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EFFECT OF SUPPLEMENTATION OF LOW DOSE INTRAVENOUS DEXMEDITOMIDINE AS BOLUS PLUS INFUSION ON CHARACTERISTICS OF SPINAL ANESTHESIA WITH HYPERBARIC BUPIVACAINE

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ABSTRACT **BACKGROUND:** Dexmedetomidine is non-selective alpha adrenoceptor agonist, found to exert its actions at spinal and supraspinal level. The study was done to evaluate effect of low dose intravenous dexmeditomidine on the characteristics of the spinal anaesthesia with hyperbaric bupivacaine. **OBJECTIVE:** We evaluated the onset, duration of sensory and motor blockade following supplementation of IV Dexmeditomidine during subarachnoid block. **MATERIALS AND METHODS:** The study was conducted on 60 patients undergoing lower abdominal and lower limb surgeries under spinal anesthesia. Group D received IV dexmedetomidine 0.5 mcg/kg bolus over 10 min prior to SAB, followed by an infusion of 0.5 mcg/kg/h for the duration of the surgery. Group C received similar volume of normal saline infusion. Time for the onset of sensory and motor blockade, level of analgesia and duration of analgesia were noted. Sedation scores using Ramsay Sedation Score (RSS) and hemodynamic parameters were assessed. **RESULTS:** Demographic parameters, duration and type of surgery were comparable. The onset of sensorimotor was earlier in group D (1.67±0.71 mins for sensory block and 2.40±0.50 for motor block) as compared to group C(3.73±0.94 for sensory block and 5.93±1.31 for motor block) with p<0.001.The mean time for two dermatomal regression of sensory blockade was significantly prolonged in group D(118.63±10.05 mins) compared to group C (94.5±11.6 mins)(p<0.001).Duration of sensory blockade was significantly prolonged in the group D (354.67±38.66 mins) compared to group C(226.67±20.57 mins)(p<0.001).Duration of motor blockade was significantly prolonged in group D(249.33±45.02 mins) compared to group C (179.73±14.20 mins)(p<0.001).Ramsay sedation scores were significantly high in group D (2.5±0.7) compared to group C (2.0±0.3).Intraoperatively there is significant reduction of heart rate and blood pressure compared to baseline noted in group D than group C.Bradycardia requiring atropine administration noted in 6 patients in group D compared with nonein group C.Lower systolic,diastolic and mean arterial pressures noted in groupD compared with groupC. **CONCLUSION:** Intravenous dexmeditomidine when administered during spinal anaesthesia hastens the onset of sensory block and prolongs the duration of sensory and motor block with satisfyingly arousable sedation.

KEYWORDS : Dexmedetomidine; intravenous; subarachnoid block.

INTRODUCTION

Subarachnoid block is widely used regional anesthetic technique particularly advantageous for lower abdominal and lower limb surgeries[1]. Various adjuvants have been used intravenously and intrathecally for prolonging spinal anesthesia with possible advantages of delayed onset of post op pain, delayed and reduced analgesic requirement[2]. Dexmedtomidine is a non-selective alpha adrenoceptor found to exert its action at spinal and supraspinal level.[3]

AIM OF THE STUDY

To evaluate effect of low dose intravenous dexmeditomidine on the characteristics of the spinal anesthesia with hyperbaric bupivacaine.

MATERIALS AND METHODS

This randomized prospective double blinded prospective study was conducted over a period of 6months from August 2021 to January 2022 among 60 patients aged between 18 to 55 years, ASA class I and II posted for lower limb and lower abdominal surgeries in Government General Hospital attached to Rangaraya Medical College, Kakinada. After obtaining institutional ethics committee approval and informed consent, the study population is randomly divided into two groups.

Group D:Recieved IV dexmeditomidine 0.5mcg/kg diluted to 20ml with normal saline and infused over 10 minutes as loading dose prior to sub arachnoid block followed by 0.5mcg/kg/hr infusion throughout the procedure.

Group C (control): Received similar volume of normal saline

Inclusion criteria:

A. Age 18-55 years
B. ASA Physical status I and II of either gender
C. Elective Lower abdominal and lower limb surgical procedures under spinal anesthesia.

