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IP Indian Journal of Immunology and Respiratory Medicine

Journal homepage: www.innovativepublication.com

Case Series

Intravenous magnesium sulphate and lignocaine in management of trigeminal neuralgia

Jyoti V Kulkarni[1], Sachin Patil[1,*], Rajashri Sonawane[1], Chetan Gopal Agrawal[1]

[1] *Dept. of Anaesthesiology, Government Medical College, Aurangabad, Maharashtra, India*

ARTICLE INFO

Article history:
Received 05-03-2020
Accepted 06-03-2020
Available online 13-04-2020

Keywords:
Trigeminal neuralgia
Magnesium sulphate
Lignocaine

ABSTRACT

Introduction: Trigeminal neuralgia (tn) is characterized by a recurrent, unilateral sharp pain in the distribution of branches of the trigeminal nerve. The prevalence of this condition is about 1 in 25000 people. It responds poorly to traditional analgesics; antiepileptic drugs are effective. Palliation of pain, restoration of therapeutic sleep, maintenance of function, and improvement in quality of life remain the mainstays of treatment. Magnesium could be expected to modulate neuropathic pain by blocking the NMDA receptor calcium ionophore. Intravenous lignocaine blocks neuropathic pain by action on sodium channel and blockade of central hypersensitivity. We want to report a series of 12 cases of resistant trigeminal neuralgia treated by intravenous magnesium sulphate and lignocaine.

Materials and Methods: Patients having history of recurrence to the treatment of trigeminal neuralgia in spite of treatment either by antiepileptics or neurolytic block were included in our study. In all patients detail preoperative evaluation and investigations were done. After intravenous catheter, patients received inj. magnesium sulphate 30mg/kg as an infusion in 500 ml of ringer lactate solution over a period of 1 hour followed by inj. lignocaine 2mg/kg in 500 ml of DNS once in a week for consecutive 3 weeks. During the infusion patients were monitored with continuous ECG, pulse oximetry and NIBP. Patients were asked to note the severity of pain measured on visual analogue scale (vas) from 0 to 10 (0 as no pain and 10 as severe pain). Also patients were asked to note the total dose of medications he or she already taking for pain relief, improvement in quality of pain and duration after which pain recurred.

Observation: Good pain control was observed in three patients up to nine months and were managed by tab carbamazepine 150 mg once a day after recurrence. Seven patients got recurrence after six months and were managed by tab carbamazepine and tab gabapentine. Recurrences of pain occurred after four months in two patients and were managed by inj. absolute alcohol for neurosis as no effective pain control after antiepileptic.

Conclusion: We had observed good pain control more than four months with improved quality of life. Antiepileptic drug dose requirement after recurrence of pain was less as compared to prior to magnesium sulphate and lignocaine therapy in all patients.

1. Introduction

Nociceptive pain results from a known or obvious source like trauma, metastasis, ischemia, arthritis while neuropathic pain may occur in the absence of an identifiable precipitating cause.[1] Diabetic peripheral neuropathy, post-herpetic neuralgia, antineoplastic therapy or HIV induced sensory neuropathy, tumour infiltration neuropathy, phantom limb pain, post mastectomy pain, complex regional pain syndromes and trigeminal neuralgia are common examples of neuropathic pain.[1] Neuropathic pain exhibits relatively poor response to traditional analgesics so palliation of pain, restoration of therapeutic sleep, maintenance of function, and improvement in quality of life remain the mainstays of treatment.[1]

* *Corresponding author*.
E-mail address: drsachinpatil112@gmail.com (S. Patil).

https://doi.org/10.18231/j.ijirm.2020.015

Trigeminal neuralgia (TN) is characterized by a recurrent, unilateral sharp pain in the distribution of branches of the trigeminal nerve.[2] The prevalence of this condition is about 1 in 25000 people. The painful attacks recur frequently throughout the day and night for several weeks at a time. The pain is usually in the distribution of the mandibular or maxillary branch.[3] A stepwise diagnostic and treatment approach is recommended for its treatment. Antiepileptic drugs are effective to control pain and surgical management is recommended if medical therapy fails.

