Applications of length 30-45 minutes were performed daily every 3-4 hours 4-5 times a day. Switching off trigger zones with the help of applications is a therapeutic effect on the third link of the pathogenetic chain of TN. The effectiveness of treatment was evaluated by pain reduction (according to the VAS scale). In patients whose pain attacks went away and they stopped taking finlepsin and its analogs (number of points 0), the result of treatment was considered as complete remission. In patients with a decrease in the intensity of pain relative to the baseline level who had no restrictions in daily activities while taking maintenance doses of carbamazepine, the result of treatment was considered as medication remission. If, as a result of treatment, there was no decrease in pain intensity, it indicated the absence of effect. Statistical processing of the material was performed using Statistics 6.0 program.

Results and Discussion. The results of treatment of 64 patients with influence on two components of pathogenesis are as follows: 24 (37,5%) patients achieved complete remission, 35 (54,7%) patients achieved drug remission and 5 (7,8%) patients had no therapeutic effect and underwent microvascular decompression of the trigeminal nerve root. Of the group of 14 patients whose therapy was directed at the three components of the pathogenesis of classical TN, 9 (64.3%) had complete remission and 5 (37.5%) had medication remission. The results show the advantage of the therapeutic effect on all three components of the pathogenesis of classical TN. The number of patients who obtained complete remission doubles. A weak correlation dependence of treatment results on the age of patients who received conservative therapy was established. The older the patient, the more pronounced were the disorders of daily activity after the course of conservative therapy. In the elderly, in addition to demyelination caused by neurovascular conflict, age-related death of myelinated fibers plays a major role. A correlation between the duration of exacerbation and the results of treatment was revealed. The less prolonged was the

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exacerbation of the disease, the better results were obtained after conservative therapy. Of 33 patients in both groups who were discharged after conservative therapy in a state of complete remission, three returned with disease recurrence (1 after 7 months, 1 after 2 years, and 1 after 3 years). All of them opted for surgical treatment - MVD. Of the 25 patients who remained, complete remission was maintained for 3 years in 18 patients, and 7 patients due to the exacerbation of the disease started taking Finlepsin with periodic interruptions as the pain subsided. Of the 40 patients discharged in a state of medication remission, two returned with a relapse (1 after 7 months, 1 after 1 year). Both of them opted for surgical treatment this time - MVD. Of the remaining 38 patients during three years of follow-up, 9 discontinued finlepsin due to disappearance of seizures. Of the 29 patients in this group, information was obtained from 22. Exacerbations in this group in the fall-winter-spring period were observed annually, which required an increase in the dose of Finlepsin. Four out of 22 patients had to increase the dose of finlepsin to 1800-2200 mg per day without noticeable improvement. They were hospitalized again, again electing conservative therapy. Three of them were discharged in a state of medication remission with a 400 mg daily finlepsin dose reduction, and one patient was discharged in a state of complete remission. No information was obtained from 7 of the 40 patients discharged in medication remission. These were mostly patients over 75 years of age who had previously undergone conservative treatment.

The developed method of conservative therapy of TN, despite its pathogenetic orientation, is palliative. At the same time, the result of drug remission is a significant reduction in the dose of dibenzoazepine drugs (from 1000-3200 mg/day at the beginning of treatment to 400-600 mg/day and less at discharge from the clinic. Reducing the dose of dibenzoazepine drugs reduces the probability of side effects. The main criterion for selection of patients for surgical treatment is absence of effect from conservative therapy during 3-4 months, despite taking high doses of carbamazepine (up to 4000 and more mg per day). Expressed side effects of the drug, such as headache, dizziness, ataxia when walking, loss of appetite, nausea, vomiting, along with severe pain paroxysms, exhausting the patient, put him before the need for surgical treatment. Indication for surgical treatment is also neuralgic status, when the patient is deprived of food and fluid intake due to pain, which leads to weight loss and metabolic disorders.

Regardless of the duration of the disease, the indication for MVD is individual intolerance to dibenzoazepine drugs. In case of prolonged drug treatment and inability to achieve complete remission within 3-4 months, we suggest the patient to perform spiral computed tomography to confirm neurovascular conflict. In this case, microvascular decompression of the CTN is recommended. The decision on the choice of treatment method remains with the patient, taking into account the comorbid somatic pathology accompanying the underlying disease.

Conclusion. Treatment of 64 patients involved two components of the pathogenesis of the disease: truncal and cortical foci of pathologically increased stimulation and a focus of demyelination in the area of neurovascular conflict. In the treatment of 14 patients three components of the pathogenetic chain were involved and additional application of anesthetics in trigger zones was carried out. This method had an advantage in the treatment of classic TN. A doubling of the number of complete remissions was observed.

