Review article

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Method of treatment of trigeminal neuralgia in the elderly

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Abstract

The treatment of trigeminal neuralgia (HTN) in the elderly, highlighted in the literature, reflects the dissatisfaction of clinicians with the results of the treatment of this chronic disease. Scientific research on the treatment of trigeminal neuralgia in the elderly is carried out by improving drug therapy through the introduction of new drugs and their combinations and through the development of new surgical techniques that affect the trigeminal nerve system (TN). Each of the therapeutic methods is mainly aimed at a single link in the pathogenetic chain of the development of trigeminal neuralgia, either to increase the threshold of excitability of sensitized neurons of the brain stem and cortex or to turn off the trigger zone.

Keywords: Trigeminal neuralgia; Chondroprotectors; Oncological diseases.

Introduction

The main link where the pathological focus of demyelination is formed (in oncological diseases of the nervous system) remains outside the sphere of influence of the existing therapeutic techniques. The need to improve therapeutic methods is also due to the fact that—in addition to primary NTN patients—there are patients with comorbid osteoporosis of the skull bones (OCD) that requires treatment, patients in whom a relapse of pain occurs after successful MIA, cases where the expansion of the channel of the branches of the trigeminal nerve is required, but in which safe expansion cannot be achieved without causing nerve damage due to technical reasons, and cases with medical contraindications to general anesthesia.

Materials and methods

Forty-eight patients received conservative therapy; among these, 32 patients did not receive surgical intervention, and 16 patients underwent expansion of the trigeminal nerve canal at various times before admission for conservative treatment, and they relapsed at various times after surgery. These patients refrained from repeated surgeries and chose conservative therapy. Twenty-eight percent of the patients were men and 72% were women. The pain was right-sided in 65% of the patients and left-sided in 35%. The duration of exacerbation before admission to the clinic for conservative treatment was different: 1 to 3 months in 26 patients, 4 to 6 months in 3 patients, 7 months to 1 year in 4 patients, and 2 years in 1 patient. The intensity of pain on the VAS scale was strong (7–9 points) in 15 patients, very strong (10 points) in 9, and moderate (4–6 points) in 8. The duration of pain paroxysms also varied widely: from 1 to 10 s (23 patients), up to 1 min (3 patients), up to 3 min (6 patients), and a series of seizures over 3 min (5 patients). The frequency of seizures per day also varied: up to 30 seizures were observed in 14 patients, 31 to 50 were observed in 8, 51 to 100 were observed in 9, and over 100 were observed in 1 patient. Consequently, the main factors that disrupt a patient's daily activities are the intensity and number of pain paroxysms.

By 2021, 48 patients were treated with our treatment method, and the effects of the drugs were directed at two pathogenetic links. Upon admission, the patient continued to take carbamazepine at 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 brain stem and cortex, but this was not enough to achieve remission.

The focus of osteoporosis is affected by drugs that promote the process of osteoporosis: chondroprotectors (alflutop), B vitamins (cyancoblamine, thiamine, pyridoxine), and lipoic acid (thioctacid, berlithione). Berliton (or thioctacid) was administered intravenously by drip at a dose of 600 mg/day. The treatment course lasted for 10 days. Milgamma (a complex of vitamins B1 and B6) was administered intramuscularly every alternate day, alternating with blockade. Drug administration was brought as close as possible to the focus of demy-

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round and oval holes. Blockade therapy consisted of corticosteroids (Kenolog 40, later DepoMedrol) in combination with vitamin B12 and lidocaine.

Since 2022, sodium ibondronate has been added to the treatment regimen in 14 patients. The tablet was administered once per week in the morning on an empty stomach with 200 ml of water per week. It was administered a total of eight times.

The effectiveness of treatment was assessed based on pain reduction (on the VAS scale). In patients who had pain attacks who stopped taking finlepsin and its analogs (score 0), the result of treatment was regarded as complete remission. In patients with a decrease in pain intensity relative to the baseline level who had no restrictions in daily activity while taking maintenance doses of carbamazepine, the result of treatment was regarded as drug remission. If there was no reduction in the intensity of pain as a result of treatment, it was considered that there was no effect. Only 2 patients with trigeminal neurinoma had surgery in the neurosurgical department. All statistical analyses were performed using the Statistics 6.0 software program.

