ORIGINAL RESEARCH PAPER

Anesthesiology

EFFECT OF INTRAVENOUS DEXMEDETOMIDINE ON BUPIVACAINE FOR SPINAL ANAESTHESIA

KEY WORDS:

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INTRODUCTION

Quinckie in 1891 demonstrated a safe,predictable means of performing lumbar puncture.August bier used quinckie'"s technique to inject cocaine in order to produce operative anaesthesia,the first real spinal anaesthesia.The first phase in the history of spinal anaesthesia from 1899 to 1905,was characterised by use of only cocaine for spinal anaesthesia.1 Bupivacaine ,a pipecoloxylidide derivative synthesised in 1957 by Ekenstam and introduced in clinical practice in 1963 is widely used now.It is a racemic mixture of D and L isomers and is relatively more cardiotoxic compared to other local anaesthetics.

Spinal anaesthesia ,however,has certain limitations like discomfort caused by the procedure itself,limited duration of block and a patient who is wide awake and restless. Vasoconstrictors like Phenylephrine, Opioids, Neostigmine and clonidine are some of the well known agents used to prolong the duration of block ;whereas hypnotic and sedative drugs are required to reduce the discomfort.

An alternative is the alpha adrenergic agonist,Clonidine which has excellent sedative and analgesic properties without respiratory depression is widely used in intrathecal, oral and intravenous routes to prolong duration of spinal analgesia.

Dexmedetomidine,also an alpha 2 agonist,has eight ties more affinity for alpha 2 receptors than does clonidine and has been used for premedication and as an adjunct to general anaesthesia.Intravenous Dexmedetomidine decreases the inhalational anaesthesia and opioid requirements during general anaesthesia.It produces sedation and anxiolysis by binding to alpha 2 receptors in the locus ceruleus,which diminishes the release of norepinephrine and inhibits sympathetic activity.

Various studies show that intravenous Dexmedetomidine prolonged the sensory and motor blockade of Bupivacaine spinal anaesthesia with good sedation and hemodynamic stability.The present study is designed to study the effects of intravenous Dexmedetomidine with a loading dose of 1 mcg/kg and a maintainance dose of 0.5 mcg/kg on bupivacaine spinal anaesthesia with respect to duration of sensory and motor blockade,level of sedation and hemodynamic stability.

AIMS AND OBJECTIVES

To evaluate the effects of intravenous Dexmedetomidine on Bupivacaine for spinal anaesthesia with respect to:

- Maximum spinal level achieved
- Time for 2 segment regression
- Duration of sensory and motor blockade
- Hemodynamic effects
- Duration of analgesia
- Level of sedation

MATERIALS AND METHODS

The study was conducted at MGM medical college between December 2011-May 2013. After obtaining ethical committee approval,60 ASA I- II patients undergoing elective lower limb orthopaedic surgeries were randomly allotted into two groups

Study design : A Prospective randomized study
Sample size : 60 patients were selected and randomly divided into two groups C and D.

Group C (n=30) received normal saline in same calculated volume of loading and maintainance dose as in group D

Group D (n = 30) received a loading dose of of 1 mcg/kg of Dexmedetomidine over 10 minutes and a maintainance dose of 0.5 mcg/kg/hr till end of surgery intravenously by intravenous infusion pump

INCLUSION CRITERIA:

- ASA grade I & II status.
- 20-60 years of age.
- Patients giving informed written consent.
- Patients scheduled to undergoing elective lower limb and lower abdominal surgery

EXCLUSION CRITERIA

- Age < 20 years and > 60 years
- ASA class > II
- Patients refusal
- Patients with hypersensitivity to local anaesthetic
- Any contraindication to spinal anaesthesia
- Patients taking alpha-2 adrenergic agonists, tricyclic antidepressants, any
- antipsychotic drugs, antiarrhythmics, betablockers, anticoagulants and opioids

DRUG SOLUTION USED AND DOSAGE

Dexmedetomidine was prepared in a 50 cc syringe using a Dexmedetomidine ampoule containing 100mcg/ml diluted with normal saline to a concentration of 4 mcg/ml.

Bupivacaine 0.5% ampoule was used,3.0 ml of it was taken in 5 ml syrine and administered intrathecally at rate of 1 ml over 3-4 seconds

All patients were prehydrated with 500 ml of ringer lactate solution via 18 G iv cannula in the dorsum of hand.

MONITORING

Baseline heart rate,systolic blood pressure,diastolic blood pressure,respiratory rate, sedation score were measured and recorded.All standard monitors were attached.

Hyperbaric Bupivacaine 0.5%,15 mg was injected intrathecally at L3-L4 subarachnoid space in sitting position using 25G spinal needle.

Patients allocated to group D received a loading dose of 1mcg/kg Dexmedetomidine over 10 minutes and a maintainance dose of 5mcg/kg till end of surgery.

Patients of group C received normal saline in same calculated volume as loading and maintainance dose in group D.

