Onset and Duration of Sensory and Motor Blockade of Bupivacaine Supplemented with Clonidine and Dexmedetomidine Administered Intrathecally – A Clinical Comparative Study

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ABSTRACT

Introduction: For prolongation of anaesthesia various adjuvants were used. Present study was done to compare and evaluate the efficacy of intrathecal hyperbaric Bupivacaine 12.5 mg supplemented with 30 µg Clonidine and 3 µg Dexmeditomidine in spinal anaesthesia for gynaecological procedures

Material and Methods: The study was of 100 patients for lower abdominal surgeries for gynecological procedures, who were randomly divided into two groups to receive intrathecally either clonidine (Group I) or dexmedetomidine (Group II) combined with bupivacaine (heavy) 0.5%, and the main goal was to compare the onset and duration of sensory and motor block provided.

Results: In our study, we found that the onset of sensory block in Group I (Bupivacaine + Clonidine) to be 2.8 ± 0.75 minutes (mean ± S.D) and that by Group II (Bupivacaine + Dexmedetomidine) to be 2.6 ± 0.68 minutes (mean ± S.D) p>0.05.

Conclusion: Both the study groups shows that intrathecal adjuvants Dexmedetomidine (3mcg) and Clonidine (30mcg) were equally potent and had similar effects on sensory and motor block without any side effects.

Keywords: Dexmedetomidine, bupivacaine, clonidine, intrathecal

INTRODUCTION

Tissue injury in surgery is due to release of inflammatory mediators[1] Peripheral receptors are activated by inflammatory mediators.[1] During normal physiology, pain sensations are elicited by activity in unmyelinated (C-) and thinly myelinated (Ad-) primary afferent neurons that synapse with neurons in the dorsal horn of the spinal cord. Sensory information is then relayed to thalamus and brainstem.

Mild to severe acute pain, can affect nearly every organ function and may adversely influence postoperative outcome leading to morbidity and even mortality. Appropriate and effective management can improve quality of life. Therefore effective management of postoperative pain is not only human but is a very important aspect of postoperative care.

Postoperative analgesic modalities include regional anaesthesia and analgesia (spinal, epidural, combined spinal and epidural), peripheral nerve blocks (both upper and lower limb blocks), oral and parenteral analgesia (drugs like opioids, NSAIDS etc) intraspinal opioids as well as TENS and physical therapy and cryoanalgesia.

The sole essence of anesthesia is pain relief in peri and post operative period. Regional anesthesia has emerged as an important technique, with simplicity, effectiveness and safety as its added advantages.

Neuraxial block for lower abdominal and lower limb surgeries are becoming popular as it has many advantages over general anesthesia. Spinal anesthesia consists of temporary interruption of nerve transmission in the subarachnoid space produced by the injection of a local anesthetic solution in the subarachnoid space.

Neuraxial anaesthesia are better utilized in the prolongation of anaesthesia. In addition to these adjuvants, alpha-2 adrenergic receptor agonist are also used. The activation of this receptors represents another inherent pain control network of the central nervous system. The alpha-2 adrenergic receptor exists in the substantia gelatinosa of the dorsal horn in humans, which is a primary site of action by which this class of drugs can inhibit somatic pain. This receptor system also exists in the brain where it can produce sedation. Cardiovascular depression from alpha-2 adrenergic agonists can occur at both brain and spinal cord sites. CLONIDINE, is a partial a2-adrenoreceptor agonist used intrathecally, along with local anaesthetics it increases the motor and sensory blockade.

DEXMEDETOMIDINE, a highly selective α_2-AR agonist with a relative high ratio of α_2/ α_1 activity (1620;1)[3], which is eight times higher than that of clonidine. It possesses all the properties of analgesics, perioperative sympatholysis and haemodynamic stabilizing properties but lack respiratory depression, making it a safe adjuvant. Our aim was to compare and evaluate the efficacy of intrathecal hyperbaric Bupivacaine 12.5 mg supplemented with 30 µg Clonidine and 3 µg Dexmeditomidine in spinal anaesthesia for gynaecological procedures.