A weak negative correlation between the age of patients and the results of therapy was established. If complete remission could not be achieved within 3-4 months, patients underwent spiral computerized angiographic study. We recommend microvascular

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decompression of CTN in cases of confirmed neurovascular conflict. Destructive interventions are acceptable in cases of ineffectiveness of organ-preserving approaches in elderly patients.

References:

1. Balyazin, V.A. Ways to reduce complications and relapses after microvascular decompression in patients with trigeminal neuralgia / V.A. Balyazin, E.V. Balyazina // Voprosy neurosurgery. N.N. Burdenko. - 2003. - № 2. - C. 6-9.

2. Balyazin, V.A. Neuralgia of trigeminal nerve. Modern approaches to surgical treatment / V.A. Balyazin, E.V. Balyazina // Mater. conf. Pain syndromes in the head, face and mouth cavity. - Smolensk, 2010. - C. 166-170.

3. Konovalov, A.N. Microvascular decompression of the trigeminal nerve root in the treatment of trigeminal neuralgia / A.N. Konovalov, O.R. Orlova, U.B. Mahmudov [et al.] // Vertebronneurology. - 2008. - T.15, №1/2. - C. 81-82.

4. Peshkova, O.P. Effectiveness of complex therapy with reflexotherapy in trigeminal neuralgia / Peshkova O.P., O.I. Zagorulko, L.A. Gnezdilov // IX All-Russian Congress of Neurologists. - Yaroslavl, 2006. - C. 279.

5. Sokov, E.L. Application of intraosseous blockades in trigeminal neuralgia / E.L. Sokov [et al] // Mater. Conf. Pain syndromes in the head, face and mouth cavity. - Yaroslavl, 2010. - C. 170-172.

6. Allam, N. Injections of botulinum toxin type a produce pain alleviation in intractable trigeminal neuralgia / N. Allam, J.P. Brasil-Neto, G. Brown, C. Tomaz // Clin. J. Pain. – 2005. – Vol.21, №2. – P. 182-184.

7. Cunningham, V.L. Human health risk assessment of carbamazepine in surface waters of North America and Europe / V.L. Cunningham, C. Perino, V.J. D'Aco, A. Hartmann, R. Bechter // Regul. Toxicol. Pharmacol. – 2010. – Vol.56, №3. – P. 343-351.

8. Domingues, R.B. Treatment of trigeminal neuralgia with low doses of topiramate / R.B. Domingues, G.W. Kuster, C.C. Aquino //Arq. Neuropsiquiatr. – 2007. – Vol. 65. – P. 3792-37

9. Gerdner, E.D. Decrease in human neurous with age / E.D. Gerdner // Anatomical record. – 1940. – Vol. 77. – P. 529- 532.

10. Jorns, T.P. Pilot study to evaluate the efficacy and tolerability of levetiracetam (Keppra) in treatment of patients with trigeminal neuralgia / T.P. Jorns, A. Johnston, J.M. Zakrzewska // Eur. J. Neurol. – 2009. – Vol.16, №6. – P. 740-744. 11. Kanai, A. Intranasal lidocaine 8% spray for second-division trigeminal neuralgia / A. Kanai, A. Suzuki, M. Kobayashi, S. Hoka // Br. J. Anaesth. – 2006. – Vol.97, №4. – P. 559- 563.

ISSN: 2692-5206
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INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

12. Lemos, L. Gabapentin supplemented with ropivacain block of trigger points improves pain control and quality of life in trigeminal neuralgia patients when compared with gabapentin alone / L. Lemos, S. Flores, P. Oliveira, A. Almeida // Clin. J.B. Pain. – 2008. – Vol.24, №1. – P. 64-75.

13. Ngeow, W.C. Injection of botulinum toxin type A (BOTOX) into trigger zone of trigeminal neuralgia as a means to control pain / W.C. Ngeow, R. Nair // Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. – 2010. – Vol.109, №3. – P. 47- 50.

14. Pérez, C. Patient-reported outcomes in subjects with painful trigeminal neuralgia receiving pregabalin: evidence from medical practice in primary care settings / C. Pérez, A. Navarro, M.T. Saldaña, S. Martínez, J. Rejas // Cephalalgia. – 2009. – Vol.29, №7. – P. 781-790.

15. Piovesan, E.J. An open study of botulinum-A toxin treatment of trigeminal neuralgia / E.J. Piovesan, H.G. Teive, P.A. Kowacs [et al.] // Neurology. – 2005. – Vol.65, №8. – P.1306-1308.