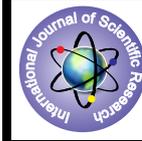


Effect of Intrathecal Dexmedetomidine Bupivacaine Combination on Duration of Subarachnoid Block and Post Operative Analgesia



Medical Science

KEYWORDS : Intrathecal, Bupivacaine, Dexmedetomidine

Dr. Rachana joshi	“PRANAV”, 5 Dwarkesh Park, behind Aalap Green City, Near Prabhu Residency, Raiya Road, Rajkot, 360007.
Dr. Jignesh mori	j-9 new lake view apartment, opp.vastrapur lake, Ahmedabad.
Dr. Kamla H. Mehta	1, 1st floor, Aalap Society, Shanti nagar, Usmanpura, Ashram road, Ahmadabad.

ABSTRACT

Objective: This study aims to compare efficacy of intrathecal bupivacaine with intrathecal bupivacaine dexmedetomidine combination on duration of subarachnoid block and post operative analgesia. **Methods:** This study included 50 patients of ASA grade I/II posted for lower abdominal surgery. Patients were divided in to 2 groups: group B received (n=25) received 0.5% heavy bupivacaine 3ml(15mg) with 0.9% normal saline 0.2ml intrathecally. group BD received (n=25) received 0.5% heavy bupivacaine 3ml(15mg) with dexmedetomidine 0.05ml (5µg) and 0.9% normal saline 0.15ml intrathecally. **Onset and duration of sensory and motor blockade, duration of analgesia and side effects were recorded. Results:** Dexmedetomidine significantly prolonged the duration of analgesia and duration of sensory and motor block as compared to intrathecal bupivacaine alone. No significant side effects were reported between two groups. **Conclusion:** In conclusion, 5µg dexmedetomidine is an attractive alternative as adjuvant to spinal bupivacaine in surgical procedures especially in those that need quite long time with minimal side effects and excellent quality of spinal analgesia.

Introduction:

Lower abdominal surgeries may be performed under local, regional (spinal or epidural) or general anaesthesia, but neuraxial blockade is the preferred mode of anaesthesia. In recent years, use of intrathecal adjuvant has gained popularity with the aim of prolonging the duration of block, better success rate, patient satisfaction and faster recovery. Dexmedetomidine is a highly selective α_2 agonist drug. It has been used in the epidural space in humans without any reports of neurological deficits.^{1, 2} Based on earlier human studies, it is hypothesized that intrathecal 5µg Dexmedetomidine would produce more postoperative analgesic effect with hyperbaric bupivacaine in spinal anaesthesia with minimal side effects.^{3, 4, 5} We planned this study to evaluate the effect of intrathecal bupivacaine dexmedetomidine combination on duration of subarachnoid block and post operative analgesia.

Material and Methods:

A clinical comparative study was carried out on 50 patients (ASA grade I/ II), 25 in each group, scheduled for elective lower abdominal surgeries. Patients were divided into the following 2 groups: Group B: 0.5% heavy bupivacaine 3ml (15mg) + 0.9% normal saline 0.2ml. Group BD: 0.5% heavy bupivacaine 3ml (15mg) + Dexmedetomidine 0.05ml (5µg) + 0.9% normal saline 0.15ml.

In the operation theatre, patients were preloaded with 10ml/kg intravenous Ringer's lactate solution before administering subarachnoid block. Sub arachnoid block was performed in left lateral position with 23G spinal needle at L3-L4 intervertebral space. After completion of procedure, patient was made supine. Standard monitoring was carried out in the form of non-invasive blood pressure, ECG and Pulse oximetry during the study period. The onset and duration of sensory blockade was assessed by using pinprick test. Motor blockade was assessed by modified bromage score. Time required for sensory block to reach level T10 was considered as sensory onset. Time for onset of grade 3 motor blockade was noted. Pulse rate, arterial blood pressure and respiratory rate were recorded every 5 minutes intraoperatively and every 30 minutes during postoperative period till the patient demand of analgesia. Hypotension was defined as >20% decrease in mean arterial pressure from baseline and treated with intravenous fluids and Mephentermine 6mg intravenous boluses. Bradycardia (pulse<60/min) was treated with intravenous atropine sulphate. Duration of surgery for each case was noted. Time for sensory regression to S2 and motor regression to bromage 0 was noted. Post operatively patients were observed for 12 hours and analgesia was given on demand of patient. Duration of analgesia was taken as time from onset of subarachnoid block to time of patient demand of analgesia.

Side effects like hypotension, bradycardia, nausea, vomiting and sedation were reported.

Statistical analysis:

The data are presented as mean±SD. The two groups were compared by unpaired two tailed t test. p<0.05 was considered significant.

Results:

There was no significant difference between the two groups with respect to age, height, weight and gender of the patients. (p>0.05). Duration of surgery was also comparable in two groups.