MATERIAL AND METHODS

100 patients in the age group 30-60 and ASA grade I and II were studied. Patients with spine deformity, infection at the injection site, coagulopathy, hypovolemia, increased intracranial pressure, indeterminate neurologic disease, communication problems, known hypersensitivity to local anaesthetics, were excluded from the study. Sample size was based on inclusion and exclusion criteria

Patients were allocated into two study groups – Group I receiving spinal anesthesia with 12.5mg (2.5ml) hyperbaric

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bupivacaine, 30 mcg clonidine (0.2ml) and 0.1 ml NS and group II receiving 12.5mg (2.5ml) hyperbaric bupivacaine + 0.3 ml (3mcg) of dexmedetomidine from a solution of 100 mcg diluted with 10 ml NS. A total drug volume of 2.8 ml is injected.

Pre-anaesthetic checkup was done for all patients and written informed consent was taken. On the day of surgery, patients were shifted to the operation theatre, and cannulated with an 18 G IV cannula and they were preloaded with Ringer Lactate 10ml/kg. Noninvasive blood pressure monitor, pulse oximeter and ECG leads were recorded. Supplementary oxygen was provided at the rate of 5 litres/min via a face mask.

Under strict aseptic condition, spinal anesthesia was performed. The study drug of 2.8 ml was injected into L3-L4 sub arachnoid space using 25 G quincke spinal needle after confirming free flow of cerebrospinal fluid and the time of injection will be recorded as 0 minutes.

A decrease in NIBP less than 30% (compared with mean blood pressure) was treated with 3mg of intravenous ephedrine. A heart rate of less than 50/min was treated with 0.6 mg iv atropine. VAS can assess the degree of sensory block. Sensory block height was assessed by loss of sensation to pin prick using a 22 G blunt hypodermic needle in the midclavicular line at 1 min interval after injection for 5 mins interval, until 2 consecutive levels of sensory block were identical.

Surgery was initiated once the level of sensory block reached T10. Block was considered as adequate when the sensory level reached T6. Assesments were continued every 15 mins after completion of surgery until regression to T10. The degree of motor block was assessed by the modified Bromage scale

Any adverse effects in the postoperative periods like nausea, vomiting, sedation, respiratory depression, pruritis, headache, backache or neurological symptoms etc. was noted.

The level of sedation score was assessed by Ramsay sedation scale.

STATISTICAL ANALYSIS

Descriptive statistics were used and mean and percentage were calculated. ANOVA was used for doing comparision between two drugs bupivacaine supplemented with clonidine and dexmedetomidine.

RESULTS

Spinal anaesthesia was successfully accomplished in all patients. The demographic profile was observed from time to time (Table-1). The overall quality of anaesthesia was also similar in both groups. The SBP showed a decreasing trend during the initial 15 min intra-operatively in both groups and

	Group–I (mean ± S.D.)	Group–II (mean ± S.D.)	"P" value
Onset of sensory block t_{10}	2.8 + 0.75	2.6 + 0.68	P = 0.1656 > 0.05

Table-1: onset of sensory block

	Group–I (Mean ± S.D.)	Group–II (Mean ± S.D.)	"p" Value
Onset of motor block B_1	2.9 ± 0.78	2.66 ± 0.47	P = 0.0654 > 0.05
Onset of motor block B_2	5.06 ± 1.07	4.88 ± 0.82	P = 0.3474 > 0.05
Onset of motor block B_3	7.7 ± 0.86	7.4 ± 0.97	P = 0.1936 > 0.05

Table-2: Onset of Motor Block

	Group–I (Mean ± S.D.)	Group–II (Mean ± S.D.)	"p" Value
Duration of Motor Block B_1	231.2 ± 27.6	225 ± 25.15	P = 0.2432 > 0.05
Duration of Motor Block B_2	211.2 ± 26.31	202.7 ± 23.06	P = 0.0890 > 0.05

Table-3: Duration of Motor Block

	Group–I (Mean ± S.D.)	Group–II (Mean ± S.D.)	"p" Value
Duration of Sensory block till T_{10}	253.4±28.11	264.3 ±34.07	P = 0.0837 > 0.05

