ORIGINAL RESEARCH PAPER

Volume-7 | Issue-12 | December-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A COMPARISON OF EPIDURAL VERSUS INTRAVENOUS ADMINISTRATION OF DEXMEDETOMIDINE AS AN ADJUVANT TO EPIDURAL ANAESTHESIA IN LOWER ABDOMINAL AND LOWER LIMB SURGERY

Anesthesiology

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ABSTRACT

BACKGROUND: Regional anaesthesia is the safe, effective means of providing surgical anaesthesia and postoperative analgesia. Drugs commonly used as neuraxial adjuvants are Opioids (morphine, fentanyl), Adrenergic agonists (epinephrine, clonidine, dexmedetomidine), and miscellaneous drugs like ketorolac, ketamine, neostigmine, butorphanol, magnesium sulphate for adding qualities like sedation, stable haemodynamics and capability to provide smooth and longer post-operative analgesia. Alpha2-adrenergic agonists such as dexmedetomidine used as an adjuvant to regional anesthesia enhance the effect of local anesthetics agents and prolong the duration of motor as well as sensory blockade and postoperative analgesia.

OBJECTIVE: This study was aimed to compare the effectiveness of dexmedetomidine given by epidural versus intravenous route as an adjuvant to epidural anaesthesia with bupivacaine for lower limb and lower abdominal surgery, regarding Onset and duration of sensory blockade, Onset and duration of motor blockade, Haemodynamic changes, Analgesia, Sedation, Side effect if any.

MATERIAL AND METHOD: Study was conducted in 54 patients of either sex belonging to ASA Grade I & II between age group of 18 to 60 years. After taking all aseptic precautions and skin infiltration with 2% lignocaine the lumber epidural block was given using 18 gauge Touhy needle in sitting position between L2-L3 or L3-L4 interspace. Epidural space was identified by loss of resistance technique. Epidural space was confirmed to exclude accidental intravascular or subarachnoid injections. Patients were divided in two groups and received drugs according to their groups. Group I: - 20ml of 0.5% bupivacaine plain + 0.5µg/kg Dexmedetomidine epidurally. Group II:-Inj. Dexmedetomidine 0.5µg/kg intravenously over a period of 10 min prior to epidural block then 20 ml of 0.5% bupivacaine plain epidurally.

RESULT: The demographic profile of patients was comparable in both the groups. Onset of sensory analgesia at T10 (9.30±1.17 vs 15.48±1.48min) and establishment of complete motor blockade (21.52±1.90 vs 28.07±2.06 min) was significantly earlier in the group I. Time to two segment dermatomal regression (142.22±9.47 vs 125.19±7.00 min) and Time to regression of motor blockade to bromage scale 1 (256.85±14.35 vs 152.59±7.12 min) were prolonged significantly in Group I as compared to Group II. Postoperative analgesia was prolonged significantly in the Group I (410.74±34.07 vs 268.15±20.20min). 92.6% patients in Group II was attained sedation score of 3 intraoperative as compared to 44.4% patients in Group I. Two patients (7.40%) in Group I had dry mouth. Nausea was observed in three patient (11.11%) in both Groups.

CONCLUSION: We conclude that Epidural Dexmedetomidine with bupivacaine provided better sensory and motor block, adequate surgical anaesthesia and stable cardiorespiratory parameters with prolonged postoperative analgesia as compared to Intravenous Dexmedetomidine.

KEYWORDS

Dexmedetomidine, bupivacaine, epidural anesthesia, lower limb and lower abdomen surgery

INTRODUCTION:

For last two decades have seen drastic evolution and improvisation in surgical methods and anaesthetic techniques. Many newer methods are tried which particularly aimed and indeed successful to a large extend to calm the patients and to eliminate the anxiety component during regional anaesthesia. [2-4]

Alpha2-adrenergic agonist very useful pharmacologic agents as an adjuvant to neuraxial anaesthesia as they provide stable haemodynamics and desirable sedation. They also decrease sympathoadrenal activity which lead to decreased oxygen demand.[5]

Recent introduction of Dexmedetomidine as alpha-2 adrenergic agonist has many beneficial effects when used as an adjuvant in regional anaesthesia. It act upon both pre and post synaptic sympathetic nerve ending and also central nervous system, hence causes decrease in sympathetic outflow and nor-epinephrine release that cause anti-anxiety, sedation, analgesia, haemodynamic and sympatholytic activity.[6]

Dexmedetomidine can be administered intravenously to achieve a desired level of sedation along with decreased inhalational anesthesia and lessen the use of opioid drugs during general anaesthesia.[7-8]

We hypothesize that intravenous dexmedetomidine might prolong the duration of epidural analgesia similar to intrathecal dexmedetomidine.