Table: 1 demographic profile

	Group B	Group BD
Age (years)	39.2±10.8	36.4±13.8
Gender (M:F)	10:10	9:11
Duration of surgery(mins)	91.5±23.9	92.6±21.9

Table -2: summary of results

Time (minutes)	Group B (Mean± SD)	Group BD (Mean± SD)
Time to T10 Sensory level	4.35±0.7	6.95±1.2
Time to Modified Bromage Scale III	5.2±0.8	8.45±1.0
Sensory regression to S2 from highest sensory level	215.5±26.4	321±24.0
Motor regression to bromage scale 0	196.5±27.2	298±23.4
Duration of analgesia	126.7±13.6	208±14.3

The time of sensory onset was prolonged in group BD (6.95±1.2) as compared to group B (4.35±0.7). The mean time to achieve modified bromage scale III was prolonged in group BD (8.45±1.0) as compared to group B (5.2±0.8) There was significant prolongation of duration of sensory (321±24.0) and motor blockade (298±23.4) in group BD (P value < 0.05) as compared to group B (215.5±26.4, 196.5±27.2 respectively). Duration of analgesia was significantly prolonged in group BD (208±14.3) as compared to group B (126.7±13.6). The incidence of hypotension, bradycardia, nausea, vomiting and sedation were not statistically significant between two groups.

Discussion:

This study showed that the supplementation of spinal bupivacaine with 5µg Dexmedetomidine delayed the onset of sensory

and motor block but significantly prolonged duration of sensory and motor block compared with bupivacaine alone in lower abdominal surgeries. Dexmedetomidine is a α_2 adrenoreceptor agonist having 10 times higher affinity for α_2 adrenoreceptor than Clonidine. Kanazi et al.⁵ found that 3 μ g Dexmedetomidine or 30 μ g Clonidine added to 13mg spinal bupivacaine produced the same duration of sensory and motor block with minimal side effects. Intrathecal Dexmedetomidine when combined with spinal bupivacaine prolongs the sensory block by depressing the release of C fibers transmitters and by hyperpolarization of post synaptic dorsal horn neurons.^{6, 7} Motor block prolongation may result from binding these agonists to motor neurons in the dorsal horn of the spinal cord.^{8, 9} Al Ghanem et al.³ reported prolongation of sensory (274.8 \pm 73.4) and motor block (240 \pm 64) with 5 μ g DXM as compared to 25 μ g Fentanyl with

10mg isobaric bupivacaine (179.5 \pm 47.4, 155 \pm 46 respectively). The patients in Dexmedetomidine group had longer analgesic duration as compared to bupivacaine alone. Intrathecal α_2 receptor agonists have been found to have antinociceptive action for both somatic and visceral pain. Hala E et al.¹⁰ observed that 10 μ g and 15 μ g Dexmedetomidine increased the duration of analgesia provided by spinal bupivacaine by about 240mins and 520mins respectively. No hemodynamic instability or any significant side effects were reported in Dexmedetomidine group.

In conclusion, 5 μ g Dexmedetomidine seems to be an attractive alternative as adjuvant to spinal bupivacaine in surgical procedures requiring long time with minimal side effects and excellent quality of spinal analgesia.

REFERENCE

1. Kalso E, Poyhia R, Rosemberg P. spinal Antinociceptive by Dexmedetomidine, a highly selective α_2 -adrenergic agonist. *Pharmacol Toxicol* 1991; 68:140-3. | 2. Savola M, Woodley J, Kending J, Maze M. Alpha 2b adrenoreceptor activation inhibits nociceptor response in the spinal cord of neonatal rat. *Eur J Pharmacol* 1990;183:740. doi:10.1016/0014-2999(91)90055-U. | 3. Al- Ghanem SM, Massad IM, Al-Mustafa MM, Al- Zaben KR, Qudaisat IY, Qutawneh AM and Abu- Ali HM. Effect of adding Dexmedetomidine versus Fentanyl to intrathecal Bupivacaine on Spinal Block Characteristics in Gynecological Procedures: A Double Blind Controlled Study. *Am J Appl Sci* 2009; 6:882-7. | 4. Al-Mustafa MM, Abu-Halaweh SA, Aloweidi AS, Murshidi MM, Ammari BA, Awwad ZM, et al. Effect of Dexmedetomidine added to spinal bupivacaine for urological procedure. *Saudi Med J* 2009;30:365-70. | 5. Kanazi GE, Aouad MT, Jabbour- Khoury SI, Al Jazzar MD, Alameddine MM, Al-Yaman R, et al. effect of low dose dexmedetomidine or Clonidine on the characteristics of bupivacaine spinal block. *Acta Anesthesiol Scand* 2006; 50:222-7. | 6. Lawhead RG, Blaxall HS, Bylund BD. Alpha 2A is the predominant α_2 adrenergic receptor subtype in human spinal cord. *Anesthesiology* 1992;77:983-91. | 7. Smith MS, Schumbra UB, Wilson KH et al. alpha 2 adrenergic receptor in human spinal cord: specific localized expression of mRNA encoding alpha 2 adrenergic receptor subtypes at four distinct level. *Brain res* 1995;34:109-17. | 8. Harada Y, Nishioka K, Kitahata LM et al. visceral antinociceptive effects of spinal Clonidine combined with morphine, enkephaline, or U50, 488H. *Anesthesiology* 1995;83:344-52 | 9. Yaksh TL, Reddy SVR. Studies in primate on the analgesic effects associated with intrathecal action of opiates, α adrenergic agonists and baclofen. *Anesthesiology* 1981;54:451-67. | 10. Hala E A Eid, Shafie M, Youssef H: Dose related prolongation of hyperbaric bupivacaine spinal anaesthesia by Dexmedetomidine; *Ain Shams Journal of Anesthesiology*; 2011; vol 4-2.