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"A COMPARATIVE STUDY OF EFFICACY OF INTRATHECAL VS INTRAVENOUS DEXMEDETOMIDINE AS AN ADJUVANT TO BUPIVACAINE IN SPINAL ANAESTHESIA FOR BELOW UMBILICAL SURGERY"

Medical Science

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ABSTRACT

Background: The aim of the present study was to compare the block characteristics of dexmedetomidine when used intravenously and intrathecally as an adjuvant to 0.5% hyperbaric bupivacaine for below umbilical surgeries. **Material and methods:** Sixty patients scheduled for below umbilical surgeries were randomly allocated to receive either 12.5mg hyperbaric bupivacaine plus 5mcg dexmedetomidine intrathecally (group D) or 0.5mcg/kg dexmedetomidine intravenously (group A) along with 12.5mg hyperbaric bupivacaine intrathecally. Haemodynamic parameters, onset and duration of sensory and motor block, sedation and pain score were assessed. **Results:** Both groups were comparable in terms of age, weight, sex, ASA grade, side effects and onset of sensory and motor block. Duration of sensory blockade was significantly prolonged in dexmedetomidine intrathecal group (344.43±34.09 minutes) compared to intravenous group (280.03±22.22 minutes) (p value <0.001). Duration of motor blockade was significantly prolonged in dexmedetomidine intrathecal group (298.16±25.23 minutes) compared to intravenous group (259.46±21.87minutes) (p value <0.001). Intra operative sedation score were higher in dexmedetomidine intravenous group whereas duration of analgesia was longer in intrathecal group. **Conclusion:** Study successfully demonstrated that the Dexmedetomidine is a better intrathecal adjuvant in spinal anaesthesia as far as patient comfort, stable cardio-respiratory parameters, intra-operative, post-operative analgesia and adverse effects were concerned.

KEYWORDS

Below umbilical surgery, Spinal anaesthesia, Dexmedetomidine.

INTRODUCTION

Preference to neuraxial anaesthesia has been an emerging trend in lower abdominal and lower limb surgeries [1,2].With the increasing awareness that postoperative analgesia plays an important role in better postoperative outcomes of patients, intrathecal adjuvants are used for better postoperative analgesia.[2] Many adjuvants such as opioids like, fentanyl, morphine and alfa-2 agonist like, clonidine and dexmedetomidine have been tried to prolong the duration of spinal anaesthesia, as well as the duration of postoperative analgesia and to reduce the dose of local anaesthetic agents and their side effects[3].

Dexmedetomidine, a highly selective alfa-2 adrenorecepter agonist has been used for premedication and as an adjuvant to general anaesthesia. Dexmedetomidine, an imidazole compound, is a pharmacologically active dextroisomer of medetomidine that display specific and selective alfa-2 adrenorecepter agonism [4]. Activation of the receptors in the brain and spinal cord presynaptically and postsynaptically inhibits neuronal firing causing hypotension, bradycardia, sedation, and analgesia.

In previous studies, authors have compared the efficacy of IV (0.5 mcg/kg) VS intrathecal bupivacaine and a significant prolongation in the durations of motor and sensory block was observed[5,6].

We hypothesized that intravenous dexmedetomidine as adjuvant in SAB might have analgesic efficacy comparable to intrathecal dexmedetomidine. Thus we undertook the study of efficacy of intravenous VS intrathecal dexmedetomidine as an adjuvant to hyperbaric bupivacaine in SAB for below umbilical surgery.

MATERIAL AND METHODS

After obtaining institutional ethical committee's approval and written informed consent of the patients, the present prospective randomized study was conducted on sixty patients of ASA grade I and II, aged between 20 and 50 years, of either sex, who were undergone below umbilical surgeries in the Department of Anaesthesiology, Government Medical College and Associated Group of Hospitals Kota (Rajasthan).

Patients were randomly divided into two groups by sealed envelope method. Group A- Patients were received intravenous dexmedetomidine 0.5 mcg/kg in 100 ml of 0.9% normal saline over a period of 15min administered 20 min prior to subarachnoid block. Subarachnoid block was instituted using intrathecal 0.5% heavy bupivacaine 12.5mg (2.5ml) with 0.5ml of normal saline.

