ORIGINAL RESEARCH

Dexmedetomidine and ropivacaine with ropivacaine alone for supraclavicular brachial plexus block: Comparison of VAS and MBS

[1]Dr. Sowmya Jain, [2]Dr. Arunashree S, [3]Dr. Shreyawati Basavaraddi

[1]Assistant Professor, [3]Senior Resident, Department of Anesthesiology, SDM College of Medical Sciences and Hospital, Dharwad, Karnataka, India
[2]Assistant Professor, Department of Anesthesiology, Karwar Institute of Medical Sciences, Karwar, Karnataka, India

Corresponding Author
Dr.Sowmya Jain
Assistant Professor, Department of Anesthesiology, SDM College of Medical Sciences and Hospital, Dharwad, Karnataka, India
Email: sowmya.janie@gmail.com

Received: 02Sept, 2023 Accepted: 25Sept, 2023

ABSTRACT
The alpha2-adrenoceptor selectivity of dexmedetomidine is dose-dependent. At low to medium doses or at slow rates of infusion, high levels of alpha2-adrenoceptor selectivity are observed, while high doses or rapid infusions of low doses are associated with both alpha1 and alpha2 activities. Thirty cases in each group were recruited for the study and were randomized to receive Ropivacaine alone or Ropivacaine with Dexmedetomidine. A pilot study was conducted to arrive at the actual mean differences, and the outcome parameters being studied with the visual analogue scale (VAS), Modified Bromage score and mean time for first analgesic requirement. Randomization was done based on computer generated randomization method. There was no statistically significant difference in mean MBS between both the groups at baseline (p=1.0), 10 minutes (p=0.897), 30 minutes (p=1.0), 45 minutes (p=1.0), 30 minutes (p=0.050), 45 minutes (p=1.0), 60 minutes (p=1.0), 75 minutes (p=1.0), 90 minutes (p=1.0), 105 minutes (p=1.0), 120 minutes (p=1.0) and 180 minutes (p=1.0). There was a statistically significant difference at 5 minutes (p=0.046) and from 240 minutes and thereafter (p<0.05) during follow up. Dexmedetomidine group had lower VAS compared to the control group.

Key words: Dexmedetomidine, ropivacaine, VAS

INTRODUCTION

Dexmedetomidine is a new alpha2-agonist that received FDA approval in 1999 for use as a short-term (less than 24 h) sedative analgesic in the intensive care unit. Alpha2-adrenoceptor agonists have different alpha2/alpha1 selectivity. Clonidine, the first developed and the most known alpha2-agonist is considered as a partial alpha2-agonist since its alpha2/alpha1 selectivity = 200, while the alpha2 /alpha1 selectivity of dexmedetomidine is 1620 and hence is 8 times more selective alpha2-adrenoceptor than clonidine and is considered as a full alpha2 adrenoceptor agonist.[1]

The alpha2-adrenoceptor selectivity of dexmedetomidine is dose-dependent. At low to medium doses or at slow rates of infusion, high levels of alpha2-adrenoceptor selectivity are observed, while high doses or rapid infusions of low doses are associated with both alpha1 and alpha2 activities.[2]

A number of sites, both supraspinal and spinal, modulate the transmission of nociceptive signals in the CNS.Dexmedetomidine has an inhibitory effect on the locus ceruleus (A6 group) located at the brain stem.

This supraspinal action explains the prolongation of spinal anaesthesia after intravenous administration of dexmedetomidine. The noradrenergic innervation of the spinal cord arises from the noradrenergic nuclei in the brain stem including the locus ceruleus, the A5, and the A7 noradrenergic nuclei. Neurons in the locus ceruleus are connected to the noradrenergic nuclei in the brain stem.[3]

Axon terminals of the noradrenergic nuclei reach lamina VII and VIII of the ventral horns of the spinal

cord. The activity of the noradrenergic neurons is decreased by agonists acting at α2-adrenergic receptors on the locus coeruleus cell bodies. Therefore, inhibition of the locus coeruleus results in disinhibition of the noradrenergic nuclei and exerted descending inhibitory effect on nociception in the spinal cord.[4]

In addition to dexmedetomidine's action in the locus coeruleus of the brainstem, it has been shown to stimulate α2 receptors directly in the spinal cord, thus inhibiting the firing of nociceptive neurons. Even peripheral α2 adrenoceptors may mediate antinociception.

