ORIGINAL RESEARCH PAPER
Volume-8 | Issue-11 | November - 2019 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

STUDY OF THE EFFECTS OF INTRAVENOUSDEXMEDETOMIDINE ON SPINAL ANAESTHESIA ANDANALGESIA IN PATIENTS UNDERGOING LOWER ABDOMINAL SURGERIES AT NMC, SASARAM, BIHAR

Surgery

Dr. Alok Kumar* M.B.B.S., M.D. (Anaesthesia), Assistant Professor, Department of Anaesthesia, Narayan Medical College, Jamuhar, Sasaram, Bihar. *Corresponding Author

Dr. Prof. Subrato Naag Professor and Head of Department, Department of Anaesthesia, Narayan Medical College, Jamuhar, Sasaram, Bihar.

Dr. Debarshi Jana Young Scientist (DST) Department of Gynecology and Obstetrics Institute of Post-Graduate Medical Education and Research, A.J.C. Bose Road, Kolkata-700020, West Bengal, India

ABSTRACT

BACKGROUND AND OBJECTIVE : Spinal anaesthesia is a well known technique used in lower abdominalsurgeries. Dexmedetomidine is a highly selective and potent alpha 2 agonist. Thepresent study was aimed to assess the effects of intravenous dexmedetomidinepremedication on spinal anaesthesia and analgesia with 0.5% HyperbaricBupivacaine.

METHODS : This one year double blind randomized placebo controlled trial wasconducted in the Department of Anaesthesia, Narayan Medical College, Jamuhar,Sasaram,Bihar. A total of 60 patients undergoing lower abdominalsurgeries were allocated into two groups namely, Group D (n=30; patientsreceived dexmedetomidine 0.1 mL/Kg [0.5 µg/kg] over 15 minutes using infusionpump 20 minutes prior to SAB) or Group P (n=30; Patients received 0.1 mL/kgnormal saline over 15 minutes using infusion pump 20 minutes prior to SAB).

RESULTS : In this study, male to female ratio was 0.46:1. Both the groups werecomparable with respect to sex, age, height and weight. No biphasic change inheart rate or mean arterial pressure or significant cardiovascular variability wasobserved after administration of dexmedetomidine. Arterial oxygen saturation inboth groups remained above 97% throughout the study and was comparable inboth groups. The requirement of Atropine and Ephedrine was found to becomparable in both groups. Patients in dexmedetomidine group had asignificantly higher level of highest sensory block level (T4.8±1.52). MotorBlock duration was significantly prolonged in group D (128.00±20.07). SensoryBlock duration in dexmedetomidine group (165.67±19.77) was significantlylonger.

CONCLUSION AND INTERPRETATION : Overall, based on the findings of this study it may be concluded that,single dose of intravenous dexmedetomidine given as premedication, prolongsthe duration of sensory and motor blockade of bupivacaine-induced spinalanaesthesia. It also provides conscious sedation and additional analgesia,maintaining haemodynamic stability.

KEYWORDS

Dexmedetomidine; Hyperbaric Bupivacaine; Motor Block; Sedation;sensory Block; Spinal Anaesthesia.

INTRODUCTION

Spinal anaesthesia remains a popular technique used for surgery to theabdomen, pelvis and lower limbs providing fast onset and effective sensory andmotor blockade. The advantages of spinal anaesthesia have been well establishedand widely accepted.

Current usage of this technique is waning in the developed world, withepidural analgesia or combined spinal-epidural anaesthesia emerging as thetechniques of choice where the cost of the disposable 'kit' is not an issue.

However spinal analgesia is the mainstay of anaesthesia in countries likeIndia, Pakistan and parts of Africa, excluding the major centres. At a low cost, asurgery of up to two hours duration can be performed below the umbilicus of thepatient.

The greatest challenge of the technique is to control the spread of the localanaesthetic through the cerebrospinal fluid (CSF) in order to produce a block thatis adequate for the proposed surgery without producing a needless extensivespread.

