(e) ISSN Online: 2321-9599

(p) ISSN Print: 2348-6805

SJIF (Impact factor) 2017= 6.261

Index Copernicus value = 80.90

ORIGINAL ARTICLE

To compare the effects of 1% chloroprocaine and 1% chloroprocaine with clonidine during spinal anaesthesia procedures

[1]Manish Kumar, [2]Himanshu Yadav

[1]Assistant Professor, Department of General Surgery, Major S D Singh Medical College & Hospital, Farukkhabad, Uttar Pradesh, India;
[2]Assistant Professor, Department of Anesthesia, Major S D Singh Medical College & Hospital, Farukkhabad, Uttar Pradesh, India

ABSTRACT:

Aim: To compare the effects of 1% chloroprocaine and 1% chloroprocaine with clonidine during spinal anaesthesia procedures. **Material and methods:** 100 total ASA Patients in grades I or II who are between the ages of 18 and 55, of either sex, and who weigh between 40 and 65 kg who are scheduled for elective infraumbilical procedures lasting less than 60 minutes. Patients having a history of local anaesthetic allergy or intolerance, refusal, uncooperation, infection at the site, coagulopathy or bleeding diathesis, cardiac, neurological, hepatic, or renal illness, pregnancy, or lactation were excluded from the research. Group C (n=50): 30 mg of 1% Chloroprocaine with 0.2 ml Normal saline. Group CC (n =50): 30 mg of 1% Chloroprocaine with 30 mcg Clonidine (0.2 ml). **Results:** With the addition of clonidine (GroupCC), the onset of sensory and motor block occurred more quickly (P<0.05). In group CC, more patients (64% vs. 32%; P <0.05) achieved the T6 T-9 level block than in group C. In comparison to group C, group CC showed longer durations of motor block, quicker time to mobilise, and post-operative analgesia. In both group C and group CC, there were no significant variations in PR and all three blood pressure measures (P>0.05). **Conclusion:** In short-duration nursery procedures, the intrathecal addition of preservative-free Clonidine (30mcg) to preservative-free 1% Chloroprocaine produces good spinal anaesthesia with extended analgesic duration and hemodynamic stability.

Corresponding Author: Himanshu Yadav, Assistant Professor, Department of Anesthesia, Major S D Singh Medical College & Hospital, Farukkhabad, Uttar Pradesh, India

This article may be cited as: Kumar M, Yadav H. To compare the effects of 1% chloroprocaine and 1% chloroprocaine with clonidine during spinal anaesthesia procedures. J Adv Med Dent Scie Res 2017;5(11):163-166.

INTRODUCTION

One of the most often utilised anaesthetic techniques for surgery on the lower abdomen and lower limbs is spinal anaesthesia. However, the use of Bupivacaine is limited in short-duration surgical procedures due to prolonged motor blockade, risk of urinary retention, and severe pain after block regression with Lidocaine. As a result, the choice of local anaesthetic for spinal anaesthesia is crucial for ambulatory surgery[1,2]. The use of day surgery, where patients are admitted, operated on, and then discharged the same day, with excellent patient satisfaction, a shorter hospital stay, less financial burden, and minimal psychological disruption for the patient and family, has greatly expanded thanks to advancements in surgery, anaesthesia, and pain management. Chloroprocaine meets the criteria for providing spinal anaesthesia for a brief period of time. Chloroprocaine was initially made available for use in spinal anaesthesia in 1952. Multiple instances of neurological deficiency in individuals who accidentally received large doses of intrathecal chloroprocaine during epidural labour analgesia were later published in the literature when sodium bisulfite was added as an antioxidant[3]. Low pH and the antioxidant sodium bisulfite were thought to be the cause of these ongoing neurologic deficits. (Wang et al., 2004) Chloroprocaine without antioxidants and preservatives has recently become more popular for intrathecal usage during quick surgical procedures[5]. In addition to other local anaesthetic medications, intrathecal clonidine has been used as an adjuvant to improve sensory blockage, extended analgesia, antiemesis, and anxiolysis compared to local anaesthetic alone[6]. Clonidine does not cause pruritis or respiratory depression, in contrast to opioids. We searched the literature but were unable to locate many studies on the use of 1% chloroprocaine with clonidine for spinal anaesthesia. In order to examine the effectiveness, duration, and safety profile of 1% Chloroprocaine alone and 1% Chloroprocaine with Clonidine in short-term surgical operations to be performed under spinal anaesthetic in the Indian population, we thus designed this research.