Group D-Patients were received 100ml 0.9% normal saline intravenously over a period of 15min administered 20min before subarachnoid block. Subarachnoid block was instituted using 0.5% heavy bupivacaine 12.5mg with 5mcg of dexmedetomidine (0.5ml).

Before spinal anaesthesia,The study drug solution was prepared by anaesthesia resident who was not being involved in the study for recording of parameters or care of patients. Both patient and anaesthesiologist performing the spinal block were blinded to the study drug.

After intravenous insertion of an 18-G cannula in the operating room, all patients were preloaded with Ringer's lactate 15 ml/kg over 10 mins. All the routine monitors were attached, and the preoperative baseline readings of non-invasive blood pressure, pulse rate (PR), and saturation were noted.

For the subarachnoid block, patients were placed in the sitting position and after adequate aseptic precautions lumbar puncture was performed at L3-L4 intervertebral space using a standard midline approach with a 25-G Quincke needle. After ensuring a free flow of CSF, the drug doses were injected intrathecally according to group A and group D as mentioned above respectively.

Following Observations Were Noted Intra And Post Operatively:

Sensory blockade was assessed by loss of sensation to pinprick method using 26 G hypodermic needle, bilaterally in the midclavicular line.Sensory level was assessed every min for the first 10 minutes and thereafter every 10 min until regression to S-2 dermatome.Time of onset of sensory blockade was defined as completion of intrathecal injection to the loss of pinprick sensation at umbilical level (T-10 dermatome).Highest level of sensory block and time taken to achieve highest level of sensory block was noted.

Duration of sensory anaesthesia was defined as a time of onset of sensory block to sensory regression to S- 2 dermatome.

Degree of motor blockade was determined according to Modified Bromage scale[7] Onset of motor block was defined as a time from intrathecal injection till the patient is unable to flex ankle and foot (modified Bromage score 3).Duration of motor block was recorded and considered as time from onset of motor block to recovery of modified Bromage score 0.

Intraoperatively, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR), oxygen saturation (SpO2) were recorded every 5 minutes until the end of surgery and every 15 minutes in the first postoperative hour followed by every 30 minutes for next 6hours. Hypotension, defined as fall of MAP by >20% from baseline or fall of SBP below 90 mmHg, was treated with incremental intravenous doses of mephentermine 6 mg and IV fluid as required. Bradycardia, defined as HR <50 bpm (beats per minute) or a decrease by >20% from baseline was treated with injection atropine 0.6 mg intravenously.

The sedation was assessed intraoperatively and postoperatively by amodified Ramsay Sedation scale[8] along with haemodynamic parameters.

All patients were observed for postoperative analgesia. Pain intensity was measured using a 10 cm Visual Analogue Scale (VAS) on 0 to 10 points (0 = no pain and 10 = worst pain), every 30 minutes for 6 hours. VAS score < 3 was considered as effective analgesia. Duration of effective Analgesia was measured as the time from intrathecal injection to the first administration of rescue analgesic (when VAS reaches ≥3). Diclofenac (75 mg) was given intravenously as rescue analgesic. Time for the first request for postoperative analgesia and the number of patients who required supplemental analgesic, were recorded.

All patients were observed postoperatively for any complain of pruritus, nausea, vomiting, hypotension, bradycardia, respiratory depression, and post-spinal shivering. Intra and postoperative complications were noted and managed accordingly.

All the data were reported as mean±S.D. and results were subjected to statistical analysis. Statistical analysis was done using Chi-square test and Student t test. P less than 0.05 was considered as statistically significant.

RESULTS

The demographic characteristics were comparable between the groups (Table-1). There was no significant difference between onset of sensory, motor block and highest sensory block level achieved. Time to reach highest sensory block level was significantly higher in group D compared to group A. Duration of sensory and motor block were significantly higher in group D (P<0.05), Duration of analgesia was significantly longer in group D and also total number of doses of rescue analgesic required in first 24 hours postoperatively was significantly less in group D (P<0.05).

Haemodynamic parameters (HR/MAP) were comparable in both the groups(Fig-1,2), Sedation score assessed by Modified Ramsay scale was higher in group A compared to group D (Fig-3), The adverse effects noted were also similar in the two groups. Six patients in group A and three patients in group D had Bradycardia.

