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Evaluation of the effect of intravenous dexmedetomidine on characteristics of spinal anaesthesia with hyperbaric bupivacaine

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Abstract

Aims: A study was performed to evaluate the effect of intravenously administered dexmedetomidine just before spinal anaesthesia, on spinal block characteristics, postoperative analgesia, haemodynamics and sedation.

Methods: Sixty patients, aged 18 - 50 years of either sex, ASA grade I & II, who were undergoing elective lower abdominal and lower limb surgeries under spinal anaesthesia with hyperbaric bupivacaine, were randomly allocated in to two equal groups of 30 patients each to recieve 20 ml of normal saline intravenously (group A) and intravenous dexmedetomidine 0.5 mcg/kg prepared in normal saline to a total volume of 20 ml (group D) over a 10 min period as a single dose just before spinal anaesthesia. Onset and duration of sensory blocks and motor blocks, highest sensory block level, time to 2 segment regression, duration of analgesia, perioperative haemodynamic parameters, VAS and sedation scores were assessed.

Results: Both groups were comparable with regard to demographic data. The onset of sensory and motor block were significantly earlier in group D as compared to group A. Duration of motor block and analgesia were significantly longer in group D as compared to group A. Sedation score were significantly higher in group D. Though HR and NIBP were significantly decreased in group D, however all patients remained haemodynamically stable.

Conclusion: Bolus dose of intravenous dexmedetomidine 0.5 µg/ kg administered just before spinal anaesthesia prolongs the duration of sensory – motor block and postoperative analgesia with arousable sedation.

Keywords: spinal anaesthesia, postoperative analgesia, dexmedetomidine

Introduction

Spinal subarachnoid block is still the first choice anaesthesia in lower abdominal and lower limb surgeries, because it blunts the "stress response" to surgeries and decreases intraoperative blood loss and the incidence of post operative thromboembolic events [1].

Different drugs such as epinephrine, phenylephrine, adenosine, magnesium sulfate, sodium bicarbonate, and neostigmine and alpha 2 agonists have been used as an adjuvants to local anaesthetics to prolong the duration of spinal anaesthesia. Among them clonidine, a alpha 2 agonist is widely used by oral, intrathecal, and intravenous routes as an adjuvant to prolong spinal anaesthesia.[2] Dexmedetomidine is a more suitable adjuvant to spinal anaesthesia compared to clonidine as it has more sedative and analgesic effects due to its more selective alpha 2A receptor agonist activity [3].

Though dexmedetomidine was initially approved by FDA for short term sedation in critical care, its unique pharmacodynamic profile has render it suitable for perioperative care during general anaesthesia or regional anaesthesia. It is increasingly used as an adjuvant to various regional techniques [4, 5]. Intravenous administered dexmedetomidine has been shown to produce analgesic effects by acting at both spinal and supra spinal levels. The analgesic effect primarily results from the inhibition of the locus ceruleus at the brain stem. In addition, dexmedetomidine infusion may result in increased activation of alpha 2 receptors at the spinal cord resulting in inhibition of nociceptive impulse transmission. The effect seems to be mediated through both pre-synaptic and the post-synaptic alpha 2 receptors [6].

Still, literature on the effect of low dose intravenously dexmedetomidine on spinal block characteristics and postoperative analgesic requirement is limited and conflicting. Thus, we had undertaken this study to evaluate the effect of low dose intravenous dexmedetomidine on spinal block characteristics and its postoperative analgesic effects in patients scheduled for lower abdominal and lower limb surgeries under spinal anaesthesia with hyperbaric bupivacaine 0.5%.

Methods

After obtaining institutional`s ethical committee`s approval and written informed consent of the patients, this randomized double blind study was conducted on sixty patients of ASA grade I and II, aged between 18 and 50 years, of either sex, who were undergone lower abdominal and lower limb elective surgeries. All the patients were considered otherwise healthy and not have any other

medical treatment. Pre-operatively patients were asked to keep nil by mouth for 6 hours. After securing intravenous line with 18-G cannula, all patients were preloaded with inj. ringer's lactate, 15 ml/kg over 10 mins. All the routine monitors were attached, and the preoperative baseline readings of noninvasive blood pressure, pulse rate (PR), and saturation were recorded.

The patients were randomly divided into two groups: Group A (*n*=30) received normal saline and Group D (*n* = 30) received 0.5 μg/kg dexmedetomidine, intravenously. The study drugs were prepared to a total volume of 20 mLby adding normal saline in a 20 mL syringe and were administered intravenously over a 10 min period as a single dose. Five minutes after completion of the infusion, the patients were placed in the sitting position and after adequate aseptic precautions, lumbar puncture was performed at L3-4 intervertebral space using a standard midline approach with a 25-G Quincke needle. After ensuring a free flow of CSF, 12.5 mg (2.5 ml) bupivacaine heavy 0.5 % was injected intrathecally. The study drug solution was prepared by anaesthesia resident who was not 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. The data were recorded by the anaesthesiologist other than who performed the block.