MATERIAL AND METHODS

100 total ASA Patients in grades I or II who are between the ages of 18 and 55, of either sex, and who weigh between 40 and 65 kg who are scheduled for elective infraumbilical procedures lasting less than 60 minutes. Patients having a history of local anaesthetic allergy or intolerance, refusal, uncooperation, infection at the site, coagulopathy or bleeding diathesis, cardiac,

neurological, hepatic, or renal illness, pregnancy, or lactation were excluded from the research.

One day before to surgery, all patients had thorough general, physical, and systemic examinations. According to hospital procedure, all necessary regular and specialised tests were performed, including complete blood count, random blood sugar, blood urea, serum creatinine, ECG, and chest x-ray.

All selected patients were randomly divided into two groups (n=50 each) by envelope method as below:

GROUP C (n=50): 30 mg of 1% Chloroprocaine with 0.2 ml Normal saline.

GROUP CC (n =50): 30 mg of 1% Chloroprocaine with 30 mcg Clonidine (0.2 ml).

Prior to the operation, all patients were kept off all food and drink for at least 6 hours. All baseline (B0) vital signs, including the patient's pulse rate (PR), non-invasive systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial blood pressure (MAP), were collected prior to surgery. Although it was not included for our study's purposes, Spo2 was also measured as part of the minimal standard monitoring technique. Preloading was carried out with lactated ringer solution roughly (10ml/kg) after intravenous access with an 18 G cannula. Subarachnoid block (SAB) was conducted using the study medication under strict aseptic guidelines, and the patient was subsequently placed in the supine position for the remainder of the research time. Following parameters were observed and recorded for data collection and statistics:

- **Time for onset of sensory level of the block upto T10 (min)** This was assessed by loss of pinprick sensation with 23 gauge hypodermic needle after injection of the study drug.
- **Time for onset of motor block Bromage 3 (min)** This was assessed by the **modified Bromage scale** as

0= no motor block
1= able to bend the knee (hip blocked)
2=able to dorsiflex the foot (hip and knee blocked)
3=complete motor block (hip, knee and ankle blocked).

- **Peak level dermatome**

Highest level dermatome was assessed by 23 gauge hypodermic needle after obtaining complete sensory block.

- **Duration of motor block(min)**

Time from end of anaesthetic injection to motor block regression (Bromage 0).

- **Duration of Analgesia(min)**

Time of onset of analgesia after spinal anaesthesia to onset of pain was recorded.

- **Time of first mobilization**
- **Time from end of anaesthetic injection to the first mobilization by the patient Haemodynamic parameters**

PR, SBP, DBP and MAP were recorded after 3, 5,10,15 ,30,60,90,120 and 150 min of study drug injection. During surgery, any fall in MAP below 20% of baseline value was treated with bolus dose of inj. Mephenteramine 6 mg i.v. PR <60 beats /min was treated with inj. Atropine sulphate 0.3-0.6 mg i.v. Total dosage of bolus drugs were recorded.

- **Side effects and complication** of the study drugs and technique including hypotension, hypertension, bradycardia, tachycardia, postoperative nausea vomiting (PONV), sedation, shivering and Transient Neurological Symptoms (TNS) were recorded if occurred.

STATISTICAL ANALYSIS

Data was composed in suitable spreadsheet i.e., SPSS 25.0. Statistical tests used were Student t-test (paired and unpaired) and Chi square test. Significance level will be 95% confidence level ($p<0.05$). Data was described as a frequency (Percentage) distribution as well as in Mean±SD.

RESULTS

All of the patients had successful spinal anaesthesia. According to Table 1, demographic information was similar between the two groups (P>0.5). With the addition of clonidine (GroupCC), the onset of sensory and motor block occurred more quickly (P 0.05). In group CC, more patients (64% vs. 32%; P 0.05) achieved the T6 T-9 level block than in group C. In comparison to group C, group CC showed longer durations of motor block, quicker time to mobilise, and post-operative analgesia (Table 2). In both group C and group CC, there were no significant variations in PR and all three blood pressure measures (P>0.05). Ramsay Sedation Score II was seen in 3 patients (6%) in group CC. Throughout the course of the trial, no further adverse effects or problems were noticed.

