International Journal of Sciences & Applied Research

www.ijsar.in

Comparison of Lignocaine 5% (hyperbaric) alone and Lignocaine 5% (hyperbaric) with Neostigmine for spinal anesthesia

Fateh Singh Bhati[1]*, Vijay Laxmi Jain[2]

[1]Dr.S.N.Medical College, Jodhpur, Rajasthan, India.
[2]S.M.S.Medical College, Jaipur, Rajasthan, India.
Correspondence Address: *Dr. Fateh Singh Bhati, 351, Mohan Nagar 'B' B.J.S. Colony, Jodhpur, Rajasthan, India.

Abstract

The present study "Comparision of lignocaine 5% (hyperbaric) alone and lignocaine 5% (hyperbaric) with neostigmine for spinal anaesthesia" was conducted in 100 female patients (ASA I & II) in age group of 20-50 years of average built (46-64 kgs) undergoing various elective gynaecological surgeries. They were randomly divided into two groups. Group I consisting of 50 patients were given only lignocaine 5% (hyperharic) 2 cc with 1 cc Dextrose 5% and group II were given lignocaine 5% (hyperbaric) 2 cc with 100 µg of neostrigmine in 1 cc of 5% Dextrose, after subarchnoid block onset of block, duration of block, side effects, heart rate & mean arterial pressure were observed at different time points. Onset was early in groupII (Lignocaine with Neostigmine) 78.80 sec. than groupI (Lignocaine alone) 97.60 seconds. Duration of analgesia & motor block was prolong in groupII[1] (Lignocaine with Neostigmine) (180 min. each) than group I (Lignocaine alone) (90 min. each). High incidence of nausea and vomiting observed in groupII[1] (Lignocaine with Neostigmine) (64% & 48% respectively) than group I (Lignocaine alone) (20% & 8% respectively). Both groups are haemodynamically stable.

Keywords: Lignocaine, Neostigmine

Introduction

Practice of spinal anesthesia for routine surgical operations assumes special importance in our country. This is chiefly because of economic reasons, lack of availability of sophisticated anesthetic apparatus and compressed gases in remote areas.

Lignocaine was discovered by Lofgren & Lundguist in 1943 in Sweden, it has enjoyed a great supremacy over several other local anesthetic agents because of being potent & yet safe with minimal incidence of side effects and toxic reactions but disadvantage is its short duration of action.

Effective control of post-operative pain remains one of the most important and pressing issues in the field of medicine as it can lead to pulmonary, circulatory, gastrointestinal, urinary dysfunction, impairment of muscle metabolism and function, thrombo embolic processes and undesirable psychological and emotional reaction. Hence, a critical need exists for effective prophylaxis and treatment of post operative pain.

Because of these reasons the search always be there to have an agent which prolong duration of action and provide effective analgesia post operatively or decrease the demand of analgesics in post operative period.

To prolong the duration of analgesia intra-operatively & post operatively intrathecal adjuncts such as opioids, vaso constrictors and α_2 – adrenergic agonists, often are added.

Thus an ideal adjunct which increase the duration without side effects has not been identified.

Neostigmine is a noval spinal analgesic with potential side effects of nausea & motor weakness. Preliminary studies suggest that small doses of neostigmine could enhance sensory anesthesia with few side effects, when added to local anesthetics for spinal anesthesia.

Neostigmine inhibits breakdown of an endogenous spinal neurotransmitter, acetylcholine which has been shown to release the nitric oxide through the action on muscarinic receptors (Predominantly) and nicotinic receptors (to a lesser extent). This nitric oxide causes analgesia in animals and preliminary clinical trials in humans, so further controlled studies are needed to clarify the analgesic and safety profile of intrathecal neostigmine.

Materials and methods

The study was conducted in J.L.N. medical College & Associated Group of Hospitals, Ajmer, on 100 patients undergoing for different types of operations. Patients including age group of 20-50 years after thorough history, physical examination and laboratory investigations to exclude any systemic disorder especially cardiac, respiratory and renal disorders. (ASA I and II) Routine investigations such as Hb estimation, BT CT, urine examination for albumin, sugar, X-ray chest PA view and standard 12 lead ECG will be done.