This study was carried out to compare the sensory and motor block characteristics, haemodynamic, sedative properties and analgesia potentiating effects of epidurally administered versus intravenously administered dexmedetomidine when combined to epidural bupivacaine.

MATERIAL AND METHOD

After approval from institutional ethics committee, written informed consent from patients were taken. 54 patients of either sex, age group (18 – 60 years), ASA grade I and grade II scheduled for epidural anaesthesia for lower abdominal and lower limb surgery were included in this study.

In present study patients were divided into two groups of 27 each between age 18-60 years either sex ASA grade I and II. Patient's refuse to participate in study, ASA grade III or higher, with known neurological, cardiovascular, metabolic disease, impaired renal or hepatic function, spine deformities, history of alcohol or drug abuse, psychiatric illness, coagulation disorder, infection at the puncture site and allergic to amide type of local anaesthetics were excluded from the study.

All patients were kept nil by mouth for six hours prior to anaesthesia. On arrival in the recovery room, preoperative baseline pulse and blood pressure were noted. Preloading was done with an infusion of Ringer's lactate 10 ml/kg after intravenous line was secured. Inj. Glycopyrrolate 0.004mg/kg was given by intramuscular route as premedication half an hour before surgery.

In the operation theatre, vital parameters was recorded. After taking all aseptic precautions, patients were kept in sitting position and skin infiltration was done with 2% lignocaine at L2-L3 or L3-L4 interspace and epidural space was located using 18 gauge Touhy needle with loss of resistance technique. Epidural space was confirmed to exclude accidental intravascular or subarachnoid injections. Patients were allocated to either of two groups according to inclusion criteria as below:

- **Group I:** - 20ml of 0.5% bupivacaine plain + 0.5µg/kg Dexmedetomidine epidurally.
- **Group II:**-Inj. Dexmedetomidine 0.5µg/kg intravenously over a period of 10 min prior to epidural block then 20 ml of 0.5% bupivacaine plain epidurally

After giving epidural anaesthesia heart rate, blood pressure, spo2, sensory and motor block, sedation score and side effects were recorded immediately (baseline), at every 5 min interval for half an hour, every 10 min interval for 1 hour then every 15 min interval till surgery was completed.

Block characteristics like Onset time of sensory block at T10, Maximum sensory block level, Time to maximum sensory block level, Time for complete motor block, Time to two segmental regression, Time for regression to bromage 1, Time to sensory regression at S1, Time to first rescue analgesia were noted. **Any side effects like** Nausea,Vomiting, Shivering, Headache, Dizziness, Dry mouth, Respiratory depression were also noted.

Sensory blockade assessed by pin prick method (0 - No sensation, 1 - Pin sensed as dull pressure, 2 – Sharp pain). **Motor blockade assessed by** modified bromage scale (0 - No block, 1 - Inability to raise extended leg, 2 - Inability to flex knee, 3 - Inability to flex ankle and foot). **Sedation assessed by** ramsay sedation score (1- Anxious or Agitated, 2- Cooperative and Tranquil, 3- Drowsy but responsive to command, 4- Asleep but responsive to glabellar tap, 5- Asleep with a sluggish response to tactile stimulation, 6- Asleep and no response).

Hypotension is defined as systolic BP<90 mm of hg. Bradycardia is defined as HR<60 beats/min. Inj. Mephentermine 6 mg i.v. was used to treat hypotension whereas inj. Atropine 0.6 mg i.v. was to treat bradycardia. For treatment of nausea and vomiting inj. Ondensetron 4 mg was used i.v.

Postoperative also heart rate, blood pressure, pain score by using VAS and sedation score were recorded every hourly up to 6 hours, every 2 hourly up to 12 hours and then 4 hourly up to 24 hours. Visual analogue scale (0-10) was used for post-operative pain assessment.

The statistical analysis was done by unpaired t-test for quantitative data and chi-square test for qualitative data with the usage of SPSS and Open EPI SOFTWARE.

