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Original Research

A comparative evaluation of carbamazepine and gabapentin in patients with idiopathic Trigeminal Neuralgia

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ABSTRACT:
Background: The cause of TN remains unclear. However, most cases are caused by compression of the trigeminal nerve root within a few millimeters of entry into the pons. The present study was conducted to compare carbamazepine and gabapentin, in patients with idiopathic TN. **Materials & Methods:** 90 cases of idiopathic TN were divided into 2 groups of 45 each. Group I patients received carbamazepine in the dose range of 400 mg to 1200 mg and group II patients received gabapentin in the dose range of 600 mg to 1800 mg and recalled after 3rd day, 15th day, 1 month and 3 months. The response of the patients to therapeutic effectiveness of drug was decided based on the frequency of attacks, i.e., good response, average response and nonresponsive. **Results:** Good response was seen among 23 on 3rd day, 25 at 15th day and 28 each at 1 month and 3rd month. Average response was seen in 14, 20, 22 and 22 at 3rd day, 15th day, 1 month and 3rd month. No response was seen in 8 patients on 3rd day. The difference was significant (P< 0.05). **Conclusion:** Good response was obtained with gabapentin, hence can be effectively used as first- or second line treatment for idiopathic TN cases.
Key words: Carbamazepine, Gabapentin, Trigeminal neuralgia.

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INTRODUCTION

The trigeminal neuralgia (TN) is defined as a "unilateral disorder characterized by brief electric shock-like pains, abrupt in onset and termination, and limited to the distribution of one or more divisions of the trigeminal nerve." According to the International Headache Society, TN can be classified as classical or idiopathic TN and the symptomatic TN.[1]

The cause of TN remains unclear. However, most cases are caused by compression of the trigeminal nerve root within a few millimeters of entry into the pons.[2] The nerve impingement is often accompanied by a demyelination of sensory fibres within the nerve root or the root entry zone, or less commonly in the brainstem.[3] Vascular compression by an aberrant loop of an artery or vein accounts for 80-90% of idiopathic TN. Other compressive causes are benign tumors of the posterior fossa such as acoustic neuroma, meningioma, and epidermoid cyst. A huge variety of pharmacological and surgical treatments are available for TN. The practice parameters and guidelines published in 2008 from the American Academy of Neurology (AAN), and the European Federation of Neurological Societies (EFNS) recommend starting treatment with drugs in patients with classic TN.[4]

Carbamazepine is established as an effective drug for controlling pain in classic or idiopathic TN. Other drugs such as oxcarbazepine, gabapentin, baclofen, phenytoin, lamotrigine, pregabalin, and topiramate can also be used for the treatment of idiopathic TN.[5] The present study was conducted to compare carbamazepine and gabapentin, in patients with idiopathic TN.

MATERIALS & METHODS

The present study was conducted among 90 cases of idiopathic TN of both genders. All were informed regarding the study and their consent was obtained.

Data such as name, age, gender etc. was recorded. Patients were divided into 2 groups of 45 each. Group I patients received carbamazepine in the dose range of 400 mg to 1200 mg and group II patients received gabapentin in the dose range of 600 mg to 1800 mg and recalled after 3rd day, 15th day, 1 month and 3 months. The response of the patients to therapeutic effectiveness of drug was decided based on the frequency of attacks, i.e., good response: no attacks of pain; average response: two to three attacks of pain per day; and nonresponsive with no decrease in the frequency of attacks of pain. Results were analyzed statistically.

RESULTS

Table I Distribution of patients

Groups	Group I	Group II
Drug	Tab Carbamazepine	Tab Gabapentin
M:F	23:22	20:25

Table I shows that there were 23 males and 22 females in group I and 20 males and 25 females in group II.

Table II Assessment of response to drugs in group I

Duration	Good response	Average response	No response	P value
3rd day	23	14	8	0.05
15th day	25	20	0	
1 month	28	22	0	
3 months	28	22	0	

Table II, graph I shows that good response was seen among 23 on 3rd day, 25 at 15th day and 28 each at 1 month and 3rd month. Average response was seen in 14, 20, 22 and 22 at 3rd day, 15th day, 1 month and 3rd month. No response was seen in 8 patients on 3rd day. The difference was significant (P< 0.05).

