

The effect of addition of intrathecal clonidine to hyperbaric bupivacaine on postoperative analgesic requirement in patient undergoing lower abdominal surgeries

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Abstract

Neuraxial anesthesia greatly expands the anesthesiologist armamentarium, providing alternatives to general anesthesia, especially in the lower abdominal surgeries. Clonidine, an alpha-2 adrenergic agonist, has a variety of actions, including potentiation of effects of local anesthetics. This study was undertaken to assess postoperative analgesia provided by low dose (30 mcg) intrathecal clonidine mixed with bupivacaine.

Material and methods: Sixty patients were randomly allocated in two groups A and B. Group A received bupivacaine 0.5%, 3 ml and Group B, bupivacaine 0.5%, 3 ml with clonidine 30 µg (0.2ml).

Result: Mean duration of regression of sensory block higher in GROUP B (310.2 ± 10.7) as compared to GROUP A (172.13 ± 7.2). Mean duration of regression of motor block higher in GROUP B (270.3 ± 8.0) as compared to GROUP A (162.3 ± 6.0). Duration of analgesia was significantly prolonged in group B (323.50 ± 26.7) as compared to group A (240.50 ± 17.6). HR decreased more in group B as compared to group A. Decrease in blood pressure was more in group B as compared to group A+

Conclusion: The findings in this study suggested that use of clonidine 30 µg added to bupivacaine for spinal anesthesia effectively increased the duration of sensory

block, duration of motor block, and duration of analgesia.

Keywords: Bupivacaine, Clonidine, Spinal Anesthesia, Analgesia.

Introduction

Human perception of pain is complicated and always has systemic consequences. Because pain is a subjective experience, it is challenging to quantify. Inadequate management of surgical pain can exacerbate anxiety and panic in patients recovering indoors, as well as postpone the therapeutic outcome. By combining a variety of adjuvant medications with local anesthetic agents, spinal anesthesia is a safe, dependable, and reasonably priced procedure that has the benefit of providing both surgical anesthesia and postoperative pain treatment. For lower abdomen and lower limb procedures, spinal anesthesia is consequently more frequently used than general anesthesia.^{1, 2}

Spinal anesthetic is used for lower abdominal surgery in order to create a motor and sensory block. Anesthesia can be made to last longer and start earlier by adding adjuvant drugs. This dramatic shift in the experience of pain reduction is due to the opiate receptors in the brain and the substantia gelatinosa of the spinal cord. As opioid adjuvants were associated with unfavorable outcomes such as nausea, itching, and delayed respiratory depression, the search for the ideal adjuvant went on.^{3,4} With a selectivity ratio of about 220:1 between α_2 and $\alpha_1.3$, the imidazoline compound clonidine preferentially and agonistically activates α_2 -adrenoceptors. In 1974, Paalzow first reported the antinociceptive properties of clonidine. The disruption of nociceptive stimulation in the spinal cord, supraspinal regions, and peripheral nervous system is the cause of the analgesic effects of

intraspinal clonidine. It stops C and A δ fibers from conduction by increasing potassium's conductivity.⁴

Spinal anesthesia has been used since long to produce sensory analgesia for surgeries below the level of the umbilicus.

In spinal anesthesia, the duration of sensory and motor block does not exceed 2.5–3.0 hours when local anesthetic (LA) is used alone. Although a variety of medications, including opioids, have been employed in the past to increase the effects of LAs, the quest for the perfect agent is still ongoing due to catastrophic adverse outcomes.^{5, 6} Many studies have been conducted on intrathecal clonidine as a potential substitute for neuraxial opioids in the treatment of pain. It has shown to be a highly effective analgesic without having at least some of the negative effects associated with opioids.⁵

In a dose-dependent way, clonidine may also result in bradycardia and hypotension. Clonidine also has sedative, antiemesis, decreased post-spinal shivering, and anxiolysis as side effects.⁷

The purpose of this study was to determine how intrathecal clonidine added to hyperbaric bupivacaine affected the need for postoperative analgesics in patients having lower abdominal surgery.