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. Time to 2 segment regression was recorded. Degree of motor blockade was determined according to Modified Bromage scale (Table-1). Onset of motor block was defined as a time from intrathecal injection till the patient was unable to flex ankle and foot (modified Bromage score 3). Duration of motor block recorded and was considered as time from onset of motor block to recovery of modified Bromage score 0.

Intraoperatively, blood pressure heart rate (HR) and 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 6 hours. Hypotension, defined as fall of MAP by >20% from baseline or fall in systolic pressure below 90 mmHg, was treated with intravenous fluids and incremental intravenous doses of mephentermine 6 mg. Bradycardia, defined as HR <50 bpm or a decrease by >20% from baseline and was treated with injection atropine 0.6 mg intravenously. The sedation was assessed intraoperatively and postoperatively by a modified Ramsay Sedation Scale 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 or less, was considered as effective analgesia. When VAS reaches 4 or more, diclofenac (75 mg) intravenously was given as rescue analgesic.

Time for the first request for postoperative analgesia and the number of patients who required supplemental analgesic, were recorded. Duration of effective analgesia was measured as the time from intrathecal injection to the first administration of rescue analgesic. All patients were observed postoperatively for any complain of nausea, vomiting, hypotension, bradycardia, respiratory depression, and post-spinal shivering. Intra and postoperative complications were noted and were managed accordingly. Data were collected independently and were entered in the attached patient proforma. All observations were analysed using Student t-test and Chi square test. P-value <0.05 was considered statistically significant.

Table 1: Modified Bromage scale

Grade	Criteria
0	No motor block
1	Unable to raise extended legs
2	Unable to flex knee
3	Unable to flex ankle and foot

Results

The demographic data were comparable between the two groups (Table 2). The onsets of the sensory block and motor block were significantly earlier in group D as compared to group A (Table. 3). The highest sensory level achieved up to T6 in group A as compared to group D. Time to reach highest sensory level was significantly earlier in group A (Table. 3).

Duration of motor block, two segment regression time and duration of analgesia were significantly longer in group D (Table. 3).

Sedation scores were significantly more in dexmedetomidine group as compared to group A. (Fig. 1) Both heart rate and mean arterial pressure were lower in group D as compared to group A (Fig. 2,3). However, patients in both groups remained haemodynamically stable and none of the patient required any intervention.

During study period, we observed insignificant incidence of nausea, vomiting, hypotension and bradycardia. However, both groups were comparable with regard to incidence of side effects.

Table 2: Demographic data

Parameter	Group A	Group D
Age (years)	32.8±9.86	33.96 ±9.4
Weight (kg)	56.33±4.58	57.66±3.87
Sex (M:F)	25 : 5	22 : 8
ASA Grade I : II	16 : 14	14 : 16
Duration of surgery (min)	83.90±7.13	85.57±6.96

Values are Mean ± SD or number

Table 3: Sensory and motor parameters

Parameters	Group A	Group D
Onset of sensory block (min)	3.51±0.51	2.55±0.44 *
Onset of motor block (min)	4.07±0.71	3.38±0.55 *
Highest sensory level achieved	T6	T4
Time to reach highest sensory block (min)	6.71±0.55	8.07±0.50 *
Two segment regression (min)	92.46±4.77	141.9±6.17 *
Duration of motor block (min)	147.8±12.32	191.5±4.74 *
Duration of analgesia (min)	131.1±12.93	225±11.8 *