Graph I Assessment of response to drugs in group I

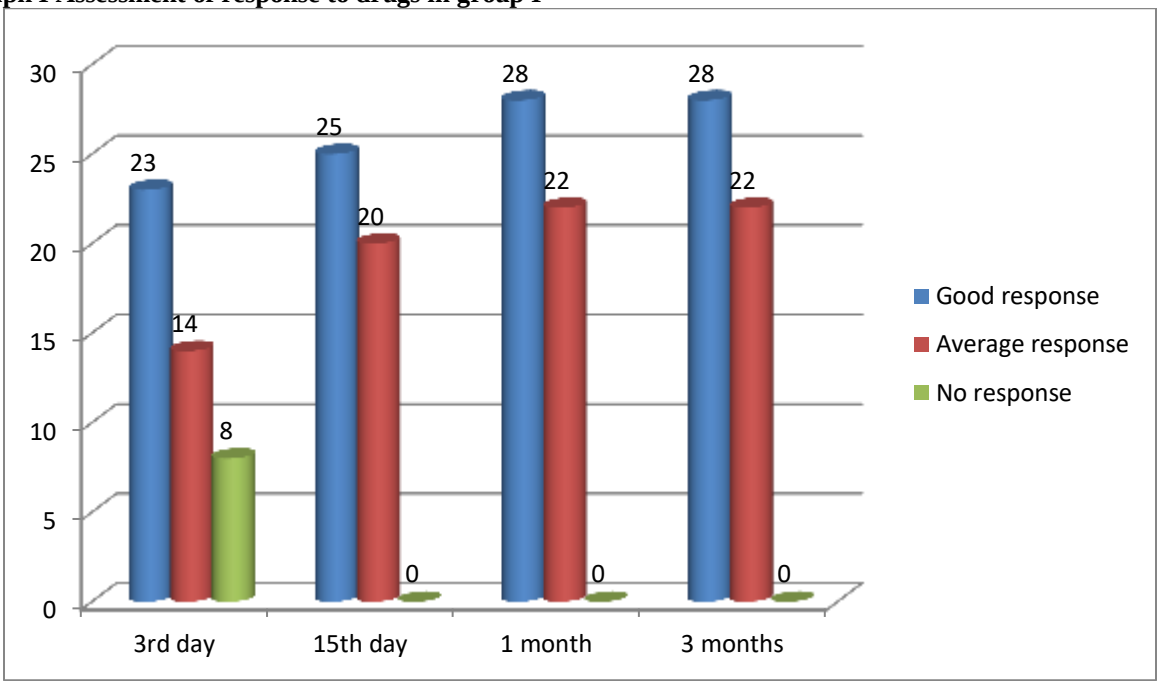
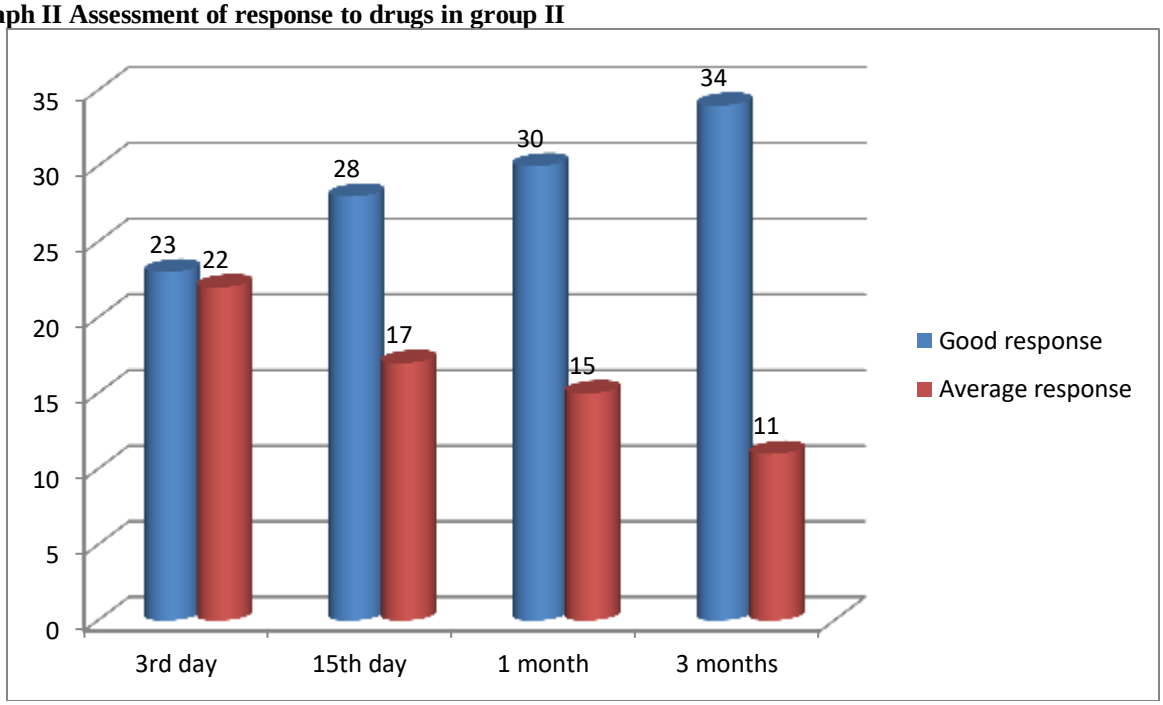