Materials and Methods

Study design

This was a comparative, observational, and prospective study conducted in SVP Hospital, NHL Municipal Medical College, Ahmedabad, India., 60 patients planned for the lower abdominal surgery were enlisted in the study. The sample size was obtained based on previous studies. ASA grade I & II, between 18-60 years of age of either sex, weight in range of 40-80kg and height in the range of 150-180 cm undergoing elective lower abdominal surgeries. All patients were randomly

distributed into two groups of 30 patients each. Group A (Bupivacaine group) received 0.5% heavy Bupivacaine 3ml (15mg) and Group B (Bupivacaine Clonidine group) received 0.5% heavy Bupivacaine 3ml (15mg) with preservative free Clonidine 0.2 ml (30 mcg).

Patient with chronic analgesic therapy, peripheral neuropathy, severe spinal deformity, and refusal; also using sympathomimetics and sympatholytic medications pregnancy and nursing. An allergy to local anesthetics is well-known. sensitivity to medications under research. H/o persistent headaches and backaches. localized illness at the location, coagulation abnormality, longer than three hours surgeries; ASA grades III, IV, and V.H/o alcohol and drug misuse, Angiotensin converting enzyme inhibitors, calcium channel blockers, alpha2-adrenergic receptor antagonists, and patients with dysrhythmias on the ECG are among the medications used by the patient.

A day before surgery, the patient had a thorough physical examination and a comprehensive preoperative history. We analyzed laboratory studies such as CBC, blood sugar, serum electrolytes, coagulation profile, chest X-ray, and ECG. The patient was given an explanation of the procedure and instructed to report any pain or discomfort they felt while having surgery. The patient was informed about the VAS score on a scale of 1 to 10. Patients and their families provided written informed permission. For six hours, all patients were given Nil by Mouth.

A patient's IV line was placed in the operating room and 10 millilitres per kilogram of Ringer lactate solution was preloaded. An ECG, non-invasive blood pressure monitor, and pulse oximeter were connected, and baseline readings were obtained. As a premedication, 4 mg of ondansetron was injected.

A lumbar puncture was carried out in the L2-L3 or L3-L4 intervertebral area with a 25G Quincke spinal needle while the patient was seated, adhering to all rigorous aseptic and antiseptic precautions. The medication was administered gradually. When the surgery was finished, the patient was placed in a supine position. It was detected when the medication was injected subarachnoidly. Following the administration of spinal anesthesia, pulse, blood pressure, SPO2, and respiratory rate were measured every 0,1,2,3,4,6,10,20,30,40,50, and 60 minutes, and then every 15 minutes for the next 120 minute.

Every 30 minutes till 300 minutes and thereafter at 60 minutes interval upto 720 minutes, in post-operative ward where further monitoring was continued.

The time interval between the study drug injection and the cessation of pinprick feeling will be used to determine the onset of sensory blockage. Using a 24-gauge needle, the degree of sensory block was measured and documented as a loss of sensation to pin prick, checking from the caudal to the cephalic direction. It was detected when the sensory blockage to the S2 dermatome would regress.

Onset of Motor Blockade will be assessed as the time from injection of study drug to the time to achieve modified Bromage grade 1, Modified bromage scale was followed according to which: 0 – able to raise the whole lower limb at the hip, 1 – able to flex the knee but unable to raise the leg at hip, 2 – able to plantarflex ankle but unable to flex the knee, and 3 – no movement of lower limb. Complete motor block recovery was assumed when Bromage score became zero. Time for onset of grade 3 motor blockade was noted. After establishment of adequate level of block, surgery was started and time of beginning of surgery was noted Onset of motor

blockade (Time required to produce grade 3 motor block) and duration from grade 3 to grade 0 was noted. Any post-operative problems, such as bradycardia, hypotension, sedation, shivering, nausea, vomiting, dry mouth, and respiratory depression, were monitored closely in the patients. Systolic blood pressure that dropped by more than 30% from the baseline was referred to as hypotension. The treatment for hypotension was Ephedrine 6 mg IV stat. >20% fall from baseline or heart rate less than 60 beats per minute was considered bradycardia. Intravenous glycopyrrolate (0.2 mg) or atropine (0.6 mg) are used to treat bradycardia.

Following surgery, patients were moved to the post-operative ward, where ECG, NIBP, and SPO2 monitors were installed.

The visual analogue scale (VAS) was used to measure patients' pain levels on a regular basis, and the timing of their requests for analgesia was recorded. Total duration of analgesia: Time of injection of study drug to first demand for rescue analgesia by patient.

The VAS measures the patient's subjective pain level by drawing a 10-centimeter line on a sheet of white paper.