METHODOLOGY

Thirty cases in each group were recruited for the study and were randomized to receive Ropivacaine alone or Ropivacaine with Dexmedetomidine. A pilot study was conducted to arrive at the actual mean differences, and the outcome parameters being studied with the visual analogue scale (VAS), Modified Bromage score and mean time for first analgesic requirement. Randomization was done based on computer generated randomization method.

TYPE OF STUDY

A prospective study was conducted in patients of either sex requiring elective upper limb surgeries after obtaining an informed consent.

INCLUSION CRITERIA

- **AGE:** 18-70 years.
- American society of anaesthesiologists (ASA) physical status I-III.
- Elective upper limb surgeries.

EXCLUSION CRITERIA

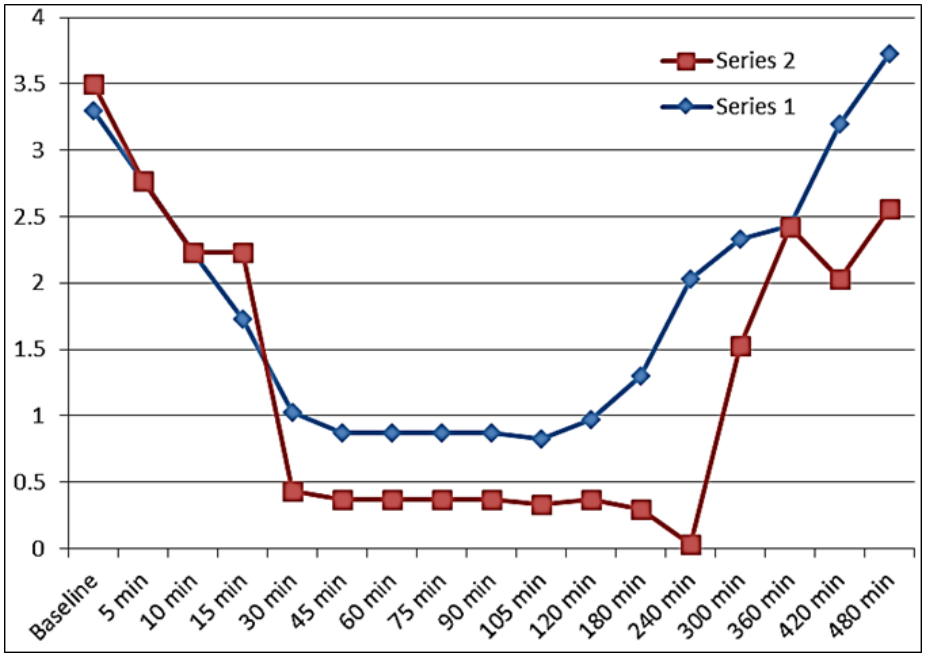
- Patient refusal for procedure.
- ASA IV and V.
- Any bleeding disorder or patient on anticoagulants.
- Severe respiratory disease.
- Neurological deficits involving brachial plexus.
- Patients with allergy to local anaesthetics.
- Local infection at the injection site.
- Patients on any sedatives or antipsychotics.
- Body mass index (BMI) >35.
- Cardiac arrhythmias.
- Advanced heart block and/or severe ventricular dysfunction.
- Those on other vasodilators or negative chronotropic agents.
- Altered sensorium and/or CNS disorders.
- Pregnant and nursing women.

Sixty patients scheduled for Elective upper limb surgery were randomized and divided into two equal groups in a double blind fashion.

Group A (control): Patients in this group (n=30) received 30millilitres (mL) of 0.5% Ropivacaine + 1mL saline.

Group B (cases): Patients in this group (n=30) received 30mL of 0.5% Ropivacaine +1microgram (µg)/kilogram (kg) Dexmedetomidine.

