USE OF DEXMEDETOMIDINE ALONG WITH BUPIVACAINE FOR BRACHIAL PLEXUS BLOCK

Rachana Gandhi[1], Alka Shah[1], Ila Patel[2]

[1]Assistant Professor, [2]Associate Professor, Department of Anesthesiology, GMERS Medical College, Sola, Ahmedabad

Correspondence:
Dr. Rachana Gandhi,
L- 183, Nareshwar Apartment, Opp. Sanskrut Apartment,
Ghatlodia, Ahmedabad-380061.
Email: rinku_doc@yahoo.co.in

ABSTRACT

Introduction: Supraclavicular brachial plexus block provides safe, effective, low cost anaesthesia with good postoperative analgesia. This study was conducted to compare the postoperative analgesic efficacy and safety of dexmedetomidine for brachial plexus blockade along with bupivacaine.

Methodology: This prospective double blind study was conducted on 70 patients of age 18 to 60 years posted for various upper limb surgeries and randomly allocated into two equal groups of 35 each. Control group-C received injection bupivacaine (0.25%) 38 milliliter plus 2 milliliter normal saline, dexmedetomidine group-D received injection bupivacaine (0.25%) 38 milliliter plus dexmedetomidine 30 microgram (2 milliliter). Assessment of motor and sensory blockade, pulse, systolic blood pressure, respiration and side effects were noted every 5 minutes for first 30 minute and every 10 minute till end of surgery. Duration of analgesia and incidence of various complications following the procedure were observed.

Results: It was observed that in control group onset of motor and sensory blockade was faster. Where as, dexmedetomidine group have better hemodynamic stability and greater postoperative analgesia. Only two cases of bradycardia and two cases of hypotension were noticed in dexmedetomidine group-D.

Keywords: Bupivacaine, Dexmedetomidine, Brachial Plexus Block

INTRODUCTION

Supraclavicular brachial plexus block via Winnie's approach[1] is a very popular mode of anaesthesia for various upper limb surgeries. This approach is attractive due to it's effectiveness in terms of cost and performance, margin of safety along with good postoperative analgesia. Supraclavicular approach gives the most effective block for all portion of upper extremity and is carried out at the level of trunks of brachial plexus.[2] The plexus is blocked where it is most compact[3] i.e. at the middle of brachial plexus, resulting in homogenous spread of anaesthetic throughout the plexus with a fast onset and complete block.[4]

A variety of adjuvant has been studied for brachial plexus blockade including opioid and non opioid agents. Dexmedetomidine has shown greater affinity as an alfa 2 adrenoreceptor agonist than clonidine. The effect of dexmedetomidine when added to lidocaine for intravenous regional anaesthesia, demonstrate that addition of 0.5 miligram/kilogram dexmedetomidine to lidocaine improves quality of anaesthesia and intraoperative as well postoperative analgesia without causing side effects.[5]

The aim of the present study was to compare the postoperative analgesic efficacy and safety of dexmedetomidine for brachial plexus blockade along with bupivacaine.

MATERIALS AND METHODS:

The study was performed at GMERS Medical college Sola, from June 2011 to Dec. 2011 after institutional approval. After obtaining informed written consent and institutional approval 70 adult patients of both gender, American Society of Anesthesiology (ASA) grade 1 and 2 between age of 18-60 years, posted for various upper extremity surgeries were selected for the purpose of this study. Patients with evidence of any contraindication to brachial block like neurological deficit, history of seizures, bleeding problems, pneumothorax, and pregnancy were excluded.

After taking history, physical examination and all routine investigations were done. Before performing the procedure venous cannula 18gauze was secured in opposite hand and routine monitors like pulse oximetry, non invasive blood pressure,

electrocardiogram were attached. Study medication was prepared in identical 50 milliliter syringes to ensure blinding of anaesthetist. Investigators, who collected postoperative data, were blinded to study drug administered.

The supraclavicular brachial plexus block was performed with a 22 gauze, 38 millimeter short bevel needle using subclavian artery as a guide until paraesthesia was noted and if paraesthesia was not elicited, the first rib was encountered and after free aspiration 40 milliliter of solution was given.

The 70 patients of both genders were randomly allocated into two equal groups of 35 each (group C and D). Total 40 milliliter of solution for supraclavicular brachial plexus blockade was administered as follows- Control group-C received injection bupivacaine (0.25%) 38 milliliter + 2 milliliter normal saline and Dexmedetomidine group-D received injection bupivacaine (0.25%) 38 milliliter + Dexmedetomidine 30 microgram (2 milliliter).