Table 1: basic profile of the patients

	GROUP C	GROUP CC	P VALUE
AGE in years	38.15±6.85	38.01±5.25	0.48
WEIGHT in kg	57.03±7.45	58.25±3.58	0.25
Male : Female	37:13	38:12	0.36
Duration of surgery(min)	34.05±6.74	35.28±5.85	0.47

Table 2: Clinical Parameter of the study groups

Clinical Parameter	Group C	Group CC	P value
Time of onset of sensory block upto T10 in min	10.25±1.25	8.14±1.36	0.015
Time of onset of motor block (Bromage 3) in min	12.36±1.58	10.74±1.85	0.016

Peak level dermatome Above T6 T6-T9 T10-T12	Nil (0%) 16 (32%) 34 (68%)	2 (4%) 32 (64%) 16 (32%)	0.014
Duration of motor block (minutes)	69.98±6.58	77.18±4.58	0.006
Duration of Analgesia (minutes)	102.58±11.25	195.85±10.25	0.00
Time of first mobilization (minutes)	121.25±4.85	211.98±4.89	0.00

DISCUSSION

Recently, preservative-free 1% Chloroprocaine has been reintroduced into clinical practise, allowing for earlier mobilisation and hospital release as well as quicker resolution of sensory and motor blockage. The early onset of postoperative discomfort restricts its usage in short-duration procedures despite its short duration and early mobilisation. In order to improve the quality of spinal anaesthesia, clonidine (1-2 mcg/kg) has been used as an adjuvant with other local anaesthetic agents[7]. However, these dosages may cause hypotension, bradycardia, and sleepiness. There aren't many research available on the use of clonidine as an intrathecal adjuvant with chloroprocaine.In our research, demographic information about both groups is similar (P>0.05). Although it had no clinical impact, both study groups exhibit a male predominance since most of the operations in our research are male urological procedures. As compared to group C, the time for the onset of sensory block (up to T10) and motor block (Bromage 3) was statistically quicker in group CC (8.14±1.36 vs. 10.25±1.25 and 10.74±1.85 vs. 12.36±1.58 min, respectively). Both Gordh T. Jret al[8] and Gaumann DM et al[9] noted that using clonidine and chloroprocaine together accelerated the onset of sensory block. They explained this by the effects of clonidine-induced postsynaptic hyperpolarization and presynaptic suppression of transmitter release. In comparison to group C, a greater proportion of patients in group CC exhibited block levels of T6-T9. Given that Davis BR et al[10] found no change when adding a lower dosage of clonidine (15 mcg) with chloroprocaine, we believe that the larger doses of clonidine (30 mcg) utilised in our investigation created this effect. As opposed to Ropivacaine alone, Kock MD et al[11] found a greater degree of block with the combination of Clonidine and Ropivacaine. When compared to the Chloroprocaine alone group, the duration of the motor block (Time for regression to Bromage 0) was substantially longer in the Chloroprocaine plus Clonidine group (P 0.05). Our findings are in line with those of Davis BR et al[10], who discovered a statistically significant difference between chloroprocaine and chloroprocaine combined with clonidine in the length of the motor block. With the addition of Clonidine intrathecally to local anaesthetics, the duration and intensity of the motor blockade were prolonged. This may be because 2 adrenoreceptor agonists cause cellular modification in the ventral horn of the spinal cord that results in the hyperpolarization of motor neurons, which facilitates the action of local anesthetics[12–14]. With the use of a combination of clonidine (30 mcg) and buprenorphine, Dobrydnjov I et al[7] also noted statistically significant lengthened duration of motor block. When comparing group CC to group C, it was shown that the duration of analgesia (MeanSD) was substantially longer in group CC (195.85±10.25 min vs 102.58±11.25 min) (P 0.05).Similar to Dobrydnjov I et al [7], they found that adding clonidine increased the duration of analgesia, but they came to the conclusion that increasing the dosage of clonidine from 15 mcg to 30 mcg did not prolong the duration of analgesia. When clonidine was administered intravenously, the spinal cord's substance gelatinosa's post synaptic 2-receptor was activated. The cholinergic action of clonidine makes more acetylcholine accessible to control analgesia[15–19].Throughout the research period, we found no statistically significant difference between the two study groups in any of the haemodynamic measures.(P<0.05) In contrast to group C (121.25±4.85 minutes), group CC had a longer initial mobilisation time (211.98±4.89 minutes). This finding is consistent with the research conducted by Davis BR et al[10]. Additionally, they said that all patients could only be mobilised once the block level declined to the S2 dermatome. We did not notice any adverse effects throughout our investigation. We monitored the patients for up to 72 hours, but we saw no patients develop TNS at all. This could be as a result of the preservative-free Chloroprocaine and Clonidine that we utilised. Our research's limitations include that, other from Davis BR et al.'s[16] work, there are no other studies on chloroprocaine and the addition of clonidine as an adjuvant with chloroprocaine that are accessible in the literature. As a result, we had several difficulties while collecting data for the study.