RESULTS:

The demographic characteristics in both the groups exhibited marked similarities and did not show any statistical significant difference (*P*>0.05). The mean age, ranged from 41.96±11.46 years to 42.00±9.83 years in both the Groups. Mean weight of patients in Group-I was 57.26±8.09 kg and in Group-II was 56.81±9.74 kg.

TABLE 1: DEMOGRAPHIC PARAMETERS OF THE STUDY GROUP

Particulars	Group I	Group II	P value
	Mean± SD	Mean± SD	
Age (years)	41.96±11.46	42.00±9.83	P>0.05
Weight (kg)	57.26±8.09	56.81±9.74	P>0.05

The onset of sensory blockade at T10 found to be earlier in Group I (9.30±1.17 min) as compared to Group II(15.48±1.48 min) (P<0.001). Maximum sensory level in both Groups was T6-8. In Group I, 70.37% patients had attained T6 level while 29.63% patients had attained T8 level. In Group II, 37.03% patients had attained T6 level while 62.97% patients had attained T8 level. In present study, motor block was assessed by modified bromage scale. Time to complete motor blockade was faster in Group I (21.52±1.90 min) as compared to 28.07±2.06 min in Group II. Maximum sensory level was achieved earlier in Group I which was 16.41±1.92 min as compared to 21.85±2.33 min in Group II and was statistically highly significant (P<0.001).

TABLE 2: INITIAL BLOCK CHARACTERISTICS:

Particulars	Group I (MEAN ± SD)	Group II (MEAN ± SD)	P value
Onset time of sensory blockade at T10(min)	9.30±1.17	15.48±1.48	P<0.001
Time to complete motor block(min)	21.52±1.90	28.07±2.06	P<0.001
Time to maximum sensory block level(min)	16.41±1.92	21.85±2.33	P<0.001

The mean time to two segment regression from maximum sensory level was prolonged in Group- I (142.22±9.47 min) while 125.19±7.00 min in Group- II which was statistically significant (P<0.001). The mean time to sensory regression to S1 level was prolonged in Group I (320.74±12.68mins) as compared to Group II (217.04±20.16 min) (P<0.001). It was inferred that the duration of analgesia was prolonged in Group I as compared to Group II. Time to regression of motor blockade to bromage scale 1 was 256.85±14.35 min in Group I and 152.59±7.12 min in Group II which was statistically significant (P<0.001). Time to first rescue analgesia was 410.74±34.07 min in Group I and 268.15±20.20 min in Group II. Hence, requirement of first rescue analgesia was earlier in Group II as compared to Group I.

TABLE 3: POSTOPERATIVE BLOCK CHARACTERISTICS

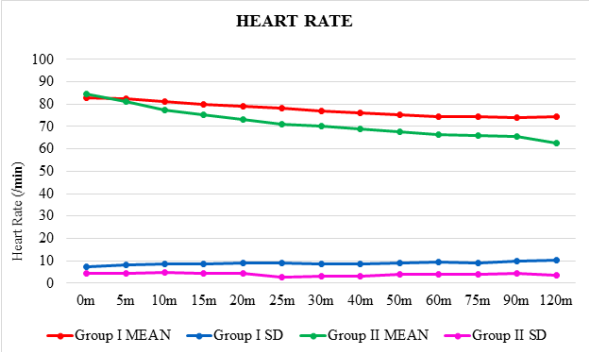
Particulars(min)	Group I (MEAN±SD)	Group II (MEAN±SD)	P value
Time to two segment regression	142.22±9.74	125.19±7.00	P<0.001
Time to regression to bromage scale 1	256.85±14.35	152.59±7.12	P<0.001
Time to sensory regression at S1	320.74±12.68	217.04±20.16	P<0.001
Time to first rescue analgesia	410.74±34.07	268.15±20.20	P<0.001

Intraoperative sedation was assessed by Ramsay Sedation Score. In Group II, 92.6% patients was attained sedation score of 3 as compared to 44.4% patients in Group I intraoperative. Thus, intraoperative sedation was better in Group II patients as compared to Group I patients.

Nausea was observed in three patient (11.11%) in both the Groups of present study. Shivering were observed in two patients (7.40%) in both Groups. Two patients (7.40%) in Group I had dry mouth. Vomiting, headache, dizziness and respiratory depression was not found in any patient during perioperative period in both the Groups.