Sensory block was tested using alcohol swabs. Similarly assessment of motor blockade was done using the Bromage three point score [0= normal motor function with full flexion and extension of elbow, wrist and fingers, 1= decrease motor strength with ability to move fingers and/or wrist only, 2= complete motor blockade with inability to move fingers]. After evaluation of blocks patients were given injection ondensetron 4 milligram intravenously. Patients were sedated with injection midazolam 2 milligram intravenously and oxygen is given by venti mask.

Vital parameters (pulse, respiration, blood pressure) were performed every 5 minutes for first 30 minutes and thereafter every 10 minutes till end of surgery. Postoperatively motor and sensory blockade and vital of the patients were noted every half hourly by nursing staff.

The duration of analgesia was taken from the time of onset of block to the first complaint of pain. Injection diclofenac sodium intragluteally in dose of 1-5 milligram/ kilogram was administered as a rescue analgesic.

Pain score used was visual analogue scale (0- 10):
0   - No pain
5   - Moderate pain
10   - Maximum pain.

Episodes of perioperative hypotension (systolic blood pressure < 80 millimeter of mercury), bradycardia (heart rate < 40 beats per minutes) and desaturation (SPO_2 < 90%) were recorded.

Data was expressed in Mean ± SD (standard deviation) and p value of less than 0.05 was considered statistically significant.

OBSERVATION AND RESULTS

This prospective double blind study was conducted on 70 patients of age 18 to 60 years posted for various upper limb surgeries and randomly allocated into two equal groups of 35 each. Table 1 shows demographic profile of the studied groups.

Table 1: Demographic data of studied groups

Demographic profile	Control group (C) (n= 35) Mean± SD	Dexmedetomidine group (D) (n= 35) Mean± SD
Average Age(years)	35.4 ± 20.2	36.6 ± 25.4
Weight (kg)	50.2 ± 8.9	51.4 ± 10.6
Gender ratio (M:F)	25:10	27:8

Table 2: Onset time and duration of motor and sensory block

Variables	Control group C Mean± SD (min) (n= 35)	Dexmedetomidine group D Mean± SD (min) (n= 35)	p-value
Onset time for complete motor blockade	8.5 ± 1.4	11.2 ± 2.1	< 0.0001
Duration of motor block	100.7 ± 48.3	660.2 ± 60.4	< 0.001
Onset time for complete sensory blockade	18.4 ± 2.5	21.4 ± 2.5	< 0.0001
Duration of sensory block	146.5 ± 36.4	732.4 ± 48.9	< 0.0001

The results regarding the characteristics of sensory block and motor block are summarized in table 2. The onset of both motor and sensory block in control group is faster than in dexmedetomedine group. The duration of sensory and motor block was longer in dexmedetomidine group D (p <0.00).

The duration of analgesia in control group is 194.8 ± 60.4 minute and dexmedetomidine group-D is 732.4 ± 95.1 minute, which is statistically significant (p <0.000). Vital parameters like mean pulse rate, systolic blood pressure, mean respiratory rate and mean arterial saturation values were similar in both the groups.

Table 3: Duration of analgesia

	Duration of analgesia Mean ± SD (Minutes)
Control group C (n= 35)	194.8 ± 60.4
Dexmedetomidine group D (n= 35)	732.4 ± 95.1
p-value <0.001	

The side effects were found to be insignificant and incidental. Only two cases of bradycardia and two cases of hypotension were noticed in dexmedetomidine group-D.

DISCUSSION

This prospective, randomized, double blind study was done in patients undergoing upper limb surgery. A volume of 40 milliliter of local anaesthetic agent was taken because this volume was associated with a more complete spread for brachial plexus block as found by Winnie and colleagues.[6]

Various animal studies have been conducted in rats, rabbits, dogs and sheeps using intrathecal dexmedetomidine at a dose range of 2.5 – 100 microgram without any neurological deficit. [7-11] In human beings, studies using epidural dexmedetomidine have been conducted without any report of neurological deficit. [12, 13] Intrathecal dexmedetomidine in combination with bupivacaine have been studied in human beings without any postoperative neurological deficit. [14] So that, we selected 30 microgram dose of dexmedetomidine.

Outer motor fibers of brachial plexus form the mantle and are blocked earlier than the sensory fibers at the core. [4] The onset of motor blockade was significantly faster than sensory block, this can be explained by "core and mantle" concept of Winnie et al, 1977.[15]

Kaslo et at. [16] reported that dexmedetomidine affinity to alfa 2 adrenoceptor agonists is 10 times as compared to clonidine when dexmedetomidine is added to lidocaine for intravenous regional anaesthesia, it has been studied that it improves quality of anaesthesia and intraoperative – postoperative analgesia without causing side effects. [17-19]

The lack of significant side effects like respiratory depression and haemodynamic stability make dexmedetomidine an attractive choice as an adjuvant for supraclavicular brachial plexus block.