Table 1: Demographic and Specific Characteristics (Mean +Sd):

	Group A (n=30)	Group B (n=30)	P Value	Interference
Ss	35.1±12.54	37.86±11.15	p>0.05	NS
Sex(M:F)	18:12	16:14		
Height	161.63± 7.75	164.96 ±7.05	p>0.05	NS
Weight	61.1±7.21	64.23± 6.46	p>0.05	NS
ASA I: II	19:11	20:10		
Duration of surgery (min)	138.67±19.42	144.67±23.15	p>0.05	NS

Table 1 shows demographic data between group A and group B. The two groups were comparable in Age, Height, Weight, Sex, ASA grade and duration of surgery and there was no statistically significant difference between the two groups (p>0.05).

Prior to surgery, every patient received an explanation that the number '0' indicates "no pain at all," while the number '10' indicates the "worst pain" the patient has ever experienced. The patient was asked to mark the scale to indicate the degree of pain. As a result, the pain score was calculated by calculating the separation between the indicated mark and the "0" end.

Statistical analysis: Statistical analysis was done using Epi info software. Data was expressed as mean, mean + SD and percentage. Data were compared by using the Z test. The level of significance used was p<0.05.

P value>0.05	Non-Significant (NS)
P value<0.05	Significant (S)

Results

The study was conducted in 60 patients (n=30 each) posted for lower abdominal surgeries under spinal anesthesia. All patients were randomly distributed into two groups of 30 patients each

Group A (Bupivacaine group): 0.5% heavy Bupivacaine 3ml (15mg). Group B (Bupivacaine Clonidine group): 0.5% heavy Bupivacaine 3ml (15mg) + Clonidine 0.2 ml (30 mcg).

Table 2: Base Line Preoperative Parameters (Mean \pm Sd):

Shows Baseline Preoperative Parameters between groups A and B. There was no statistical significant difference with regard to Baseline Heart Rate, SBP, DBP, MAP, RR and SPO2 between the two groups ($p>0.05$).

Parameters	Group A	Group B	P value	Interference
Pulse (/min)	86.16 \pm 5.84	87.6 \pm 4.73	$p>0.05$	NS
Systolic blood pressure (mm Hg)	128.93 \pm 4.35	130.06 \pm 3.25	$p>0.05$	NS
Diastolic blood pressure (mm Hg)	77.82 \pm 4.48	81.00 \pm 4.05	$p>0.05$	NS
Mean arterial pressure (mm Hg)	94.66 \pm 3.17	97.36 \pm 3.17	$p>0.05$	NS
Respiratory rate (min)	14.66 \pm 0.95	14.86 \pm 1.19	$p>0.05$	NS

Table 3: Characteristics of Sensory Blockade (Mean \pm Sd)

	Group A	Group B	P value	Interference
Onset of sensory blockade (min)	3.63 \pm 0.92	3.45 \pm 0.79	$p>0.05$	NS
Time for regression of sensory block to S2 dermatome(min)	172.13 \pm 7.27	310.2 \pm 10.73	$p<0.05$	S

Table 3 shows that there was no significant difference in onset of sensory block among both the groups ($p>0.05$).

There was statistically significant difference in time for regression of sensory block to S2 dermatome among both the groups ($p<0.05$).

Chart: 1 Time of Sensory Onset

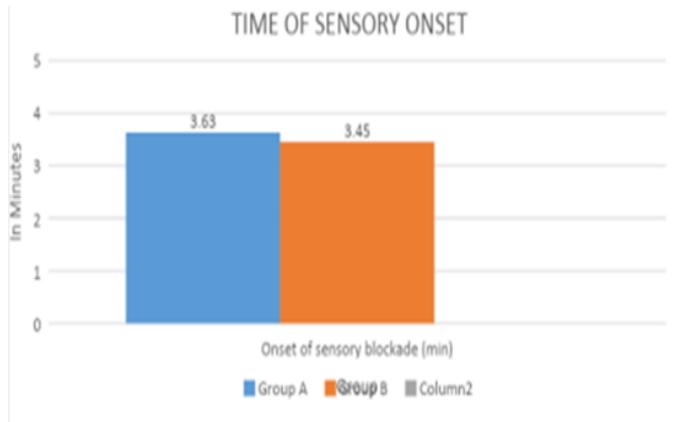
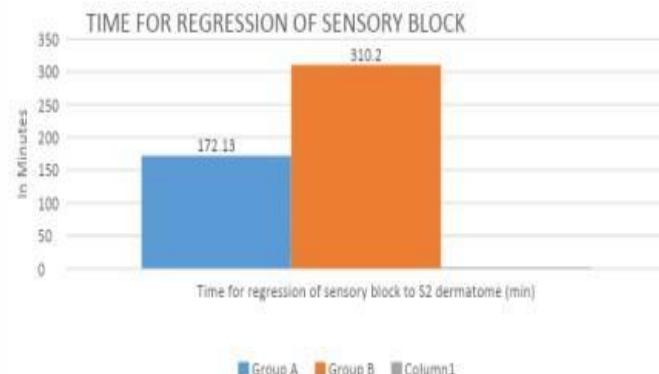