SENSORY BLOCKADE

The sensory blockade was tested by pin prick method immediately after making patient supine.The extent of sensory blockade was assessed at 2 minute interval and highest sensory blockade achieved was noted.After that at every 15 minute interval,level of sensory blockade was noted.The time taken for 2 segment regression and time for regression to S1 segment was noted.

MOTOR BLOCKADE

The motor blockade was assessed by Bromage scale at 2 minutes interval until complete motor blockade is achieved.

MODIFIED BROMAGE SCALE

1 - Complete block(unable to move feet or knees)
2 - Almost complete block(able to move feet only)
3 - Partial block(just able to move knees)
4- Detectable weakness of hip flexion
5- No detectable weakness of hip flexion while supine(full flexion of knees)
6- Able to perform partial knee bend

ASSESSMENT OF PAIN:

Pain was evaluated using Visual Analogue Scale.
0-1 Excellent
2-4 Good
5-6 Fair
7-8 Poor
9-10 No relief
Inj. Paracetamol 20gm IV was administered as a rescue analgesic when the pain score crossed a score of 3.

Duration of Analgesia:

It is the period from the time of subarachnoid block to the time when the patient needs the first dose of rescue analgesic drug.

The patient was discharged from PACU after sensory regression to S1 segment and Bromage scale 0.

DISCUSSION

Different drugs like epinephrine, phenylepinephrine, adenosine, magnesium sulphate and alpha2 agonists like clonidine,dexmedetomidine have been used as adjuvants to local anaesthetics to prolong the duration of spinal anaesthesia.Recent studies have shown the efficacy of both intrathecal and intravenous dexmedetomidine in prolonging spinal anaesthesia.

Intravenous dexmedetomidine has been shown to produce analgesic effects by acting at both spinal and supraspinal levels.

The present study was conducted in Dept of Anaesthesiology, MGM Medical college,Kamothe,Navi Mumbai from December 2011 to May 2013.

A total of 60 patients were selected and randomly divided into two groups C and D.

Group C (n=30) received normal saline in same calculated volume of loading and maintainance dose as in group D

Group D (n = 30) received a loading dose of of 1 mcg/kg of Dexmedetomidine over 10 minutes and a maintainance dose of 0.5 mcg/kg/hr till end of surgery intravenously by intravenous infusion pump

Dexmedetomidine infusion used as a loading dose followed by an infusion has been found to prolong the duration of sensory blockade.In our study maximum level of sensory blockade achieved was similar in both groups.Level T8 was achieved by maximum number of patients 14/30 in study group and 13/30 in control group.

In our study mean time for two segment regression of sensory blockade was significantly prolonged in dexmedetomidine group 111±16.31 mins as compared to control group 55.83±10.67minutes(p value<0.0001).This was comparable to study done by Harsoor et al where they observed that mean time to two segment regression was 111.52±30.9 in dexmedetomidine group and 53.6±18.22 in the control group with a p value <0.0001.

The total duration of sensory blockade was significantly prolonged in dexmedetomidine group 263±20.74 minutes as compared to control group 174.23±19.17 minutes(p value<0.0001).Significant prolongation were found in other studies like Al Mustafa et al-261.5±34.8 mins vs 165.2±31.5 mins(p value<0.05),Whizar-Lugo et al -208±43.5 mins vs 137±121.9 mins.

Thus in our study intravenous Dexmedetomidine infusion prolonged mean time for two segment regression of sensory blockade as well as total duration of sensory blockade .

In the present study ,time taken for motor blockade to reach modified Bromage scale 0 was significantly prolonged in dexmedetomidine group 199.73±18.52 mins as compared to control group 141.27±14.96 mins(p value<0.001).Several studies indicated prolonged duration of motor blockade like in studies by Al Mustafa et al-199±42.8 mins vs 138.4±31.3 mis(p value<0.05),Tekin et al-215mins vs190.8 mins(p value<0.001)

The incidence of bradycardia in our study was 23.33% in dexmedetomidine group.Higher incidence of bradycardia in dexmedetomidine group 16.66% compared to control group 8.3%(p value 0.46) was reported by Al Mustafa et al,Whizar-Lugo et al ,32% in dexmedetomidine group compared to 20% control group.

Dexmedetomidine inhibits release of Substance P from dorsal horn of spinal cord ,leading to primary analgesic effects. Dexmedetomidine was found to be effective in providing post operative analgesia in the present study.The time for first request for post operative analgesic was significantly prolonged in dexmedetomidine group 219.97±19.22 mins as compared to control group 153±15.52 mins(p value<0.001)

In our study intraoperative Ramsay Sedation scores were significantly higher in dexmedetomidine group with mean of 3.57±0.82 as compared to control group with mean of 2(p value <0.001).

CONCLUSION

Intravenous dexmedetomidine supplementation significantly prolongs duration of sensory blockade, analgesia and motor blockade after bupivacaine spinal anaesthesia. Dexmedetomidine causes decrease in heart rate and blood pressure with statistically significant hypotension and bradycardia. Further ,IV dexmedetomidine supplementation during spinal anaesthesia produces satisfactory arousable sedation without causing respiratory depression.

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