The protocol was approved by the medical ethics committee of our hospital pre anaesthetic evaluation of each patient was done on the day before surgery. All patients were explained regarding the type of anaesthesia and the procedure and informed consent was taken.

Patients were randomly divided into 2 groups : -

Group I :- Were received Lignocaine 5% (Hyperbaric) 2cc with Dextrose 5% 1 cc Intrathecally.

Group II :- Were received Lignocaine 5% (Hyperbaric) 2cc with Neostigmine 100μg in Dextrose 5% 1 cc Intrathecally.

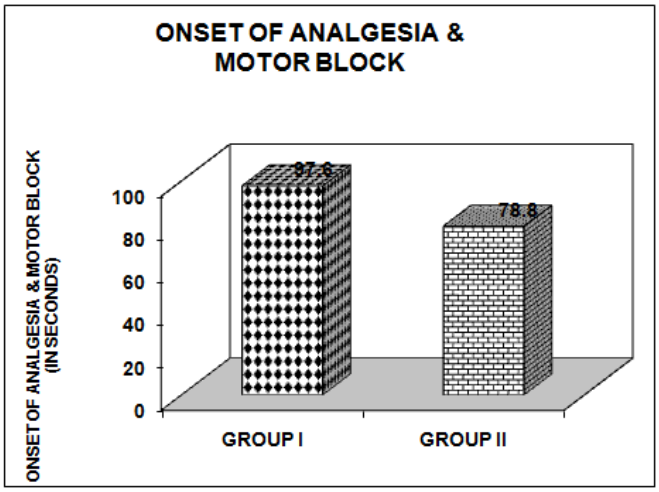
I.V. line started with 20G canula. Patient preloaded with 500 ml of Lactated Ringer's solution. After all aseptic precuations in lateral decubitus position in L_{4-5} interspace, Lignocaine 5% (hyperbaric) 2cc with Dextrose 5% 1 cc injected intrathecally in Group I, while in Group II Lignocaine 5% (Hyperbaric) 2cc with Neostigmine 100 μg in Dextrose 5% 1 cc injected intrathecally and then put patients again in supine position. Pinprick level for sensory block, pain at the site of surgical incision on visual analogue scale (VAS) and motor block (according to Bromage Motor Scale) were recorded from their onset to till they wean off. Parameters like Pulse, BP, O_2 saturation noted initially at every 5 minutes later on every 15 minutes till 4-5 hours any adverse effects like nausea, vomiting, sedation, pruritis, shivering increased salivation, respiratory depression were also noted, whenever it occurs. The severity of pain was measured using a 10 cm visual analogue scale for every 30 min till 5 hrs. The data will be recorded and will be analyzed statistically.

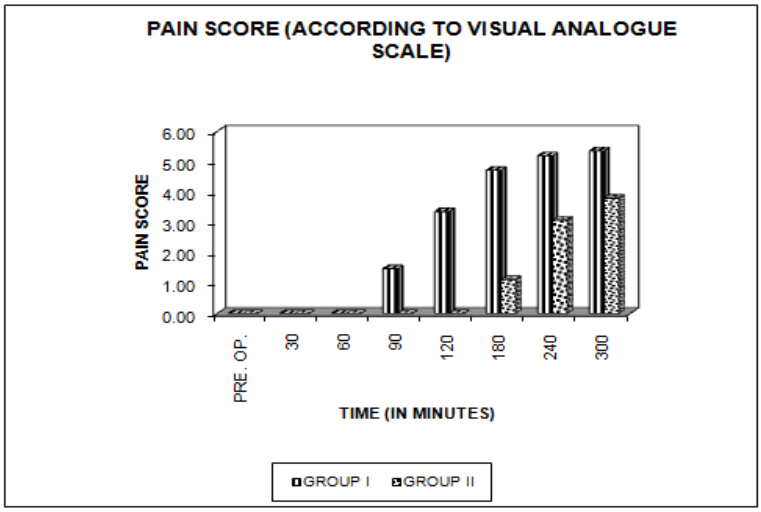
All patients were given 1500 ml of lactated Ringer's solution blood loss was replaced adequately. Nausea and vomiting was treated with I.V. metoclopramide 10mg or ondanselron 8 mg. Bradycardia (HR<60) was treated with I.V. Atropine 0.6 mg, while

hypotension was treated with I.V. mephentermine as required.