Administration of a spinal anaesthetic to higher levels may affect theability to breathe by paralysing the intercostal respiratory muscles, or even thediaphragm in extreme cases (called a "high spinal", or a "total spinal", with whichconsciousness is lost), as well as the body's ability to control the heart rate via thecardiac accelerator fibres. Also, injection of spinal anaesthesia higher than thelevel of L1 can cause damage to the spinal cord, and is therefore usually notdone.

Subarachnoid block is a safe, inexpensive and 'easy-to-adminis ter'technique which also offers a high level of post–anaesthesia satisfaction forpatients. The risk of general anaesthesia including mishaps due to airwaymanagement are avoided in patients having an irritable airway (bronchial asthmaor allergic bronchitis), anatomical abnormalities which make endotrachealintubation very difficult (micrognathia), in borderline hypertensives whereadministration of general anaesthesia or endotracheal intubation can furtherelevate the blood pressure, and procedures performed in geriatric patients.

Bupivacaine, an anilide compound, is the most widely used drug forspinal anaesthesia currently. Different agents, like epinephrine, phenylephrine,adenosine, magnesium sulfate and clonidine, have been used as adjuncts to localanaesthesia for prolonging the duration of spinal analgesia via the intrathecalroute.

Alpha 2 Agonists produce diverse responses, including analgesia, anxiolysis, sedation, and sympatholysis, each of which has been reported in thetreatment of surgical and chronic pain patients and in panic disorders as well.

Recently, the Food and Drug Administration registered two novel alpha 2-adrenergic agonists Clonidine and Dexmedetomidine.

Dexmedetomidine is a highly selective and potent alpha 2 agonist (1620 :1 alpha 2 : alpha 1), and is seven to ten times more selective for alpha 2 receptorscompared to Clonidine, and has a half life of 2 to 3 hours.

Dexmedetomine is known to induce sedation, decrease anaesthetic drugrequirement and improve perioperative haemodynamics by attenuating bloodpressure and heart rate responses to surgical stimulation, and protection againstperioperative myocardial ischaemia. It provides sympathoadrenal stability andsuppresses renin angiotensin activity.

Supplementation of bupivacaine spinal block with a low dose ofintrathecalDexmedetomidine produces a significantly shorter onset of motorblock and a significantly longer sensory and motor block than bupivacaine alonewithout any significant hemodynamic instability or sedation.

Although a synergistic interaction between intrathecalDe xmedetomidineand local anesthetics has been observed in previous studies,7 there is further needfor study on the effects of intravenous

Dexmedetomidine premedication on theduration of sensory and motor block, and analgesia during spinal anaesthesia.

Hence the present study was conducted to assess the effects ofintravenous Dexmedetomidine on spinal anaesthesia and analgesia in patientsundergoing lower abdominal surgeries.

OBJECTIVES

The objectives of the study were to assess the effects of intravenousDexmedetomidine on spinal anaesthesia and analgesia in patients undergoinglower abdominal surgeries under spinal anaesthesia with 0.5% HyperbaricBupivacaine.

The effects of intravenous Dexmedetomidine on spinal anaesthesia andanalgesia were assessed through the following variables.

- Haemodynamic variables
- Duration of sensory blockade.
- Duration of motor blockade.
- Highest level of sensory block achieved
- Time to first request for rescue analgesia.
- Sedation

MATERIALAND METHODS

The present study was conducted in the Department of Anaesthesia,Narayan Medical College, Jamuhar,Sasaram,Bihar.

STUDY DESIGN

A one year double blind randomized placebo controlled trial.

SOURCE OF DATA

Patients undergoing lower abdominal surgeries under spinal anaesthesia atNMC,Sasaram,Bihar.

SAMPLE SIZE

A total of 60 patients divided into two groups using computerr andomization.

SAMPLING PROCEDURE

Based on the results of previous study and standard statistical formula,time for sensory two dermatome regression for the two drugs was taken todetermine the sample size.

The values were 261 ± 35 minutes for Dexmedetomidine and 165 ± 31minutes for control group.