*p <0.05 Gp A vs D

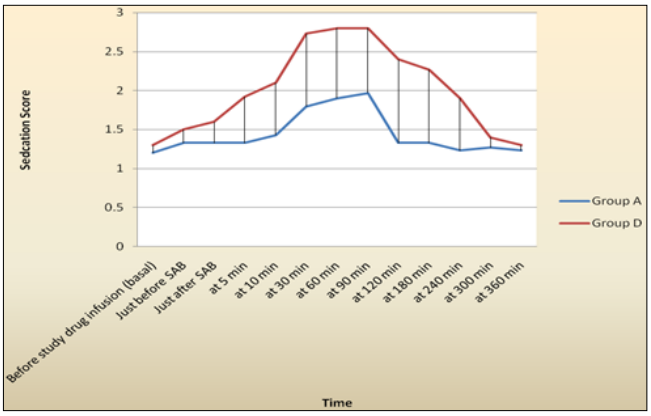

Fig 1: Sedation score

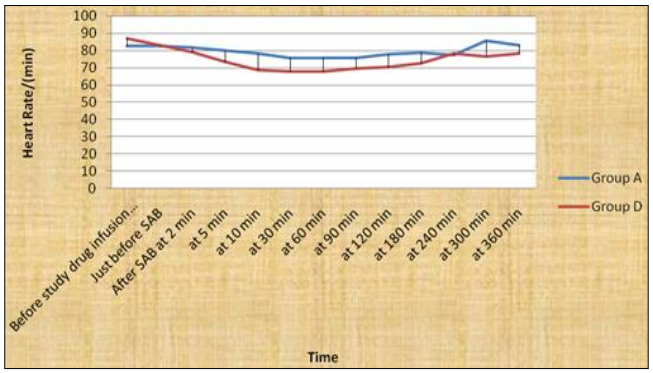

Fig 2: Changes in heart rate

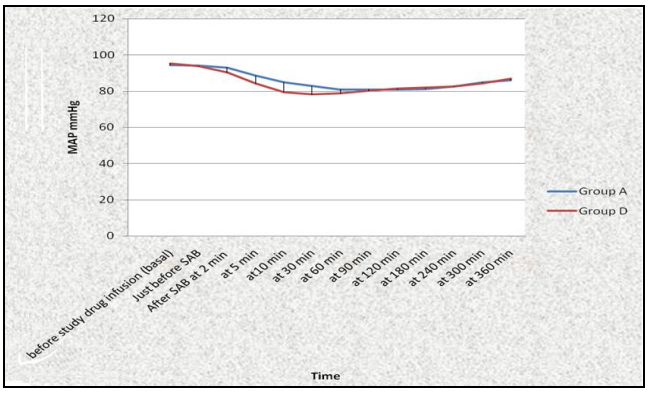

Fig 3: Changes in map

Discussion

Though dexmedetomidine was initially approved by US FDA for short term sedation in critical care, its unique pharmacodynamic profile has render it suitable for perioperative care during general anaesthesia or regional anaesthesia. Dexmedetomidine is a highly selective alfa-2 adrenergic agonist that produces analgesic and anaesthetic effects [7-9]. It is increasingly used as an adjuvant to various regional techniques.

The results of our study indicate that intravenous dexmedetomidine administered before subarachnoid block hastens the onset of sensory block and motor block and prolongs the duration of analgesia. Faster onset of sensory block may be due to alpha 2 receptor activation induced inhibition of nociceptive impulse transmission. In the present study, onset time of sensory and motor blocks were significantly earlier in patients who received intravenous dexmedetomidine. Similar results were reported by Harsoor SS *et al* [5] and Sethi *et al* [10]. However, in contrast to our results Agarwal *et al* [7], Gupta K *et al* [11] and Contracter HU *et al* [8] reported a delayed onset of sensory and motor onset in patients who received spinal anaesthesia with intravenous dexmedetomidine. In contrast to our results, Bhagwatha A *et al* [12] observed a much shorter time to reach highest sensory block, which was 4.48 ± 0.55 min in patients who received intravenous dexmedetomidine as compared to 8.07 ± 0.50 min in our study. Our results are in accordance to Kumar SR *et al* [13], Gupta K *et al* [11] and Kilick K *et al* [14] who also found a longer time to two segment regression and motor block duration in patients who received intravenous dexmedetomidine along with spinal anaesthesia.

Several studies reported prolonged duration of analgesia after spinal anaesthesia following the use of intravenous dexmedetomidine. However in majority of the studies, intravenous dexmedetomidine was used as bolus dose followed by infusion of dexmedetomidine throughout the surgery [15]. In our study, we also noticed a significantly longer duration of postoperative analgesia in patients who had IV dexmedetomidine and our results are comparable with these studies despite single bolus dose of intravenous dexmedetomidine used in our study. Similar to study of Harsoor *et al* [5] we also observed a slight decrease in HR and BP in patients receiving IV dexmedetomidine. Though majority of the studies observed bradycardia and hypotension as major side effect with iv dexmedetomidine 1 µg/ kg [16, 17]. However, incidence of bradycardia was lower in our study. This may be attributed to lower bolus dose (0.5 µg/kg) of dexmedetomidine used in present study.

As dexmedetomidine produces sedation through its central effect [18, 19] in our study, patients who received dexmedetomidine were sedated but readily arousable. None of the patient in dexmedetomidine group had sedation score more than 3 during the observation period. This was in contrary to other studies [20] who noticed excessive sedation, however they used dexmedetomidine in higher doses in comparison to lower dose of dexmedetomidine used in our study.

Conclusion

Thus we concluded that single dose intravenous dexmedetomidine 0.5 µg/kg administered just before spinal anaesthesia provides longer duration of sensori-motor block and postoperative analgesia with arousable sedation and haemodynamic stability.

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