Results

1. Onset of sensory & motor block is early in groupII (Lignocaine with Neostigmine) (78.80 ±14.80 seconds) than groupI (Lignocaine alone) (97.60 ±20.06 seconds).
2. Duration of complete analgesia in groupII (Lignocaine with Neostigmine) 180 min. while in groupI (Lignocaine alone) 90 min. analgesia is highly significant ($p<0.005$) up to 240 min. and also significant till 300 min ($p<0.02$).
3. Duration of motor block more in groupII (Lignocaine with Neostigmine) (180 min.) while less in groupI (Lignocaine alone) (90 min).
4. There was no significant change in MAP and heart rate (mean ±S.D) in both the groups at different time point.
5. Incidence of bradycardia and hypotension are more in group I (Lignocaine alone) (36% & 28% respectively) than group II (Lignocaine with Neostigmine) (20% each respectively)
6. High incidence of nausea and vomiting in group II (Lignocaine with Neostigmine) (64% & 48% respectively) than group I (lignocaine alone)(20% & 8% respectively). Comparatively more in elderly than younger patients and not responding to antiemetics (metoclopremide & ondansetron).





Statistical analysis

The two groups of patients included in the study, did not differ significantly with regard to age, weight, type and duration of surgery. The average age in control group (Gr. I) was 37.77 ±5.88 years (Mean ±S.D.) and in study group (Gr. II) was 36.44 ±7.68 years (Mean ±S.D.).

The weight in group I in kgs was 55.40 ±4.22 (Mean ±S.D.) and in group II was 54.96 ±4.96 kgs (Mean ±S.D.) (Table No. 2). The duration of surgery in group I was 55.92 ±13.69 min while in group II was 84.80 ±31.67 min.

Onset of sensory and motor block is observed and onset in control group (Gr. I) was 97.60 ±20.06 seconds while in study group (Gr. II) was slightly earlier that was 78.80 ±14.80 seconds.

Pulse rate & mean arterial pressure (MAP) in both the groups are observed preoperatively & perioperatively at different points of time. No significant difference in haemodynamics of both the groups.

Motor block observed by bromage motor scores at different points of time respectively in both the groups group II had prolong motor block 180 min than group I 90 min.

According to visual analogue scale pain scores observed . It is noted that analgesia in patients of group I was up to 90 min while in group II was up to 180 min. The difference in analgesia is highly significant at all the point of time ($p<0.005$) till 240 min and also significant at 300 min ($p<0.02$).

Side effects were observed Bradycardia and Hypotension were more in group I (36% & 28% respectively) than group II (20% each respectively) while nausea & vomiting were more in group II (64% & 48% respectively) while in group I (20% & 8% respectively. Shivering was observed in one patient of group II (2%).

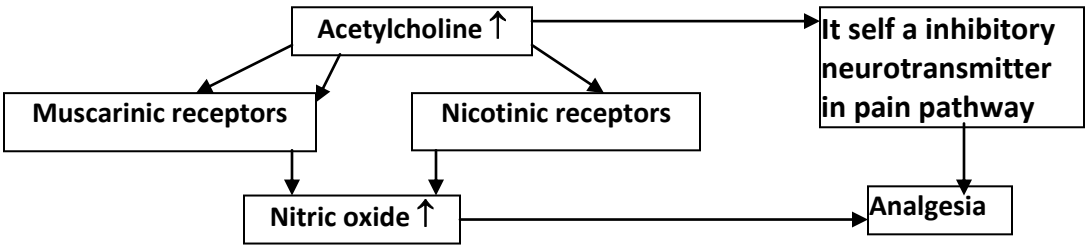
Nausea & vomiting (significant side effect of neostigmine group) also observed according to age it is less in young patients (<30 years) while more in older patients (31-50 years).