The formula used was;

$$n = \frac{(2\alpha + 2\beta)^2\ 2s^2}{(x_1 - x_2)^2}$$

Level of significance is taken as 5%
Power of the test used is taken as 80%
Hence $Z\alpha = 1.96$ and $Z\beta = 0.84$
$2s^2=(35)^2+(31)^2$, $x_1 = 261$, $x_2 = 165$
With these values, the minimum sample size obtained was 9.
However, for the sake of consistent results 'n' was taken as 30 that is, atotal of 60 patients divided equally into two groups namely group D and P byusing computerised randomization.

SELECTION CRITERIA

INCLUSION

- Patients undergoing lower abdominal surgeries lasting 60 to 120 minutes.
- Patients with
- Age between 20 to 60 years.
- Height between 140 to 165 cms.
- Weight between 40-60 kgs.
- Patients with ASA Grade I and II.

EXCLUSION

- Patient refusal.
- Known allergy to Bupivacaine.
- History of bleeding diathesis.
- Infection at the site of spinal needle insertion.
- Severe spinal abnormalities like spina bifida, meningocele.
- On treatment with alpha adrenoreceptor antagonists. use of any opioid or sedative medications in the week prior to surgery, a history of alcohol or drug abuse
- Pre-existing neurological deficits in the lower extremities, and cardiovascular, respiratory, neurological, psychological, hepatic, or renal disease.

METHOD OF COLLECTION OF DATA

After the enrollment, demographic data such as age and sex were recordedand patients were asked for the history. General physical examination, systemicexamination was carried out. Routine investigations such as haemogram, bloodgroup, bleeding time, clotting time, prothrombin time, activated partialthromboplastin time, International Normalized Ratio (if the patient was onanticoagulant therapy), electrocardiogram, Chest X-ray if required were done andthe data was recorded.

RANDOMIZATION

Patients were randomly allocated into one of the two groups by computergenerated randomization that is

- Group D (n=30) Patients received Dexmedetomidine 0.5 µg/kg over 15minutes using infusion pump 20 minutes prior to SAB.
- Group P (n=30) Patients received 0.1 mL/kg normal saline over 15minutes using infusion pump 20 minutes prior to SAB.

PROCEDURE

Status of nil by mouth (NBM) was confirmed. Patient was taken in preanaesthetic room. Monitors like, ECG, NIBP, Pulse oximetry were applied. TwoIV lines were secured with appropriate IV canula and 500 ml of crystalloids werestarted in one line. The other line was used for study drug infusion. Heart rate,MAP, SpO2 using pulse oximeter was monitored before, during and after thesurgery.

The study drug in the prefilled coded 20 ml syringe was started 20minutes before administering spinal block, using infusion pump at the rate of 0.1mL/kg body weight over 15 minutes as a single dose. Dexmedetomidine wasprepared in a dilution of 5µg/ml of NS. The code number of the study drugsyringe was noted down in the proforma. Dexmedetomidine was given in a doseof 0.5 µg/kg.

Five minutes after the end of the infusion, under strict aseptic precautionsa 23G Quinkes spinal needle was inserted in L3-L4 intervertebral space. Afterconfirming free flow of CSF, 3 ml of 0.5% Hyperbaric Bupivacaine was injectedinto the subarachnoid space. Patients received Oxygen 4 L/min.

STUDY VARIABLES

SENSORY BLOCKADE

Sensory blockade was assessed using pinprick in the mid-axillary linebeginning at the time of spinal block and at an interval of 5 minutes each upto 10minutes and thereafter at an interval of 10 minutes each till two dermatomeregression was achieved. Recovery time for sensory blockade was defined astwo-dermatome regression of anesthesia from the maximum level. When sensorylevels of anesthesia were not equal bilaterally, the higher level was used for thestatistical analysis.

MOTOR BLOCK

Motor block was assessed immediately after sensory block assessmentusing a Modified Bromage Scale59 as below.

- Bromage 0 - Patient is able to move the hip, knee and ankle.
- Bromage 1 - Patient is unable to move the hip, but is able to move theknee and ankle.
- Bromage 2 - Patient is unable to move the hip and knee, but is able tomove the ankle.
- Bromage 3 - Patient is unable to move the hip, knee and ankle.