CONCLUSION

In short-duration nursery procedures, the intrathecal addition of preservative-free Clonidine (30mcg) to preservative-free 1% Chloroprocaine produces good spinal anaesthesia with extended analgesic duration and hemodynamic stability.

REFERENCES

1. Choi S, Mahon P, Awad IT. Neuraxial anesthesia and bladder dysfunction in the perioperative period: a systematic review. Can J Anaesth. 2012; 59:681– 703.
2. A prospective, double-blinded, randomized, clinical trial comparing the efficacy of 40 mg and 60 mg hyperbaric 2% prilocaine versus 60 mg plain 2% prilocaine for intrathecal anesthesia in ambulatory surgery. Camponovo C, Fanelli A, Ghisi D, Cristina D, Fanelli G. *Anesth Analg*. 2010;111:568–572.

3. Ureteroscopic lithotripsy as day care procedure: our early experience in Regional Institute of Medical Sciences. Khumukcham S, Gupta S, Lodh B, Kangjam SM, Akoijam KS, Sinam RS. *J Med Soc.* 2013;27:52–55.
4. Wang BC,Spielholz NI. Chronic neurological deficits and Nescaine –CE:an effect of the anesthetic, 2-chloroprocaine, or the antioxidant, sodium bisulfite? Anesth Analg 1984;63:445- 447.
5. Palas T. 1% Chlororprocaine for spinal anesthesia. RegAnesth Pain Med 2003;28:A52.
6. Dobrydnjov I, Holmstrom B. Postoperative pain relief following intrathecal bupivacaine combined with intrathecal or oral clonidine. Acta Anaesthesiol Sc and 2002;46: 806-814.
7. Dobrydnjov I, Holmstorm B et al. Clonidine combined with small dose bupivacaine during spinal anesthesia for inguinal herniorrhaphy: A randomised double blind study. AnesthAnalg 2003; 96:1496-1503.
8. Gordh T Jr,Post C. Interaction between noradrenergic and cholinergic mechanisms involved in spinal nociceptive processing. ActaAnaesthesiolSc and 1989; 33(1):39-47.
9. Gaumann DM, Jirounek P. Clonidine enhances the effects of lidocaine on C-fibre action potential. AnesthAnalg 1992; 74:719-725.
10. Davis BR, Kopacz DJ. Spinal 2-Chloroprocaine: the effect of added clonidine. AnesthAnalg 2005;100(2):559-565.
11. Kock MD, Hody JL. Intrathecal Ropivacaine and Clonidine for ambulatory surgery knee arthroscopy: a dose –response study. Anesthesiology 2001;94:574-578.
12. Dobrydnjov I, Samaruetal J. Enhancement of intrathecal lidocaine by addition of local and systemic clonidine. Acta Anaesthesiol Sc and 1999;43:556-562.
13. Bonnet F, Buisson VB, Francois Y, Catoire P, Saada M. Effects of oral and subarachnoid Clonidine on spinal anesthesia with bupivacaine. Reg Anesth 1990;15(4):211-214.
14. Racle J P, Benkhadra A, Poy J Y, Gleizal B. Prolongation of isobaric bupivacaine spinal anesthesia with epinephrine and clonidine for hip surgery in the elderly. AnesthAnalg 1987; 66:442-446.
15. Howe J R, Wang J Y, Yaksh T L. Selective antagonism of the antinociceptive effect of intrathecally applied alpha adrenergic agonists by prazosin and intrathecal yohimbine. J Pharm ExpTher 1983;224:552-558.
16. Probst A,Palacios M. Distribution of alpha -2-adrenergic receptors in the human brainstem-an audiographic study using(3H) p-amino–clonidine. Eur J Pharmacol 1984;106:477- 488.
17. Luttinger D,Haubrich DR. Pharmacological analysis of alpha-2- adrenergic mechanisms in nociception and ataxia. J Pharmacol ExpTher 1985;232:883-889.
18. Brandt SA, Livingston A. Receptor changes in spinal cord of sheep associated with exposure to chronic pain. Pain 1990;42:323-329.
19. Reddy SVR, Yaksh TL. Spinal noradrenergic terminal system mediates antinociception. Brain Res. 1980;189:391-401