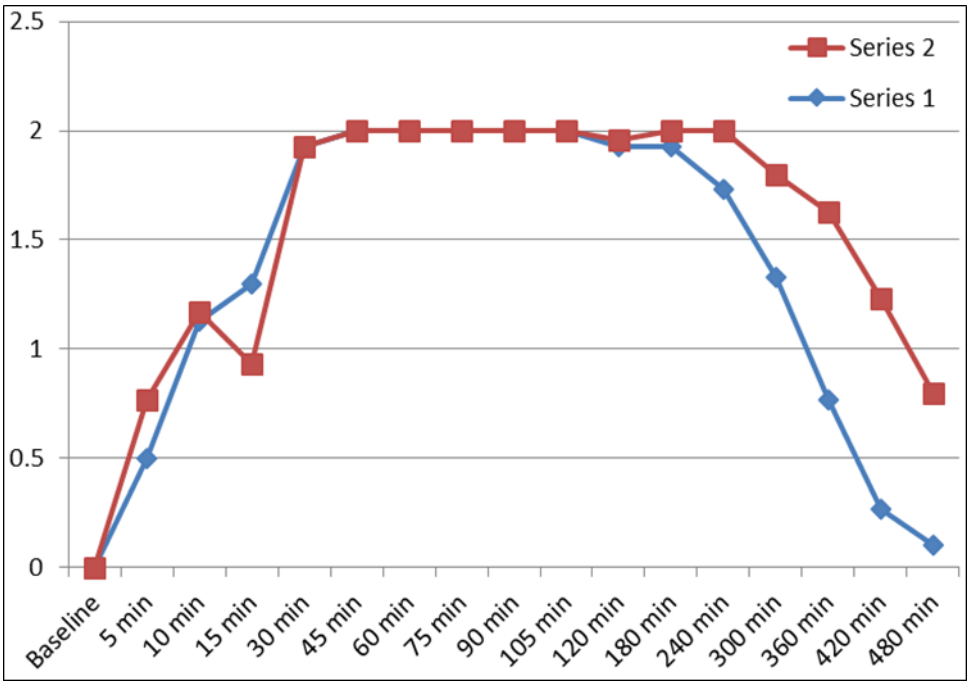
RESULTS



Graph 1: Line diagram comparing the mean of VAS between the groups at different time intervals

There was no statistically significant difference in mean VAS between both the groups at baseline (p=0.784), 5 minutes (p=0.897), 10 minutes (p=0.936), 15 minutes (p=0.196), 30 minutes

(p=0.050), 45 minutes (p=0.084), 60 minutes (p=0.084),75 minutes (p=0.062), and 105 minutes (p=0.141). There was a statistically significant difference at 90 minutes (p=0.044) and from 120 minutes onwards and thereafter (p<0.05) during follow up. Dexmedetomidine group had lower VAS compared to the control group.



Graph 2: Line diagram comparing the mean of MBS between the groups at different time intervals (series 1- Group A, series 2- Group B)

There was no statistically significant difference in mean MBS between both the groups at baseline (p=1.0), 10 minutes (p=0.897), 30 minutes (p=1.0), 45 minutes (p=1.0), 30 minutes (p=0.050), 45 minutes (p=1.0), 60 minutes (p=1.0), 75 minutes (p=1.0), 90 minutes (p=1.0), 105 minutes (p=1.0), 120 minutes (p=1.0) and 180 minutes (p=1.0).

There was a statistically significant difference at 5 minutes (p=0.046) and from 240 minutes and thereafter (p<0.05) during follow up. Dexmedetomidine group had lower VAS compared to the control group.

DISCUSSION

In a study by Shivinder Singh and Amitabh Aggarwal,[5] found that clonidine added to bupivacaine is an attractive option for improving the quality and duration of supraclavicular brachial plexus block in upper limb surgeries.