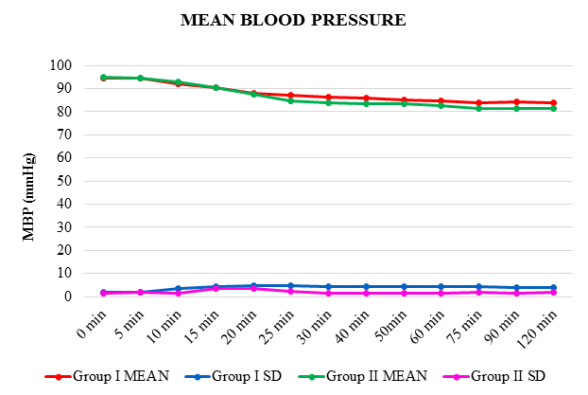
The haemodynamic parameters like heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were observed throughout intraoperative period. Fall in mean heart rate from baseline value was more in Group I as compared to Group II which was statistically significant after 10 min of epidural injection till the completion of surgery (p<0.05). None of the patient developed bradycardia in perioperative period in both the Groups.

GRAPH 1: COMPARISION OF INTRAOPERATIVE MEAN HEART RATE



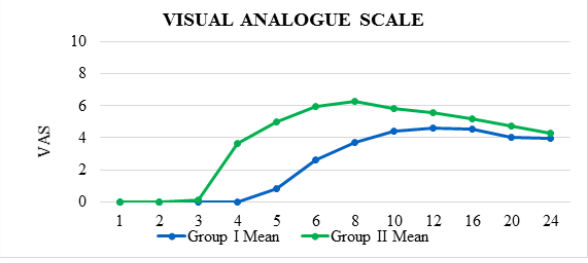
Fall in mean blood pressure from baseline 94.44±1.85 mmHg to 83.79±3.9 mmHg in Group I and 94.69±1.67 mmHg to 81.33±1.83 mmHg in Group II was observed. however the fall was within the physiological limits not requiring any treatment. Thus, patients in both the Groups proved to be heamodynamically stable.

GRAPH 2: COMPARISION OF INTRAOPERATIVE MEAN BLOOD PRESSURE



Postoperatively, all the patients were assessed for pain by 10 point visual analogue scale. When VAS ≥4, rescue analgesic inj. Diclofenac sodium 75 mg i.m. was administered. From graph 3 it was proved that the total duration of analgesia was longer in group I as compared to group II.

GRAPH 3: COMPARISION OF POSTOPERATIVE PAIN BY MEAN VISUALANALOGUE SCALE



DISCUSSION:

Neuraxial anaesthesia greatly expands the anaesthesiologists' armamentarium, providing alternatives to general anaesthesia when appropriate[9]. A variety of neuraxial (primarily epidural) and peripheral regional analgesic techniques may be employed for the effective treatment of postoperative pain.[10] Epidural anaesthesia can attenuate the "stress response" to surgery, improve the quality of postoperative analgesia in comparison with systemic opioids, and hasten recovery of gut function[11]. The addition of adjuvants like opioids or α-2agonists provide dose-sparing effects of local anaesthetics and would accelerate the onset of sensory blockade of epidural anaesthesia and decrease the effective dose of local anaesthetic.[12] Dexmedetomidine acts on pre and post-synaptic sympathetic nerve terminal and central nervous system to decrease the sympathetic outflow and nor-epinephrine release causing sedation, analgesia, sympatholytic and haemodynamic effects. Motor blockade tends to be denser with dexmedetomidine[1].

The demographic profile in the present study was comparable to similar other studies and did not show any significant difference on statistical comparison.

The time to reach peak sensory level was significantly (P<0.001) shorter in group I (16.41±1.92 min) as compared to group II (21.85±2.33 min). The onset of sensory blockade at T_{10} found to be earlier in Group I (9.30±1.17 min) as compared to Group II (15.48±1.48 min) (P<0.001). Time to complete motor blockade was faster in Group I which was 21.52±1.90 min as compared to 28.07±2.06 min in Group II. Time to regression of motor blockade to bromage scale 1 was 256.85±14.35 min in Group I and 152.59±7.12 min in Group II.

It was inferred that the duration of analgesia was prolonged in Group I as compared to Group II. Dexmedetomidine decreases sympathetic outflow by its action on pre and post-synaptic sympathetic nerve terminal and Cenrtal Nervous System. It enhances the effect of local anaesthetics by blocking conduction through Aa and C fibers. Thus addition of Dexmedetomidine to bupivacaine epidural anaesthesia results in early sensory block and prolonged duration of sensory analgesia.