Chart 2: Time for regression of sensory block

Table 4: Characteristics of Motor Block (MEAN \pm SD):

	Group A	Group B	P value	Interference
Onset time to achieve score 3 motor block (min)	9.2 \pm 0.88	9.1 \pm 0.75	$p>0.05$	NS
Time for regression of motor block	162.3 \pm 6.08	270.3 \pm 8.01	$p<0.05$	S
Time for regression of motor block from score 3 to score 0 (min)	162.3 \pm 6.08	270.3 \pm 8.01	$p<0.05$	S

Table 4 shows that there was no significant difference in time to achieve score 3 motor blocks among both the groups ($p>0.05$). There was a statistically significant difference in time for regression of motor block from score 3 to score 0 among both the groups ($p<0.05$).

Chart 3: Perioperative Blood Pressure (MMHG) (MEAN \pm SD)

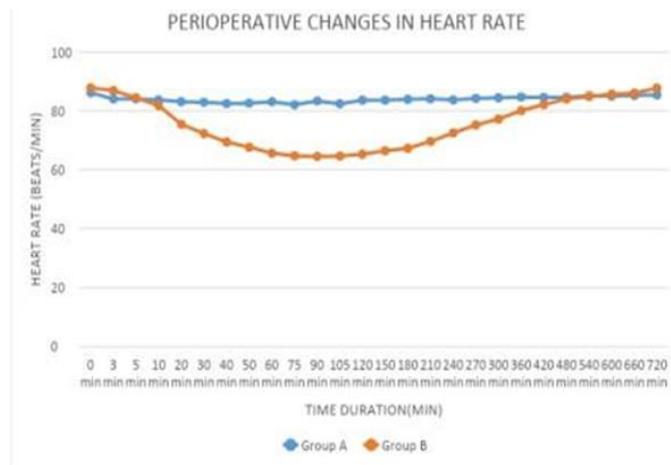


Chart 3 compares perioperative blood pressure among the groups. At 20 min onwards till 360 min (intraoperative and early postoperative period) after giving subarachnoid block, there was statistically significant difference in blood pressure among all the groups ($p<0.05$).

Decrease in blood pressure was more in group B as compared to group A. Postoperatively from 360 min onwards, there was no significant difference in blood pressure among both the groups ($p>0.05$)

Chart 4: Perioperative Blood Pressure (Mmhg)(MEAN \pm SD)

Chart 4 compares perioperative blood pressure among the groups. At 20 min onwards till 360 min (intraoperative and early postoperative period) after giving subarachnoid block, there was statistically significant difference in blood pressure among all the groups ($p<0.05$).

Decrease in blood pressure was more in group B as compared to group A. Postoperatively from 360 min onwards, there was no significant difference in blood pressure among both the groups ($p>0.05$).