Discussion

Enhancement of sensory block by neostigmine can be explained by its intrinsic analgesic efficacy, neostigmine inhibits the breakdown of an endogenous spinal neurotransmitter acetylcholine that induces analgesia[2]. It produces analgesia directly as an inhibitory neurotransmitter in pain pathway[3] and by nitric oxide production, because increased levels of spinal cord nitrate are observed after the spinal cord administration of acetylecholine[4].

Pain, systemic opioids and spinal α_2 agonists (eg.clonidine) stimulate the release of acetylcholine in the spinal cord[5].

Opioids, α_2 agonists and anticholinesterases all increases acetylcholine which by releasing nitric oxide produces pain[6,7,8].



Prolong motor block is due to increase levels of spinal acetylcholine cause reduction in motor neuron outflow[9] and therefore motor weakness in lower extremity.

Less incidence of bradycardia & hypotension[10] in neostigmine group may be because of increase activity of sympathetic neurons by spinal neostigmine which counter acts the sympatholytic effects of spinal anaesthesia[11].

So our study correlates with previous studies and has proved that intrathecal neostigmine cause early onset of sensory & motor block[12], increases the duration of analgesia & motor block[1]. However, the higher incidence of nausea and vomiting[1] might restrict the usefulness of intrathecal neostigmine as the sole analgesic. Though intrathecal neostigmine is still under clinical trial further studies need to be conducted to establish its efficacy and side effect.

References

1. Spencer S. Liu et al, 1999.Dose Response effects of spinal neostigmine added to bupivacaine spinal anesthesia in volunteers: Anesthesiology Mar, 90: 710-7.
2. Naguib M. Yaksh TI. Et al, 1997. Characterization of muscarinic receptor subtypes that mediate antinociception in the rat spinal cord. Anesth Analg ;85:847-53.
3. Morgan GE, Mikhail MS, 1996. Pain management, Clinical Anesthesiology 2nd edition, London. Appliton&Lange ; pp281.
4. Eisenach JC, Hood DD, Carry R, 1997. Phase 1 human safety assessment of intrathecal neostigmine containing methyl and propyl parabens.Anesth Analg ; 85-842-6.
5. Hood DD, et al,1996. Interaction between intrathecal neostigmine and epidural clonidine in human volunteers.ANESTHESIOLOGY ;85:315-25.
6. Xu ZM, et al, 1997. Intravenous morphine increases release of nitric oxide from spinal cord by an α-adrenergic and cholinergic mechanism.J Neurophysiol ;78:2072-8.
7. Pan H-L, et al, 1998: Role of spinal nitric oxidein antiallodynic effect of intrathecal clonidine in neuropathic rats.ANESTHESIOLOGY ;89:1518-23.
8. Lothe A, et al, 1994 Spinal cholinergic α_2 adrenergic interactions in analgesia and hemodynamic control: Role of muscarinic receptors subtypes and nitric oxide: J Pharmacol Exp Ther ;270:1301-6.
9. Hood DD, et al, 1995. Intrathecal neostigmine mehtylsulfate in humans.ANESTHESIOLOGY, ;10:331-43.
10. Carp H, et al, 1994. Intrathecal cholinergic agonists lessen bupivacaine spinal block induced hypotension in rats.Anesth Analg ;79:112-6.
11. Pan H-L, et al, 1998. Effects of intrathecal neostigmine, bupivacaine, and their combination on sympathetic nerve activity in rats.ANESTHESILOGY ;88:481-6.
12. BJ Shah, Chemali Deb, 2000. Effects of addition of neostigmine with intrathecal local analgesics.Ind. J. Aneasth. 44:63.