Table 1: Demographic characteristics

Variable	Group A (N=30)	Group D (N=30)
Age (years)	41.13±8.0	41.46±7.48
Weight (kg)	60.06±4.33	60.04±4.01
Sex (Male/Female)	12/18	12/18
ASA grade (1/2)	17/13	12/18

Table 2:Patient characteristics

Variable (minutes)	Group A (N=30)	Group D (N=30)
Onset time of sensory block(minutes)	3.14±0.53	3.19±0.678
Onset time of motor block(minutes)	4.61±0.41	4.54±0.54
Highest sensory level	T8 (T6-T10)	T6 (T6-T10)
Time to achieve highest sensory level(minutes)	6.87±0.45	8.12±0.45*
Time to 2 segment regression of sensory block(minutes)	118.73±13.19	175.13±17.72*
Duration of sensory block(minutes)	280.03±22.22	344.43±34.09*
Duration of motor block(minutes)	259.46±21.87	298.16±25.23*
Durationof analgesia(min)	355.2±23	597±18.61*
Total doses of rescue analgesic used in 24 hours	2.26±.086	1.76±0.62*

Data presented as Mean±SD or n

*P < 0.05 statistically significant, Group D vs Group A

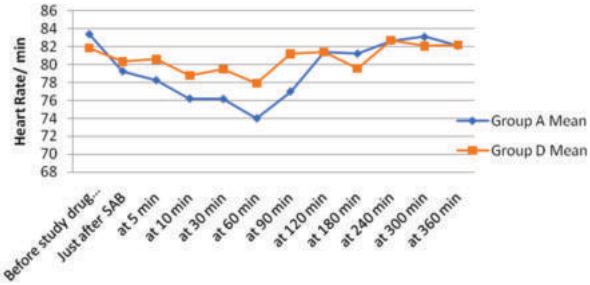

Fig 1: Change in Heart Rate

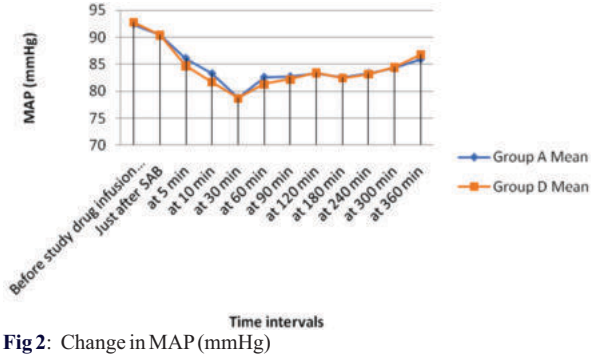

Fig 2: Change in MAP (mmHg)

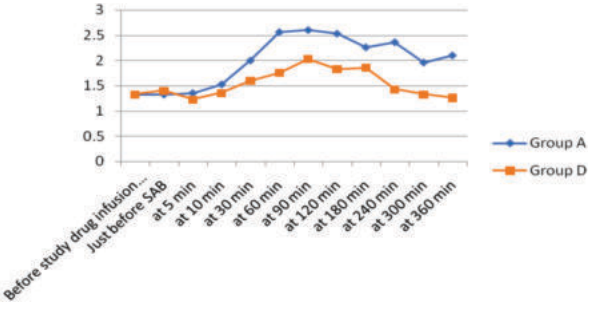

Fig 3: Sedation score

DISCUSSION

The molecular mechanisms of the analgesic action of alfa-2 agonists are through activation of inwardly rectifying G1-protein-gated potassium channels, resulting in membrane hyperpolarization thus decreasing the firing rate of excitable cells in the central nervous system[(4)]. In addition, alfa-2 agonists inhibit neurotransmitter release through reduction in calcium conduction in to the cell. These two mechanisms represent two very different ways of affecting analgesia, first by preventing the nerve from firing and second, by inhibiting propagation of the signal into its neighbour. This encouraged the researchers to use dexmedetomidine with spinal anaesthesia either intravenously or intrathecally. In this study, majority of patients were middle aged. The sex ratio, weight,ASA grade, type of surgeries performed were also identical in both groups. This avoids variations in intraoperative and postoperative outcome of patients.