Chart 4: Perioperative change in diastolic blood pressure

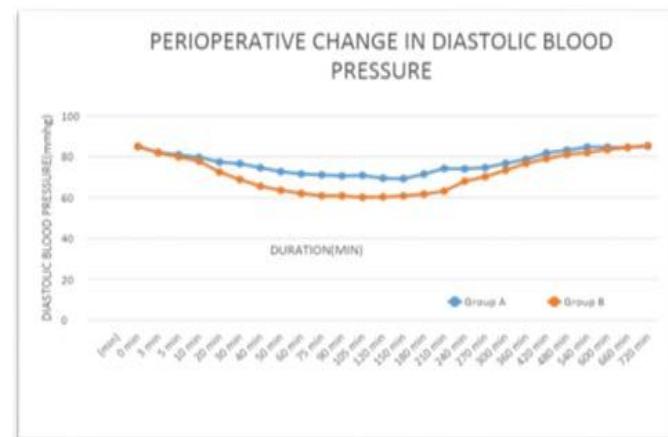


Chart 5: Perioperative change in systolic blood pressure

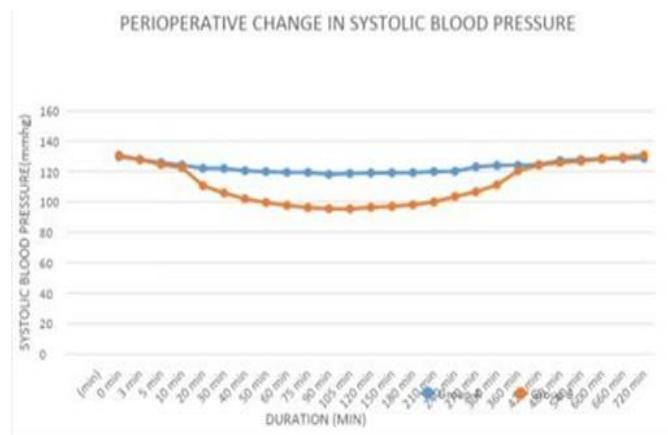


Table 5: shows perioperative change in Respiratory Rate and SpO2 between two groups which were normal and comparable in both groups and there is no statistical difference between two groups ($p>0.05$).

Table 5: Perioperative Respiratory Rate and Spo2 (Mean \pm Sd)

Respiratory Rate					SPO2			
Duration (min)	Group A	Group B	P value	Interference	Group A	Group B	P value	Interference
0 min	13.76 \pm 1.8	14.06 \pm 1.08	p>0.05	NS	98.8 \pm 0.48	98.70 \pm 0.53	p>0.05	NS
1 min	13.86 \pm 1.00	13.96 \pm 1.21	p>0.05	NS	98.3 \pm 0.70	98.67 \pm 0.55	p>0.05	NS
2 min	13.9 \pm 0.7	14.06 \pm 1.11	p>0.05	NS	98.6 \pm 0.62	98.67 \pm 0.48	p>0.05	NS
3 min	13.7 \pm 0.79	14.03 \pm 1.21	p>0.05	NS	98.43 \pm 0.62	98.60 \pm 0.50	p>0.05	NS
4 min	13.9 \pm 0.88	13.9 \pm 1.24	p>0.05	NS	98.5 \pm 0.50	98.67 \pm 0.48	p>0.05	NS
5 min	13.76 \pm 0.62	13.96 \pm 1.12	p>0.05	NS	97.06 \pm 7.78	98.63 \pm 0.56	p>0.05	NS
10 min	13.96 \pm 0.92	14.03 \pm 1.15	p>0.05	NS	98.56 \pm 0.50	98.70 \pm 0.53	p>0.05	NS
20 min	13.63 \pm 1.06	13.93 \pm 1.20	p>0.05	NS	98.5 \pm 0.50	98.60 \pm 0.50	p>0.05	NS
30 min	13.83 \pm 0.69	13.87 \pm 1.25	p>0.05	NS	98.36 \pm 0.61	98.63 \pm 0.61	p>0.05	NS
40 min	13.83 \pm 0.64	13.87 \pm 1.25	p>0.05	NS	98.5 \pm 0.50	98.63 \pm 0.61	p>0.05	NS
50 min	13.96 \pm 0.76	13.93 \pm 1.20	p>0.05	NS	98.6 \pm 0.56	98.67 \pm 0.55	p>0.05	NS
60 min	13.7 \pm 0.65	14.07 \pm 1.34	p>0.05	NS	98.53 \pm 0.50	98.73 \pm 0.64	p>0.05	NS
75 min	13.76 \pm 0.72	14.10 \pm 1.24	p>0.05	NS	98.53 \pm 0.50	98.67 \pm 0.55	p>0.05	NS
90 min	13.86 \pm 0.73	14.13 \pm 1.31	p>0.05	NS	98.53 \pm 0.50	98.73 \pm 0.45	p>0.05	NS
105 min	13.73 \pm 0.86	13.93 \pm 1.14	p>0.05	NS	98.6 \pm 0.56	98.73 \pm 0.45	p>0.05	NS
120 min	13.56 \pm 0.97	14.00 \pm 1.31	p>0.05	NS	98.43 \pm 0.62	98.67 \pm 0.48	p>0.05	NS
150 min	13.66 \pm 0.92	14.07 \pm 1.34	p>0.05	NS	98.43 \pm 0.62	98.67 \pm 0.48	p>0.05	NS
180 min	13.73 \pm 0.73	14.07 \pm 1.34	p>0.05	NS	98.6 \pm 0.56	98.67 \pm 0.48	p>0.05	NS
210 min	14.06 \pm 0.78	13.97 \pm 1.25	p>0.05	NS	98.46 \pm 0.62	98.67 \pm 0.48	p>0.05	NS
240 min	13.83 \pm 0.69	14.07 \pm 1.36	p>0.05	NS	98.46 \pm 0.62	98.73 \pm 0.45	p>0.05	NS
270 min	13.73 \pm 0.73	14.00 \pm 1.14	p>0.05	NS	98.53 \pm 0.50	98.67 \pm 0.55	p>0.05	NS
300 min	13.6 \pm 0.67	14.00 \pm 1.14	p>0.05	NS	98.6 \pm 0.62	98.67 \pm 0.55	p>0.05	NS
360 min	13.76 \pm 0.77	14.13 \pm 1.11	p>0.05	NS	98.56 \pm 0.50	98.67 \pm 0.55	p>0.05	NS
420 min	13.76 \pm 0.72	14.00 \pm 1.26	p>0.05	NS	98.46 \pm 0.62	98.67 \pm 0.55	p>0.05	NS
480 min	13.83 \pm 0.69	14.00 \pm 1.31	p>0.05	NS	98.56 \pm 0.50	98.70 \pm 0.53	p>0.05	NS