Magnesium could be expected to modulate neuropathic pain by blocking the NMDA receptor calcium ionophore.[4] Intravenous Lignocaine blocks neuropathic pain by action on sodium channel and blockade of central hypersensitivity.[5] We want to report a series of 12 cases of resistant Trigeminal neuralgia treated by intravenous Magnesium sulphate and Lignocaine.

2. Case Series

Amongst twelve patients of trigeminal neuralgia three patients had underwent inferior alveolar neurectomy one year back and were receiving Tab Oxycarbamazepine 150 mg every 6hrly with poor pain control. One patient received inferior alveolar nerve block with Inj. Phenol 15 month back but had recurrence of pain after 9 months. He was receiving Tab Gabapentine 300 mg twice a day with poor pain relief. Giddiness and drowsiness were the limiting factors to increase the dose since last 6 months. Rest of the patients were started with Tab Carbamazepine 150 mg once a day and dose was increased to 150 mg every six monthly with poor pain control.

In all patients detailed preoperative evaluation and investigations were done. Written informed valid consents were taken from all the patients. After securing intravenous catheter, patients received Inj. Magnesium sulphate 30mg/kg as an infusion in 500 ml of Ringer lactate solution over a period of 1 hour followed by Inj. Lignocaine 2mg/kg in 500 ml of DNS. During the infusion patients were monitored with continuous ECG and NIBP. Same dose was repeated every week for consecutive three weeks. Patients were asked to note the severity of pain measured on visual analogue scale (VAS) from 0 to 10 (0 as no pain and 10 as severe pain). VAS score $>$ 5 is considered as recurrence and is treated with Tab Carbamazepine.

Patients were asked to note the severity of pain measured on visual analogue scale (VAS) from 0 to 10 (0 as no pain and 10 as severe pain). VAS score $>$ 5 is considered as recurrence and is treated with Tab Carbamazepine. Patients were asked to note the duration after which pain regained and total dose of drug required for pain relief. We had done follow up by telephonic contact and OPD visit by the patient up to one year.

3. Observations

Patient's age range was from 35 to 60 year and seven were female and five were male. Three patients had good pain control up to nine months and after that there was recurrence of pain which was managed by Tab Carbamazepine 150 mg once a day. Seven patients got recurrence after six months and were managed by Tab Carbamazepine 150mg once a day for first 3 months 150mg twice a day after that. Two patients got recurrence of pain after four months. In these patients medical management was not effective so we managed them by Inj. Absolute Alcohol for neurolysis. In all patients pain relief was good and patients were having improved quality of life in the form of good sleep and decreased severity and frequency of pain.

4. Discussion

Trigeminal neuralgia also known as tic douloureux or 'Fotergill's disease is neuropathic type of pain.[4] The painful attacks recur frequently throughout the day and night for several weeks. It can be triggered by stimulating certain areas on the face. Pain in TN is caused by the demyelination of the trigeminal nerve, due to either vascular compression, multiple sclerosis, amyloid infiltration, or other sources of trauma.[2] It has poor response to traditional analgesics. No cure for neuropathy exists but palliation of pain, restoration of therapeutic sleep, maintenance of function, and improvement in overall quality of life is the mainstay of treatment.[1]

A stepwise diagnosis and treatment approach is recommended. First-line therapy is Carbamazepine (300–1200mg/day) or Oxycarbamazepine (300–1800mg/day) followed by lamotrigine (200–400mg/day), pregabalin (150–600mg/day), gabapentin (1200–4200mg/day) or topiramate (100–400mg/day). If the combination therapy fails baclofen (40–80mg/day) can be tried.[6,7] surgical management is recommended if medical therapy failed. Neurolysis of trigeminal nerve either by Phenol or Absolute Alcohol, gasserian ganglion procedures, gamma knife surgery, micro-vascular decompression are different modalities of treatment.[7] As per AANEFNS (American Academy of Neurology- European Federation of Neurological Societies) guidelines, surgical options are considered in case of failure to respond to medical therapy.[3]