In present study, time of onset of sensory block was comparable in both groups. Time of onset of motor block in group D is comparatively faster than group A. but the results were statistically insignificant. Similar results were observed in studies conducted by Hamed AMS and Taalaat SM (2013)[4], Songir S et al (2016)[5], Sharma A et al (2020)[6], Sharma I et al (2020)[1]

In contrast to our study, Elgebaly A S et al (2018)[2], Sudhakar B et al (2017)[7]observed delayed onset of sensory block that was 5.9±0.9 min in dexmedetomidine intravenous group and 2.6±0.6 min in dexmedetomidine intrathecal group.

In presentstudy time from injection to time to highest sensory block level in group D was significantly higher as compared to group A.Howeverstudy conducted by Hamed AMS and Talaat SM (2013)[4], Magdy H et al (2013)[8], Jain R (2017)[9]observed no significant change between two groups.

In contrast to our study, Sudhakar B et al (2017)[7] observed that mean time for attaining maximum height of sensory block was 8±4 min for intrathecal group and 11±5 min for intravenous group with p value of 0.02 which was statistically significant.

In our study time to two segment sensory regression in group D was significantly longer as compared group A. Similar results were observed in study conducted by Sharma A et al (2020)[6] where time to 2 segment regression was 194.23±42.70 min in dexmedetomidine intrathecal group and 174.60±31.32 min in dexmedetomidine intravenous group.

In accordance with our results, Hamed AMS and Talaat SM (2013)[4], Jain R (2017)[9] also found a longer time to 2 segment sensory regression in patients who received dexmedetomidine intrathecally.

Duration of motor block was significantly longer in group Das compared to group A,(P value of < 0.001). Similar results were observed in study conducted by Hamed AMS and Talaat SM (2013)[4], Songir S et al (2016)[5], Sudhakar B et al (2017)[7],Sharma A et al (2020)[6],where duration of motor block in dexmedetomidine intrathecal group was higher as compared to intravenous group. In contrast to our results, Magdy H et al (2013)[8] observed that there was no statistically significant change in duration of motor block between dexmedetomidine intrathecal or intravenous groups. Elgebaly A S et al (2018)[2]observed a significantly longer in dexmedetomidine intravenous group than intrathecal group.

In our study duration of sensory block was higher in group D as compared to group Aand difference was significant with p value of <0.001. Similar to our results, Magdy H et al (2013)[8], Sudhakar B et al (2017)[7], Sharma A et al (2020)[6], Sharma I et al (2020)[1] also observed prolonged duration of sensory block in dexmedetomidine intrathecal group than intravenous group.

In contrast to our study, duration of sensory block was found to be more with dexmedetomidine intravenous group (294.1±15.1) than intrathecal group (230.4±2.5) as observed by Elgebaly A S et al (2018)[2].

Similar to observations of Sudhakar B et al (2017)[7], Sharma A et al (2020)[6], the incidence of fall in Blood Pressure and Heart Rate was comparable in both groups.

In our study, VAS scores in both the groups were comparable till 90 min after subarachnoid block, and thereafter the VAS scores were significantly lower in group A as compared to group D. Similar results were observed by Abdou AMH et al (2018)[10] who also recorded a significantly lower VAS score in patients who received intravenous dexmedetomidine compared to patients who had received intrathecal dexmedetomidine.In contrast to our study Sharma I et al (2020)[1] observed lower VAS score in intrathecal group than intravenous group. Patients who received intravenous dexmedetomidine had more sedation than patients who received intrathecal dexmedetomidine and difference was statistically significant. This finding was in accordance with the study of Jain R et al (2017)[9].

Dexmedetomidine an alpha-2 adrenergic agonist used in subarachnoid block with hyperbaric bupivacaine significantly prolongs duration of sensory and motor blockade and duration of analgesia. Dexmedetomidine is a better intrathecal adjuvant in spinal anaesthesia as far as patient comfort, stable cardio-respiratory parameters, intra-operative, post-operative analgesia and adverse effects were concerned. Over all the experience with dexmedetomidine used intrathecally was quite satisfactory as compared when it was used as intravenous infusion because of its superior sensory and motor blockade during the surgical procedure under regional anaesthesia. Use of dexmedetomidine as an intrathecal adjuvant is an alternative to achieve good anaesthetic quality.

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Conflict Of Interest: None to declare

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