Table III Assessment of response to drugs in group II

Duration	Good response	Average response	No response	P value
3rd day	23	22	0	0.02
15th day	28	17	0	
1 month	30	15	0	
3 months	34	11	0	

Table III, graph II shows that good response was seen among 23 on 3rd day and 28 at 15th day and 30 at 1 month and 34 at 3rd month. Average response was seen in 22, 17, 15 and 11 at 3rd day, 15th day, 1 month and 3rd month. The difference was significant (P< 0.05).

Graph II Assessment of response to drugs in group II



DISCUSSION

The IHS divides TN into classic and symptomatic categories based on presumed etiology. Trigeminal neuralgia is termed classic (or idiopathic) when investigation identifies no cause other than a neurovascular compression; and examination shows no clinical evidence of neurological deficit.[6] This accounts for 80-90% of TN cases. The classification symptomatic (or secondary) is reserved for patients with TN when major neurological diseases such as MS, skull deformity, or benign compressions in the posterior fossa have been identified. Neurological examination may show sensory impairment in trigeminal nerve distribution.[7] The present study was conducted to compare carbamazepine and gabapentin, in patients with idiopathic TN.

In present study, there were 23 males and 22 females in group I and 20 males and 25 females in group II. Kaur et al[8] in their study a total of 42 patients with a mean age of 52.78 years were randomly divided into two groups A and B and were given the tablets of carbamazepine in the dose range of 400mg to 1200 mg and gabapentin in the dose range of 600mg to 1800mg and recalled after 3rd day, 15th day, 1 month and 3 month period to evaluate the response to the drugs. The therapeutic effectiveness of carbamazepine recorded as good response in 52.38% of patients of group A after 72 hours of recall while 28.57% patients had an average response and 19% patients had not relieved off pain attacks at the dose of 400mg of carbamazepine. The therapeutic effectiveness of gabapentin recorded as good response in 52.38% of group B patients after 72 hours of recall while 42.8% patients had an average response at the dose of 600mg of gabapentin.

Carbamazepine acts by inhibiting voltage-gated sodium channels and reduces the excitability of neural membranes. It also potentiates gamma amino butyric acid (GABA) receptors made up of α-1, β-2, and γ-2 subunits. In the newly diagnosed cases of TN, the usual starting dose is 100–200 mg twice daily.[9] The daily dose should be increased by 100 mg every other day until relief from pain is attained or until intolerable side

effects that prevent further upward titration. Gabapentin, a GABA receptor agonist, acts primarily on presynaptic calcium channels of neurons to inhibit the release of excitatory neurotransmitters. Gabapentin has been used in randomized control trials of neuropathic pain and was proven effective. Its use and effectiveness were also reported in several TN studies.[10] We found that good response was seen among 23 on 3rd day and 28 at 15th day and 30 at 1 month and 34 at 3rd month. Average response was seen in 22, 17, 15 and 11 at 3rd day, 15th day, 1 month and 3rd month. Campbell et al[11] reveals that the efficacy of carbamazepine is approximately 80% initially and with time higher doses may be needed to maintain efficacy, which declines to approximately 50% of patients due to auto induction of carbamazepine.

Several authors have instead emphasized the importance of physical impact of the blood vessel on the nerve. Nerve dislocation or atrophy raised the specificity to 97%. Two prospective studies have corroborated these results. Location of the neurovascular contact also appears to be relevant. Compression of the trigeminal nerve root at its entry into the brainstem increased specificity and positive predictive value to 100%, with high interobserver consistency. The degree of morphologic root changes is therapeutically relevant. Long-term outcome after surgical revision of mere neurovascular contact is uncertain compared to the decompression of dislocated, distorted, or flattened nerve roots.[12]

CONCLUSION

Good response was obtained with gabapentin, hence can be effectively used as first or second line treatment for idiopathic TN cases.

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