Results and Discussion

The results of the treatment in 48 patients with exposure to two pathogenetic links were as follows: complete remission, n=18 (37.5%); drug remission, n=26 (54.7%); and no therapeutic effect, n=4 (7.8%). Patients with no therapeutic effect underwent expansion of the trigeminal nerve canal. Of the 16 patients in whom therapy was aimed at three components of the pathogenesis of classical NTN, 10 (64.3%) achieved complete remission and 6 (37.5%) achieved drug remission. The results showed the advantage of therapeutic effects on all three components of the pathogenesis of classical NTN. At the same time, the number of patients who achieved complete remission doubled. The treatment results showed a weak correlation with age in patients receiving conservative therapy. The older the patient, the more pronounced the disruption of daily activity that remained after the course of conservative therapy. In the elderly, in addition to demyelination caused by neurovascular conflict, age-related death of myelinated fibers plays an important role [9]. The duration of the exacerbation was found to be correlated with the results of treatment. The less prolonged the exacerbation of the disease, the better the results obtained after conservative therapy.

Of the 29 patients in both groups who were discharged after conservative therapy in a state of complete remission, three returned with a relapse of the disease (7 months, n=1; 2 years, n=1; 3 years, n=1). All were treated according to the surgical method recommended by the Ministry of Internal Affairs. Of the 25 remaining patients, complete remission persisted for 3 years in 14 patients, while 5 patients began taking finlepsin (with periodic interruptions as pain subsided) due to an exacerbation of their disease.

Of the 28 patients discharged in a state of drug remission, two returned with a relapse (after 7 months, n=1; after 1 year, n=1). Both cases were surgically treated. Of the remaining 20 patients, 9 stopped taking finlepsin during follow-up due to the disappearance of seizures. Among the 29 patients in this group, information was obtained from 22. In this group, exacerbations that required an increase in the dose of finlepsin were observed annually in the autumn-winter-spring period. In 4 of 20 patients, the dose of finlepsin was increased to 1,800-2,200 mg per day without noticeable improvement. The patients were hospitalized repeatedly, but chose conservative therapy. Three patients were discharged in a state of drug remission with a reduction in the dose of finlepsin to 400 mg per day, and one patient was in a state of complete remission. No information was received from 7 of the 28 patients who were discharged in a state of medical remission. These were mainly patients who were >75 years of age, and who had previously undergone conservative treatment.

Our method of conservative therapy for NTN, despite its pathogenetic orientation, is palliative. At the same time, drug remission is associated with a significant reduction in the dose of dibenzoazepine-type drugs (1000-3200 mg/day at the beginning of treatment and 400-600 mg/day (or less) at discharge from the clinic. Reducing the dose of dibenzoazepine-type drugs reduces the likelihood of adverse effects.

The main criterion for selecting patients for surgical treatment was a lack of therapeutic effect in patients who had received conservative therapy for 3-4 months, despite taking high doses of carbamazepine (up to 4000 mg or more per day). Pronounced side effects of the preparation, such as headache, dizziness, ataxia when walking, loss of appetite, nausea, vomiting, and severe pain paroxysms that exhaust the patient necessitate surgical treatment. A neuralgic status in which pain limits the ability of the patient to consume food and fluids (leading to weight loss and metabolic disorders) is another indication for surgical treatment.

Regardless of the timing of the disease, individual intolerance to dibenzoazepine-type drugs is an indication for the expansion of the trigeminal nerve channel. In cases with prolonged medical treatment and the inability to achieve complete remission within 3-4 months, we suggest that the patient undergo spiral computed tomography to confirm neurovascular conflict. In this case, microvascular decompression of the CTN is recommended. The decision regarding the choice of treatment method remains with the patient, considering the accompanying somatic pathology of the underlying disease.

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