Exclusion criteria:

A. Patient refusal
B. Infection at the puncture site
C. Coagulopathy
D. Diabetes milletus,Hypertension
E. History of psychiatric and neurological disease

PROCEDURE

The patient was shifted to operating room, the standard monitors including pulse oximetry, electrocardiogram, and noninvasive blood pressure were attached. An IV line was obtained with 18 gauge cannula and all patients were preloaded with Ringer lactate solution 10 ml/kg body weight. Group D patients received IV dexmedetomidine 0.5 mcg/kg diluted to 20 ml with normal saline and infused over 10 min as a loading dose, prior to SAB. Group C received similar volume of normal saline. Immediately after administering the initial loading dose, patients were put in lateral position and SAB was administered at L3-L4 or L4-L5 level using standard technique with 12.5 mg of hyperbaric bupivacaine. After SAB, Group D patients received dexmedetomidine maintenance infusion at the rate of 0.5 mcg/kg/h and the same rate of infusion of normal saline was administered in Group C, throughout the duration of procedure. Sensory block was assessed by loss of pin prick sensation and motor blockade was determined using Bromage Scale[4] (0 = no paralysis; 1 = unable to raise extended leg; 2 = unable to flex knee; 3 = unable to flex ankle.

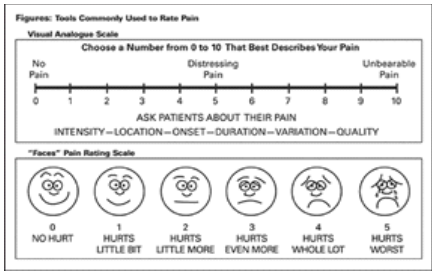
The demographic data (age, weight, ASA status, type, and duration of surgery) and the following parameters were recorded: time for the onset of sensory and motor blockade, maximum cephalad level of sensory block achieved, time for two segment regression, total duration of analgesia and motor blockade. Onset of sensory block was considered when the loss of pinprick sensation was noted at T10 and onset of motor block when complete loss of motor power was achieved (Bromage Scale 3). The degree of sensory and motor blockage was assessed every 2 minutes until the maximum level of the block was reached, and then every 5 minutes after that. Using the Ramsay Sedation Score(RSS)[5], the level of sedation was assessed throughout the procedure. Heart rate, blood pressure, and SpO2 were monitored intraoperatively every five minutes until the end of the procedure, then every fifteen minutes for the first hour following the procedure, and then every half hour for the following three hours.

In the postoperative anesthesia care unit (PACU), the visual analogue scale is used to assess pain till first rescue analgesia. Injection tramadol 100mg intravenous was used as rescue analgesic. If VAS was >3, the patients will receive supplementary IV tramadol 100 mg as rescue analgesia. The time from SAB to the first rescue analgesia with injection of tramadol was noted and considered as duration of analgesia. Patients were observed for any adverse effects

Ramsay sedation score

Table 2: Ramsay Sedation Score

Score	Clinical response
1	Anxiety and completely awake
2	Completely awake
3	Awake but drowsy
4	Asleep but responsive to verbal commands
5	Asleep but responsive to tactile stimulus
6	Asleep and not responsive to any stimulus



RESULTS

All data were statistically analysed using students unpaired t test and fischers test.

DEMOGRAPHIC DATA

PARAMETERS	GROUP D	GROUP C	P VALUE
Age(year)	41.6±4.6	39.6±8.6	0.754
Sex(M:F)	24/6	12/18	0.558
Weight(kg)	50.2±6.2	52.9±6.9	0.589
Duration of surgery	64.67±29.9	68.9±39.94	0.278

COMPARISON OF SENSORY AND MOTOR PARAMETERS IN BOTH THE GROUPS

PARAMETERS	GROUP D	GROUP C	P VALUE
Onset of sensory blockade(to T10)in mins	1.67±0.71	3.73± 0.94	0.0001
Median Highest level of sensory blockade attained	T6(T4-T8)	T8(T6-T10)	0.363
Time for attaining highest level of sensory blockade(mins)	3.40±0.89	9.03±1.40	0.0001
Two segment regression (mins)	118.63±10.05	94.50±11.6	0.0001
Duration of analgesia(mins)	354.67± 38.66	226.6±20.57	0.0001
Onset of motor blockade(modified bromage 3 in mins)	2.40±0.50	5.93 ±1.31	0.0001
Duration of motor blockade(modified bromage 0 in mins)	249.33±45.02	179.73±14.20	0.0001
Time for first rescue analgesia(mins)	423.67±37.92	288.67±29.33	0.001

The onset of sensorimotor was earlier in group D (1.67±0.71 mins for sensory block and 2.40±0.50 for motor block) as compared to group C(3.73±0.94 for sensory block and 5.93±1.31 for motor block) with p<0.001.