540 min	13.73±0.78	14.00±1.31	p>0.05	NS	98.6±0.62	98.60±0.50	p>0.05	NS
600 min	13.76±0.77	14.00±1.31	p>0.05	NS	98.46±0.62	98.60±0.50	p>0.05	NS
660 min	13.83±0.69	13.97±1.25	p>0.05	NS	98.53±0.50	98.73±0.64	p>0.05	NS
720 min	13.7±0.91	14.00±1.26	p>0.05	NS	98.6±0.62	98.73±0.64	p>0.05	NS

Table 5 shows perioperative change in Respiratory Rate and SpO2 between two groups which were normal and comparable in both groups and there is no statistical difference between two groups (p>0.05)

Table 6: Perioperative Complications

Complications	Group A		Group B	
	Intra-op	Post-op	Intra-op	Post-op
Hypotension	1(3.33%)	0	3(10%)	0
Bradycardia	1(3.33%)	0	3(10%)	0
Nausea/ Vomiting	0	0	1(3.33%)	0
Respiratory depression	0	0	0	0
Shivering	2(6.66%)	0	0	0
Urinary retention	0	0	0	0
Dryness of mouth	0	0	1(3.33%)	1(3.33%)

Table 6 shows Perioperative Adverse Effects between both groups, with incidence of Hypotension and Bradycardia being more in Group B, 10% and 10 % respectively as compared to group A ,3.3 % and 3.3% respectively. The incidence of Nausea and vomiting

was 3.33% in group B as compared to group A while Shivering was observed in 6.66 % in group A as compared to group B and dryness of mouth was observed in 3.33% in group B in both intra n post op period as compared to group A.

Table 7: Sedation Score

Sedation score	Group A	Group B
1	0	0
2	0	0
3	0	12(40%)
4	0	18(60%)
5	0	0
6	0	0

Table 7 compares sedation score of both the groups. In group A 100% patients were awake. In group B 40% patients were awake, 60% patients were awake and comfortable

Chart 6: Time To First Rescue Analgesia

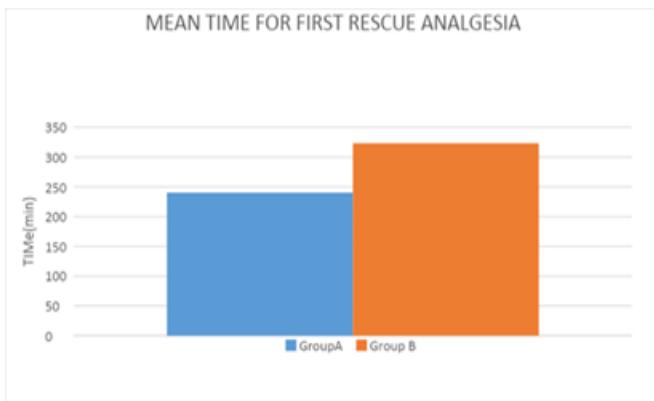


Chart 6 compares duration of analgesia among both the groups. There was statistically significant difference regarding duration of analgesia among both the groups ($p<0.05$). Duration of analgesia was significantly prolonged in group B as compared to group A.