It is observed that neuropathic pain is due to spontaneous activity of nociceptive, myelinated, small afferent fibres. Hyper excitability of primary afferents is characterized by spontaneous impulses and repetitive firing due to changes in dorsal root ganglion Na+ channels. Damaged sensory fibres have a higher concentration of sodium channels that would increase spontaneous firing.[1] Pain is generated through a focal inflammatory process involving mRNA regulation of fast sodium channels producing

ectopic discharges.[8] Transmission of peripheral nociceptive stimulus depends on the presence of voltage gated sodium channels. Intravenous Lignocaine has both central and peripheral effects.[8] it acts on sodium channel and blocks central hypetexcitability.[5] Lignocaine is known to have dissociative effects on nerve conduction and ectopic discharges, i.e. suppression of ectopic discharges without blocking nerve conduction.[9] Lignocaine can also induce changes in neuropathic pain behaviours. However, the half-life of lignocaine ($t_{1/2}$) is 90 to 120 minutes but its pharmacological effect lasts longer. The recommended dose of Lignocaine is 3-5mg/kg intravenously.[9] We used Lignocaine in a dose of 2mg/kg to avoid conduction blockade due to additive effect of magnesium. Streptomycin sulphate dissolved in 2% lignocaine solution was deposited at the peripheral branches on the involved nerves in case of post herpetic neuralgia.[10]

The NMDA receptor plays an important role in central sensitization of the spinal cord and is important for the establishment of several chronic neuropathic pain states.[4] The high incidence of psychokinetic adverse effects of ketamine limits its use in neuropathic pain so magnesium needs to be investigated.[11]

Painful nerve stimulation leads to activation of N-methyl-D-aspartate (NMDA) receptors on the postsynaptic membrane in the dorsal horn of the spinal cord. Release of NMDA, a modulating neurotransmitter, is coupled with subsequent release of glutamate, an excitatory neurotransmitter. The resultant extended depolarization (influx of calcium and sodium and efflux of potassium) produces much larger than usual postsynaptic potentials, known as synaptic potentiation. Spinal windup has been described as "continuous increased excitability of central neuronal membranes with persistent potentiation." Neurons of the peripheral and central nervous system continue to transmit pain signals beyond the original injury, thus activating an on-going, continuous central pain response.[1,9]

Physiologically, magnesium has been demonstrated to block the ion channel on the NMDA receptor, thus preventing extracellular calcium ions from entering the cell, leading to secondary neuronal changes. This mechanism could prevent nociceptive associated central sensitisation and lessen the increased activity of wide dynamic range neurons in the dorsal horn after prolonged stimulation.[4,11] Anti-nociceptive effects of magnesium, including the inhibition of intracellular calcium influx, magnesium seems to attenuate or even prevent central sensitisation after peripheral tissue injury or inflammation because of inhibition of dorsal horn NMDA receptors.[11] Intravenous magnesium sulphate is known to reduce pain in post-herpetic neuralgia 20 and 30 minutes after infusion. There is increasing evidence that NMDA receptors are also involved in peripheral sensitization and visceral pain.[9]

We observed giddiness in four patients; decreased blood pressure and pulse rate was observed but didn't need any treatment. Hypermagnesaemia presents with headache, nausea, vomiting and diarrhoea, hypotonia and muscle weakness (serum Magnesium >4-5mmol/L). Respiratory muscle weakness and respiratory arrest occurs at serum Magnesium >5-7.5mmol/L. Hypotension, bradycardia, prolonged AV conduction, wide QRS complex and cardiac arrest occur at serum Magnesium >10-12.5mmol/L.[12]

We had observed good pain control with improved quality of life and decreased dose of antiepileptic drug for significant duration even after recurrence in all patients. So it needs to be investigated further to find effective dose and duration of therapy.

5. Source of Funding

None.

6. Conflict of Interest

Nil.

7. Acknowledgements

The authors thank nursing and paramedical staff associated with operation theatre. Special thanks to ENT departments for their co-operation.

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Author biography

Jyoti V Kulkarni Associate Professor

Sachin Patil Junior Resident 3

Rajashri Sonawane Associate Professor

Chetan Gopal Agrawal Senior Resident

Cite this article: Kulkarni JV, Patil S, Sonawane R, Agrawal CG. **Intravenous magnesium sulphate and lignocaine in management of trigeminal neuralgia**. *IP Indian J Immunol Respir Med* 2020;5(1):68-71.