The mean time for two dermatomal regression of sensory blockade was significantly prolonged in group D(118.63±10.05 mins) compared to group C (94.5±11.6 mins)(p<0.001) Duration of sensory blockade was significantly prolonged in the group D (354.67±38.66 mins) compared to group C(226.67±20.57 mins)(p<0.001).

Duration of motor blockade was significantly prolonged in group D(249.33±45.02 mins) compared to group C (179.73±14.20 mins)(p<0.001)

Ramsay sedation scores were significantly high in group D (2.5±0.7) compared to group C (2.0±0.3)

Intraoperatively there is significant reduction of heart rate and blood pressure compared to baseline noted in group D than group C.Bradycardia requiring atropine administration noted in 6 patients in group D compared with none in group C.

Lower systolic,diastolic and mean arterial pressures noted in groupD compared with groupC.

DISCUSSION

IV administered dexmedetomidine produces analgesic effects by acting at both spinal and supraspinal levels. Inhibition of locus ceruleus at the brain stem and increased activation of alpha-2 receptors at the spinal cord resulting in inhibition of nociceptive impulse transmission is the cause for analgesic affects[6,7]. The effect seems to be mediated through both presynaptic and the post-synaptic alpha-2 receptors. The results of this study shows that infusion of dexmedetomidine fastens the onset of sensory block, though the onset of motor blockade was not affected.

Lugo et al.[8] in their study used 1 mcg/kg bolus followed by 0.5 mcg/kg/h infusion of dexmedetomidine noted prolongation of sensory block and duration of analgesia without significant effect on motor block. Al-Mustafa et al.[9] also observed similar findings in their study and in addition, there was prolongation of motor blockade with a similar dose of dexmedetomidine. Recently, administration of a single bolus of 1 mcg/kg,[10] and 0.5 mcg/kg,[11] also were reported to prolong the duration of analgesia and sensory blockade. Despite using a lower initial loading dose of 0.5 mcg/kg, compared with 1 mcg/kg, the duration of sensory block and analgesia in our study were comparable with above studies.

In a study done by Kaya et al.[11] a single dose of 0.5 mcg/kg of dexmedetomidine was used and no affect was noted on the duration of motor block. The prolongation of motor block observed by us inspite of use of 0.5 mcg/kg initial loading dose may be attributed to continuous infusion following loading dose.

Following dexmedetomidine infusion, the hemodynamic response depends upon the dose and speed of infusion.Higher dose and rapid infusion of dexmedetomidine results in a sequence of transient hypertension with reflex bradycardia, followed by hypotension. The subsequent decrease in heart rate and blood pressure may be due to decrease in central sympathetic outflow[12,13].

Most of the studies noted bradycardia as prominent side effect varying 30-40%Subsequent decrease in the heart rate and blood pressure in present study was minimal. Incidence of hypotension in present study was comparable to other studies(kaya et al. Used single intravenous dexmed dose of 0.5mcg/kg)[11].

Sedation with easy arousability noted in this study contrary to other studies(al-mustafa et al.,Hong et al. Higher dose of dexmed is used & excessive sedation noted in 3/25 and 2/26 patients in their study) No respiratory depression and desaturation noted in the present study compared to other studies (hong et al. Noted desaturation in 2 patients probably due to old age).

CONCLUSION

Low dose IV dexmeditomidine(0.5mcg/kg bolus followed by infusion of 0.5mcg/kg/hr) significantly fastens the onset of sensory and motor blockade and prolongs the duration of block of bupivacaine spinal anesthesia. The hemodynamic changes induced by it are transient and are responsive to pharmacological agents and iv fluid administration. It is effective in providing satisfactory arousable sedation without causing respiratory depression intraoperatively.

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