Table-4: Duration of Sensory Block till T_{10}

	Group–I (*n* = 50)		Group–II (*n* = 50)		"p" Value
	N	%	n	%	
Hypotension	12	20.00	36	60.00	> 0.05
Bradycardia	20	33.33	16	26.67	> 0.05
Sedation (Ramsay's Scale): 1	0	0.00	0	0.00	> 0.05
2	52	86.00	40	66.67	> 0.05
3	8	14.00	20	33.40	> 0.05
4	0	0.00	0	0.00	> 0.05
5	0	0.00	0	0.00	> 0.05
6	0	0.00	0	0.00	> 0.05
Respiratory Depression (RR < 10/min)	0	0.00	0	0.00	> 0.05
Shivering	12	20.00	8	13.33	> 0.05
Nausea/Vomiting	4	6.67	0	0.00	> 0.05
Pruritus	0	0.0	0	0.00	> 0.05

Table-5: Quality of Surgical Anaesthesia

thereafter it was stable, but these changes were statistically not significant when compared at corresponding time intervals. No significant changes were observed in case of HR and DBP

In both the groups, sensory and motor block characteristics were same. From table-1 and 2 we can see that the onset is statistically significant. Table-3 and 4 shows that the duration is statistically significant. Table 5 depicts that side effects and sedation were almost similar in both groups.

DISCUSSION

L. Neimi et al[4] reported the duration of sensory analgesia (until block regression to L2) to be 217 min (mean) in patients who had received 0.5% bupivacaine 15 mg intrathecally, and a range of analgesia of 345 mm to 1110 minutes with a mean of 613 minutes in patients receiving 15 mg 0.5% bupivacaine with 3 mcg/kg clonidine The greater duration of analgesia in contrast to our study could have been due to the higher dosage of bupivacaine as well as clonidine.

Random assessment in 3 groups –N,D5 and D10 were done.[5] The onset times to reach T10 sensory and Bromage 3 motor block, and the regression times to reach S1 sensory level, were recorded. The mean time of sensory block to reach the T10 dermatome was 4.7 +/- 2.0 minutes in D10 group, 6.3 +/- 2.7 minutes in D5, and 9.5 +/- 3.0 minutes in group N. The mean time were different in these groups to reach S1 level. Our results were similar to that of group D5, but slightly lower due to the dose of 3mcg we used.

Our study tallied with the studies of Shukla D et al[6] where they used 15 mg hyperbaric bupivacaine plus 0.1 ml (10 µg) dexmedetomidine (group D, n=30). The onset of sensory block till T10 was 2.27 ± 1.09 mins, motor block was 3.96 ± 0.92 min, regression of sensory block was 352 ± 45 mins.

Gupta R, et al[7] conducted a study on 60 patients, they were randomly allocated to receive either 12.5 mg hyperbaric bupivacaine plus 5 µg dexmedetomidine (group D, n=30) or 12.5 mg hyperbaric bupivacaine plus 25µg fentanyl (group F, n=30) intrathecal. And their conclusion was intrathecal dexmedetomidine is associated with prolonged motor and sensory block, hemodynamic stability, and reduced demand for rescue analgesics in 24 h as compared to fentanyl.

Hyperbaric bupivacaine 0.5%, an amide local anaesthetic is presently the most common drug used for obstetric anaesthesia. Hyperbaric bupivacaine in 8% glucose is often used. Recently, several studies have confirmed that plain bupivacaine is indeed hypobaric in comparison with human CSF.[8,9] The effects of baricity on the block characteristisc have been contradictory in literature: while some studies that report the difference in baricity does not affect block characteristics[10] on the one hand, there are also studies reporting that motor block develops and disappears faster when hyperbaric solutions are used[11,12] on the other hand.

CONCLUSION

Bupivacaine 12.5 mg supplemented with 30 µg Clonidine or 3 µg Dexmeditomidine in spinal anaesthesia for gynaecological procedures produces similar effect. Dexmedetomidine 3 mcg and clonidine 30 mcg have an equipotent effect on the characteristics of the block without any significant hemodynamic instability or any side effects.

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