A double blind randomized control study compared Dexmedetomidine and Clonidine as adjuvants in supraclavicular brachial plexus block and found that Dexmedetomidine enhanced the duration of sensory and motor block and also the duration of analgesia. It also enhanced the quality of block as compared to clonidine.[6]

In a prospective randomized controlled trial, an ultrasound-guided infraclavicular brachial plexus block using Bupivacaine alone or combined with Dexmedetomidine for pain control in upper limb surgery was performed. It was found that there was improved analgesic efficacy, improved pain management and prolonged anaesthesia duration of local anaesthetics with the use of Dexmedetomidine as an adjunct mixed with local anaesthetics for brachial plexus blockade.[7]

Dexmedetomidine is a more highly selective α2-agonist, has a shorter duration of action than clonidine, and produces hypnosis similar to normal sleep, without ventilatory depression, making it a near ideal sedative. It suppresses nociceptive neurotransmission, terminating propagation of pain signals leading to analgesia. Its sympatholytic effect causes hypotension and bradycardia, an effect judiciously used to attenuate the stress response of surgery. Other useful effects include decreased salivation, increased glomerular filtration, decreased intraocular pressure and decreased shivering threshold.[8]

In a study by Rachana Gandhi *et al.* [9] it was found that Dexmedetomidine was a useful drug for combination with bupivacaine, as it prolongs the duration of analgesia in supraclavicular brachial plexus block.

In a study by Abdallah FW and Brull R[10] it was found that dexmedetomidine was a potential local anaesthetic adjuvant that can exhibit a facilitatory effect when administered intrathecally as part of spinal anaesthesia or peripherally as part of a brachial plexus block.

CONCLUSION

There was no statistically significant difference in mean VAS between both the groups at baseline (p=0.784), 5 minutes (p=0.897), 10 minutes (p=0.936), 15 minutes (p=0.196), 30 minutes (p=0.050), 45 minutes (p=0.084), 60 minutes (p=0.084), 75 minutes (p=0.062) and 105 minutes (p=0.141). There was a statistically significant difference at 90 minutes (p=0.044) and from 120 minutes onwards and thereafter ($p<0.05$) during follow up. Dexmedetomidine group had lower VAS compared to the control group.

REFERENCES

1. Ray M, Mondal SK, Biswas. Caudal analgesia in paediatric patients: Comparison between bupivacaine and ropivacaine. Indian J Anaesth.2003; 47(4):275-278.
2. Kuthiala G, Chaudhary G. Ropivacaine: A review of its pharmacology and clinical use. Indian J Anaesth. 2011; 55(2):104-10.
3. Concepcion M, Arthur GR, Steele SM, Bader AM, Covino BG. A New Local Anaesthetic, Ropivacaine: Its epidural effects in humans. AnesthAnalg 1990; 70:80-5.
4. Scott DB, Lee A, Fagan D, Bowler GMR, Bloomfield P, Lundh R. Acute toxicity of ropivacaine compared with that of Bupivacaine. AnesthAnalg 1989; 69:563-9.
5. Singh S, Aggarwal A. A randomized controlled double-blinded prospective study of the efficacy of clonidine added to bupivacaine as compared with bupivacaine alone used in supraclavicular brachial plexus block for upper limb surgeries. Indian J Anaesthesia 2010; 54:552-7.
6. Swami SS, Keniya VM, Ladi SD, Rao R. Comparison of dexmedetomidine and clonidine (α2 agonist drugs) as an adjuvant to local anaesthesia in supraclavicular brachial plexus block: A randomized double-blind prospective study. Indian J Anaesth 2012; 56:243-9.
7. Amany SA, Khaled MM. Ultrasound-guided single injection infraclavicular brachial plexus block using bupivacaine alone or combined with dexmedetomidine for pain control in upper limb surgery: A prospective randomized controlled trial. Saudi J Anesth.2012; 6(2):109–114.
8. Grewal A. Dexmedetomidine: New avenues. J Anaesthesiol Clin Pharmacol.2011, 27(3):297-302.
9. Gandhi R, Shah A, Patel I. Use of dexmedetomidine along with bupivacaine for brachial plexus block. National journal of medical research 2012; 2(1).
10. Abdallah FW and Brull R. Facilitatory effects of perineural dexmedetomidine on neuraxial and peripheral nerve block: a systematic review and meta-analysis. Br J Anaesth 2013; 110(6):915-25.