CONCLUSION:

Dexmedetomidine is a useful drug for combination with bupivacaine, as it prolongs the duration of analgesia in supraclavicular brachial plexus block.

REFERENCES

1. Winnie AP, Collins VJ. The subclavian perivascular technique of brachial plexus anaesthesia. Anesthesiology 1964; 25: 353.

2. Urmey WF. Upper extremity blocks. In: Brown DL, editor. Regional Anaesthesia and Analgesia. Philadelphia: W.B.Saunders, 1996:254-278.

3. Lanz E, Theiss D, Jankovic D. The extent of blockade following various techniques of brachial plexus block. Anesth Analg 1983; 62: 55-58.

4. Fischer HBJ. Brachial plexus anaesthesia. In : principles and practice of regional anaesthesia; Wildsmitis JAW, Armitage EN, Mc Clure JH, editors, third edition. London: Churchill Livingstone, 2003: 193-204.

5. Esmaoglu A, Mizrak A, Akin A, Boyaci A. Addition of Dexmedetomidine to lidocaine intravenous regional anaesthesia. Eur J Anaesthesia. 2005 Jun; 22(6): 447-51.

6. Winnie AP, Radonjic R, Akkineni SR, et al. Factors influencing distribution of local anaesthetic injected into the brachial plexus sheath. Anesth Analg 1979; 58: 225-234.

7. Eisenach JC, Shafer SL, Bucklin BA, Jackson C,Kallio A. Pharmacokinetics and pharmacodynamics of intraspinal dexmedetomidine in sheep. Anesthesiology 1994; 80: 1349-59.

8. Lo WC, Harris J, Clarke RW. Endogenous opioids support the spinal inhibitory action of alfa 2-adrenoceptor agonistin the decerebrated spinalised rabbit. Neurosci Lett 2003; 340: 95-8.

9. Talke P, Xu M, Paloheimo M, Kalso E. Effects of intrathecally administered dexmedetomidine, MPV-2426 and tizanidine on EMG in rats. ActaanaesthesiolScand 2003; 47: 347-54.

10. Xu M, Kontinen VK, Kalso E. Effects of radolmidine, a novel alfa 2 adrenergic agonist compared with dexmedetomidine in different pain models in rat. Anesthesiology 2000; 93: 473-81.

11. Horvath G, Joo G, Dobos I et al. The synergistic antinociceptive interactions of endomorphin-1 with dexmedetomidine and/or S (+)- ketamine in rats. AnesthAnag 2001; 93: 1018-24.

12. Fukushina K, Nishimi Y, Mori K. Effects of epidurally administered dexmedetomidine on sympathetic activity and postoperative pain in man. AnesthAnalg 1996; 82: S121.

13. Maroof M, Khan SA, Jain D et al. Evaluation of effect of dexmedetomidine in reducing shivering following epidural anaesthesia. Anesthesiology 2004; 101: A495.

14. Kanazi GE, Aouad MT, Jabbour- Khoury SI et al. Effect of low dose dexmedetomidine or clonidine on the characteristics of bupivacaine spinal block. ActaAneshesiolScand 2006; 50: 222-7.

15. Winnie AP, Tay CH, Patel KP, Ramamurthy S, Durrani Z. Pharmacokinetics of local anaesthetics during plexus blocks. Anaesth Analg 1977; 56: 852-861.

16. Kalso EA, Poyhia R, Rosenberg PH. Spinal antinociception by dexmedetomidine, a highly selective alfa 2-adrenergic agonist. Pharmacological 1991; 68: 140-3.

17. Coskuner I, Tekin M, Rati I, Yagmur C, Elcicerk K. Effects of Dexmedetomidine on the duration of anaesthesia and wakefulness in bupivacaine epidural block. Eur J Anaeshesiol 2007; 24: 535-40.

18. Memis D, Turan A, Raramanlioglu B, Pamukcu Z, RUrt I. Adding Dexmedetomidine to lidocaine for intravenous regional anaesthesia. Anesth Analg. 2004 Mar; 98(3): 835-40.

19. Jaakola ML, Salonen M, Lehtinen R, Scheinin H. The analgesic action of Dexmedetomidine- A novel alfa 2 adreno receptor agonist in healthy volunteers. Pain 1991; 46: 281-5.