Motor block duration was taken as the time for return to ModifiedBromage Scale 2.

Sensory and motor block were assessed beginning at the time of spinalblock and at an interval of 5 minutes each upto 10 minutes and thereafter at aninterval of 10 minutes each untill modified bromage scale 2 was achieved. Thehighest sensory block level and recovery time of both sensory and motor blockwere recorded. All durations were calculated considering the time of spinalinjection as time zero.

SEDATION SCORE

The Ramsay sedation score60 was used to evaluate sedation.

- 1 = anxious and agitated.

- 2 = Cooperative and tranquil.
- 3 = Drowsy but responsive to command.
- 4 = Asleep but responsive to a glabellar tap.
- 5 = Asleep with a sluggish response to tactile stimulation.
- 6 = Asleep and no response.

Excessive sedation was defined as a score greater than 4/6. These scorewere assessed at 10, 30, 50, 70, 90, 110 and 120 minutes duration.
Haemodynamic parameters

Intraoperatively HR, BP and SpO_2 were measured and noted. The vitalswere monitored every 15 mins till the end of the surgery.

Hypotension was defined as a decrease in systolic blood pressure by 30%from baseline, or a systolic blood pressure lower than 90 mm Hg and was treatedwith incremental doses of intravenous ephedrine (6 mg) and a bolusadministration of 250 ml lactated Ringer's solution over 10 minutes.

Bradycardia was defined as HR < 50 beats/min, and was treated with 0.6mg of intravenous atropine.

TIME TO REQUEST FOR RESCUE ANALGESIA

In the post anaesthesia care unit, time to request for rescue analgesia wasnoted. Postoperative pain score was measured by using VAS of 'zero' to 'ten'where 'zero' indicated no pain and 'ten' indicated worst imaginable pain. Rescueanalgesia of injection tramadol 50 mg IV was given if the VAS score was morethan three.

BLINDING

The blinding was done by preparing the study drug (Dexmede tomidine)and the control drug (Normal Saline) in prefilled and coded identical 20 mlsyringes as per the randomization protocol, in dilutions of normal saline 20 mland Dexmedetomidine 20 ml (5 mcg/ml).

All prefilling, coding and decoding was done in the Department ofClinical Pharmacy. The investigators involved in the study did not know aboutthe content of the syringes. The doctor who performed the spinal analgesia wasblinded as to which group the patient was allocated. The anesthesiologist whoperformed the block recorded and monitored the baseline values of vital signs.

Patients were explained only about the study, but did not know what drug wasused. The study drug in prefilled and coded syringes was obtained from theClinical Pharmacy on the day of the surgery. At the end of the study, decodingwas done by the pharmacist and accordingly the cases were divided into Group D(study group) and P (placebo group) and results were analysed.

STATISTICALANALYSIS

Data obtained was coded and entered into Microsoft excel spreadsheet.The categorical data was expressed in terms of rates, ratios and percentage andcontinuous data was expressed as mean ± standard deviation (SD). The data wasanalysed by chi-square test, test of proportion, student's unpaired 't' test andMann Whitney test. For A probability value (p value) of less than or equal to0.05 was considered as statistically significant.

RESULTS

Spinal anaesthesia is a well known technique used in lower abdominal,urological and lower limb surgeries. Dexmedetomidine is a highly selective andpotent α_2 agonist (1620 : 1 α2:α1) and has a half life of 2 to 3 hours. The presentstudy was aimed to assess the effects of intravenous Dexmedetomidinepremedication on spinal anaesthesia and analgesia in patients undergoing lowerabdominal surgeries with 0.5% Hyperbaric Bupivacaine.

A total of 60 patients undergoing lower abdominalsurgeries under spinal anaesthesia were randomly allocated into one of the twogroups by computer generated randomization that is, Group D (n=30; patientsreceived Dexmedetomidine 0.1 mL/Kg [0.5 μg/kg] over 15 minutes usinginfusion pump 20 minutes prior to SAB) or Group P (n=30; Patients received 0.1mL/kg normal saline over 15 minutes using infusion pump 20 minutes prior toSAB).