Discussion

Nowadays, regional anesthesia is frequently used for lower abdominal procedures. A good anesthetic approach is to provide appropriate muscular relaxation during the intraoperative phase and good analgesia to relieve pain during the postoperative phase. Since pain affects a patient's morbidity and death, it's critical to relieve pain for the best possible outcome for the patient.

Lower abdominal procedures are frequently conducted under spinal anesthesia because, in contrast to epidural and general anesthesia, spinal anesthesia is a simple, one-shot procedure. However, the primary issue with spinal anesthesia is that postoperative analgesia only lasts for a significant amount of time.

In this study, the effects of 0.5% hyperbaric Bupivacaine alone and 0.5% hyperbaric Bupivacaine with 30 μ g of clonidine added are compared, and the length of postoperative analgesia and intraoperative hemodynamic stability in patients having lower abdominal procedures are assessed.

Ghodki PS et al⁷ in 2010 studied 30 mcg of Clonidine intrathecally and concluded that it significantly prolongs the duration of spinal anaesthesia thus extending the analgesia as indicated by delayed demand for rescue analgesia in the postoperative period. After pre anesthetic check-up and getting informed consent, 60 patients of ASA grade I and II of both sex, between 18-60 years of age, who were scheduled to lower abdominal surgeries were included in the study. They were randomly allocated into 2 groups of 30 patients each.

Bupivacaine group A: 3 milliliters (15 milligrams) of 0.5% strong Bupivacaine. Group B (Bupivacaine Clonidine group): 0.2 ml (30 mcg) of clonidine and 3.ml (15 mg) of strong 0.5% Bupivacaine. Table 1 displays the demographic variables (age, sex, weight, height), length of operation, and ASA Grade that were comparable ($p>0.05$) between the two groups in our study.

Table 2 indicates that, prior to surgery, all patients in both groups had hemodynamically comparable values for pulse, systolic and diastolic blood pressure, respiratory rate, and SpO₂ ($p>0.05$).

The study found no statistically significant difference in the period of sensory blockage beginning, with 3.63 ± 0.92 (min) in Group A and 3.45 ± 0.79 (min) in Group B, as indicated in Table 3. This finding is consistent with the following research: According to research by Bansal Sangeeta et al. 8 (2014), intrathecal bupivacaine and clonidine (45 mcg) had no influence on when sensory blockage started.

In a 2010 study, Ghodki PS et al⁷ examined two groups: one that received intrathecally administered Bupivacaine and the other that received 30 μ g of clonidine in addition to Bupivacaine. They found that the addition of clonidine had no influence on the start of sensory

blockage. In a 2011 study, Bhavini Shah found that intrathecal Bupivacaine did not alter the start of sensory blocking when clonidine (1 mcg/kg) was added. Chendraya Perumal, R S, and H S Suraj, 15 (2019) It was discovered that the onset of the motor and sensory blocks was comparable in the two groups that received clonidine and midazolam. The greatest level of block attained did not show a statistically significant difference between the two groups.

In our study, statistically significant difference ($p < 0.05$) was found among both the groups regarding duration of regression of sensory blockade to S2 dermatome. It was 172.13 ± 7.27 (min) in Group A and 310.2 ± 10.73 (min) in Group B as shown in table 3. Kanazi GE et al¹²(2006) studied effect of low dose Clonidine (30 μ g) with hyperbaric Bupivacaine(12mg) intrathecally and observed mean time of sensory regression to the S1 segment was 272 ± 38 (min) with Clonidine group while in Bupivacaine group it was 190 ± 48 9(min) B.S. Sethi et al¹¹(2007) noted time for regression of sensory blockade by two segments was 150-240 min in Clonidine (1 mcg/kg) group, which was significantly longer than duration of 90-130 min in control group. Thakur et al⁹ observed that mean time to two-segment regression, regression to L3 dermatome, and time to first analgesic request was significantly more in Clonidine groups (15 mcg, 30 mcg) than in control group.