The haemodynamic parameters like HR, SBP, DBP and MAP were observed throughout intraoperative period. Fall in HR, SBP, DBP and MAP was statistically significant in Group II as compared to Group I, however the fall was within the physiological limits not requiring any treatment. Thus, patients in both the Groups proved to be heamodynamically stable. These hemodynamic changes were due to decrease in central sympathetic outflow and nor-epinephrine release. Presynaptic α-2 receptors are stimulated, thereby decreasing norepinephrine release, causing a fall in blood pressure and heart rate. The dose dependent bradycardia of dexmedetomidine is primarily mediated by the decrease in sympathetic tone and partly by baroreceptor reflex and enhanced vagal activity.

Postoperatively, all the patients were assessed for pain by 10 point visual analogue scale. When VAS ≥4, rescue analgesic inj. Diclofenac sodium 75 mg i.m. was administered. Time to first rescue analgesia was 410.74±34.07 min in Group I and 268.15±20.20 min in Group II. This proved that the total duration of analgesia was longer in group I as compared to group II. In the spinal cord, activation of both α2-C and α2-AR receptors, situated in the neurons of superficial dorsal horn directly reduces pain transmission by reducing the release of pro-nociceptive transmitter, substance P and glutamate from primary afferent terminals and by

hyperpolarizing spinal interneurons.[13] In very few patinets side effects like only nausea and shivering was observed which was insignificant in both the Groups.

Bajwa S J, Arora V and their Colleagues studied the effect of Dexmedetomidine and Fentanyl for epidural analgesia in lower limb orthopaedic surgeries. They divided patients randomly into two Groups. Group RD of 50 patients received 0.75% Ropivacaine 15ml + 1 µg/kg Dexmedetomidine and Group RF of 50 patients received 0.75% Ropivacaine 15ml + 1 µg/kg Fentanyl epidurally. They concluded that Dexmedetomidine seems to be a better alternative to Fentanyl as an adjuvant as it provides comparable stable haemodynamics, early onset and establishment of sensory blockade, prolonged postoperative analgesia and lower consumption of local anaesthetic agent postoperatively for epidural analgesia with better sedation.[14]

Velayudha Sidda Reddy and their co-workers studied Intravenous Dexmedetomidine versus Clonidine for prolongation of Bupivacaine spinal anaesthesia and analgesia**.** they concluded that Single dose of intravenous Dexmedetomidine resulted in an early onset action of Bupivacaine, rapid establishment of both sensory and motor blockade, prolonged duration of analgesia into the postoperative period and stable cardiovascular parameters thereby making Dexmedetomidine an effective adjuvant than Clonidine for Bupivacaine spinal anaesthesia.[15]

Vaibhav Shahi and their colleagues studied the effect of magnesium sulphate vs Dexmedetomidine as an adjunct to epidural Bupivacaine in lower limb surgeries. They stated that addition of 0.5 mg/kg epidural Dexmedetomidine seems to be an attractive alternative as adjuvant to epidural Bupivacaine for prolonged surgeries, with minimal side effects and excellent postoperative analgesia.[16]

Kumkum Gupta et al studied Epidural 0.5% Levobupivacaine with Dexmedetomidine versus Fentanyl for vaginal hysterectomy. they concluded that Epidural Levobupivacaine with Dexmedetomidine provided better sedation, adequate surgical anaesthesia and stable cardiorespiratory parameters with prolonged postoperative analgesia for vaginal hysterectomy with no difference in the level of sensory block was found between Dexmedetomidine and Fentanyl.[12]

Hadil Magdy et al studied the effect of intrathecal compared with intravenous Dexmedetomidine as an adjuvant to spinal Bupivacaine anaesthesia for caesarean section. they concluded that Adding intrathecal Dexmedetomidine or intravenous Dexmedetomidine as an adjuvant to spinal Bupivacaine in women undergoing elective caesarean section improved the intraoperative condition like sedation and analgesia and the quality of postoperative analgesia without neonatal or maternal side effects.[17]

CONCLUSION:

From the above study it was concluded that Epidural Dexmedetomidine with bupivacaine provided better sensory and motor block, adequate surgical anaesthesia and stable

cardiorespiratory parameters with prolonged postoperative analgesia as compared to Intravenous Dexmedetomidine with minimum or none of the side effects of any significant by either of the route.

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