In this study out of 60 patients studied, 19 (31.6%) were males and 41(68.4%) were females with male to female ratio of 0.46:1. Overall the mean agewas 37.06±10.03 years and mean weight was 52.7±6.56 kgs. Both the groupswere comparable with respect to sex, age, height and weight. No biphasic changein heart rate or mean arterial pressure or significant cardiovascular variability wasobserved after administration of Dexmedetomidine. Arterial oxygen saturation inboth groups remained above 97% throughout the study and was comparable inboth groups. The requirement of Atropine and Ephedrine was found to becomparable in both groups. Patients in Dexmedetomidine group had asignificantly higher level of highest sensory block level achieved (T4.8±1.52) ascompared to placebo group (T5.8±0.96). Motor Block duration was significantlyprolonged in group D (128.00±20.07) compared to group P (79.00±11.55).

Sensory Block duration in Dexmedetomidine group (165.67±19.77) wassignificantly longer than placebo group (132.33±9.35). Time to first request forpost operative analgesia was significantly longer in group D (270.17±41.57)when compared to group P (155.73±23.39). Excessive sedation (Ramsay sedationscore > 4) was observed in only one patient in Dexmedetomidine group. Evenwith excessive sedation (score of more than 4), the oxygen saturation remainedcomparable to the placebo group.

Overall, based on the findings of this study it may be concluded that,single dose of intravenous Dexmedetomidine given as premedication, prolongsthe duration of sensory and motor blockade of bupivacaine-induced spinalanaesthesia. Further, it prolongs the time to first request for analgesia andprovides conscious sedation without ventilatory depression and maintains goodhemodynamic stability.

CONCLUSION

The results of the present study shows that, a single dose of intravenousDexmedetomidine given as premedication, prolonged the duration of sensory andmotor blockade of bupivacaine-induced spinal anaesthesia. It provided consciousssedation without respiratory depression and maintained good haemodynamicstability. It also prolonged the time to first request for analgesia.

REFERENCES

1. Al-Mustafa MM, Badran IZ, Abu-Ali HM, Al-Barazangi BA, Massad IM,Al-Ghanem SM. Intravenous dexmedetomidine prolongs bupivacainespinal analgesia. Middle East J Anesthesiol. 2009;20(2):225-31.
2. Al-Mustafa MM, Abu-Halaweh SA, Aloweidi AS, Murshidi MM,Ammari BA, Awwad ZM, et al. Effect of dexmedetomidine added tospinal bupivacaine for urological procedures. Saudi Med J2009;30(3):365-70.
3. Kaya FN, Yavascaoglu B, Turker G, Yildirim A, Gurbet A, Mogol EB, etal. Intravenous dexmedetomidine, but not midazolam, prolongsbupivacaine spinal anesthesia. Can J Anaesth 2010;57(1):39-45.
4. Elcicek K, Tekin M, Kati I. The effects of intravenous dexmedetomidineon spinal hyperbaric ropivacaine anesthesia. J Anesth 2010;24(4):544-8.
5. Eid HEA, Shafie MA, Youssef H. Dose-Related Prolongation ofHyperbaric Bupivacaine Spinal Anesthesia by Dexmedetomidine. AinShams Journal of Anesthesiology 2011;4(2):83-95.
6. Hong JY, Kim WO, Yoon Y, Choi Y, Kim SH, Kil HK. Effects ofintravenous dexmedetomidine on low-dose bupivacaine spinal anaesthesiain elderly patients. ActaAnaesthesiol Scand. 2012;56(3):382-7.
7. Ugur F, Gulcu N, Boyaci A. Intrathecal infusion therapy withdexmedetomidine-supplemented morphine in cancer pain. ActaAnaesthesiolScand 2007;51:388.
8. Rutkowska K, Knapik P, Misiolek H. The effect of dexmedetomidinesedation on brachial plexus block in patients with end-stage renal disease.Eur J Anaesthesiol. 2009;26:851-5.