Table 4 displays a statistically significant difference ($p < 0.05$) in the regression time of motor block from score 3 to score 0 (min), with 162.3 ± 6.08 (min) for Group A and 270.3 ± 8.201 (min) for Group B. It agrees with the research listed below: According to B.S. Sethi et al. (2007)¹¹ the Clonidine group (1 mcg/kg) experienced a mean length of motor block of 205 minutes, while the control group experienced a mean

duration of 161 minutes. Regression of motor block takes longer in the Clonidine group (216 ± 35 min) than in the Bupivacaine alone group (163 ± 47 min) ($p < 0.05$), according to Kanazi et al.¹² In comparison to the control group (74.5 ± 7.16), Bhavini Shah et al. 10(2011) found that the addition of Clonidine (1 mcg/kg) considerably increased the time of motor blockage (129.55 ± 14.55). As of 2008, Van Tuijl et al.¹³ had determined that postoperatively from 360 min onwards, there was no significant difference in HR and blood pressure among both the groups ($p > 0.05$). In our study, no significant difference in the perioperative RR and SPO2 was noted among both the groups as shown in table 7. Bhavini Shah et al¹⁰ in 2011 studied Clonidine (1 μ g/kg) intrathecally and concluded that heart rate at 15 min. compared to 2 min. is significantly less in the Clonidine group. Hemodynamic parameters were on the lower side in the Clonidine group during the first hour of surgery. B.S. Sethi et al¹¹ and Grandhe et al¹⁴ observed a decrease in mean heart rate from 45 mins until 6 hours in Clonidine group compared to control group. Respiratory rate and Spo2 remain stable in both the groups in our study.

10% of patients in group B and 3.33% of patients in group A had intraoperative bradycardia. Group B experienced intraoperative bradycardia (<60 bpm) and was treated with injectable atropine 0.6 mg intravenously B. S. Sethi et al.¹¹ (2011) reported significantly lower heart rates in the Clonidine group compared to the control group ($p < 0.05$).

During the perioperative phase, group B reported 3.33% of cases of nausea/vomiting and 6.66% of cases of dry mouth, while group A reported none of these symptoms. According to B.S. Sethi et al.¹¹(2011), the Clonidine group experienced 10% incidence of nausea/vomiting

and 33% incidence of dry mouth, while the control group experienced 3.33% incidence of nausea/vomiting and 16.6% incidence of dry mouth. There were 6.66% of patients in group A who shivered, but none in group B.

In our study in group B 12(40%) patients show grade 3 and 18(60%) patients show grade 4 sedation score. In group A 25(100%) patients show grade 1 sedation score, as shown in Table 9, which shows sedative effect of Clonidine. B.S. Sethi et al¹¹ in 2007 studied that patients who received Clonidine were more sedated than those in the control group (16 out of 30 patients were sedated in Clonidine group but none in control group). Time for first rescue analgesia was 240.5 ± 17.63 mins in group A compared to 323.50 ± 26.70 min in group B which was statistically significant difference ($p < 0.05$) as shown in table 10.

After comparing 30 mcg of Clonidine with Bupivacaine intrathecally, Ghodki PS et al.⁷ (2010) found that this combination significantly lengthens the duration of spinal anesthesia and, consequently, the duration of analgesia. This is demonstrated by the delayed demand for rescue analgesia in the post-operative period, which was 195.5 minutes for the Bupivacaine group and 261.5 minutes for the Clonidine group. In patients undergoing gynecological surgery, B.S. Sethi et al. (2011)¹¹ discovered that the intrathecal Bupivacaine 12.5 mg + Clonidine (1 μ g/kg) group experienced a considerably longer duration of effective analgesia (614 min) than the Bupivacaine group (223 min). In a 2003 study, Dobrydnjov et al.¹⁴ examined the effect of intrathecal Clonidine with Bupivacaine on the duration of time until the first analgesic request; with Bupivacaine, this was 171 ± 65 minutes, while with Bupivacaine + Clonidine, it was 274 ± 94 minutes, and with Bupivacaine + Clonidine, it was 253 ± 71 minutes.

Conclusion

Addition of intrathecal clonidine provides adequate analgesia and motor paralysis at significantly lower dose of bupivacaine. On the basis of current study, we can draw the conclusion that intrathecal administration of 30 mcg Clonidine in combination with 0.5% hyperbaric Bupivacaine produces better quality of analgesia compared to Bupivacaine alone in lower abdominal surgeries. Advantages are: Superior quality of analgesia, longer duration of sensory and motor block, Prolonged postoperative analgesia, Delayed rescue analgesic requirement, Minimal side effect.

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