

ORIGINAL RESEARCH

A Comparative Study of Epidural Bupivacaine with Buprenorphine and Bupivacaine with Fentanyl in Lower Limb Surgeries

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ABSTRACT

Background: The current study compares the hemodynamic, sedative, and analgesia-potentiating effects of fentanyl and buprenorphine delivered epidurally in combination with bupivacaine.

Methods: A prospective study was performed by Department of Anaesthesia, Guntur Medical College, Guntur, India at Government General Hospital, Guntur. This study included 60 individual undergoing lower limb surgeries that were divided in two groups. Wherein group A received solution of 15 ml of 0.5% bupivacaine hydrochloride and 3 g/kg buprenorphine. And group B administered with solution consisting of 15 ml of 0.5% bupivacaine hydrochloride and 1 g/kg fentanyl.

Result: The mean time for onset of motor block in group A was 9.53 ± 1.14 minutes and in group B it was 6.43 ± 1.04 minutes. The mean time of onset of motor block was significantly lower in group B when compared to group A. The mean duration of return to Bromage score 0 in group A was 230.17 ± 12.70 mins and in group B it was 332.83 ± 14.42 mins. There was a statistically significant difference in the mean duration of return to Bromage score 0 across the groups. The time required in group B was significantly higher when compared to group A.

Conclusion: Buprenorphine performs better than fentanyl when administered epidurally in terms of providing effective long-term postoperative analgesia. For preoperative analgesia, buprenorphine, fentanyl, and 0.5% bupivacaine can be injected epidurally in a single dose.

Keywords: Epidural anaesthesia, bromage, local anesthetic.

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INTRODUCTION

For lower extremity procedures, central neuraxial anaesthesia is the method of choice. The two most often utilised regional anaesthetic methods for lower limb orthopaedic surgery are intrathecal anaesthesia (ITA) and epidural anaesthesia (EA).^[1,2] However, there are a number of drawbacks to spinal anaesthesia, including a brief period of postoperative analgesia and postdural puncture headache (PDPH). Modern anaesthesiology is quickly adopting EA as a useful and adaptable technique since it enables the practitioner to deliver both anaesthesia and analgesia. Compared to subarachnoid block anaesthesia, the advantages of EA include

the capacity to give surgical anaesthesia that is effective for longer periods of time, prolonged post-operative analgesia, and a lower incidence of hemodynamic changes brought on by sympathetic blockade.^[2,3] The anaesthetic most frequently used for epidural anaesthesia is bupivacaine. Recent studies have shown that postoperative analgesia with the buprenorphine group was unquestionably of a longer duration when compared to the fentanyl group; therefore, when used as an adjunct to bupivacaine, epidural buprenorphine is better at providing sustained satisfactory postoperative analgesia in comparison to fentanyl.^[3]

A localised anaesthetic and an opioid combination can offer greater analgesia during the perioperative and postoperative period. The mu-receptor is a partial agonist and antagonist of buprenorphine. A opioid drug agonist with a phenylpiperidine derivative, fentanyl. The current studies were created to compare epidural bupivacaine combined with buprenorphine to bupivacaine combined with fentanyl for less invasive orthopaedic surgical procedures.^[3,4]

The FDA has given buprenorphine approval to treat both acute and chronic pain as well as opioid addiction. It is a drug used in agonist substitution therapy, a technique for drug addiction treatment that involves replacing a medication (such buprenorphine or methadone) with a more potent full agonist opioid (such as heroin). Buprenorphine is a thebaine derivative, it is 33 times more potent than morphine, and it is a mu-receptor partial agonist and antagonist. It is effective in relieving moderate to severe pain.^[3,4] The physician then gradually reduces the alternative, and the patient goes through opiate withdrawal with little discomfort.^[4,5] The patient is able to focus on therapy rather than the uncomfortable withdrawal symptoms thanks to the use of buprenorphine substitution therapy. In individuals receiving addiction treatment, it is a potential choice for treating opioid addiction, lowering cravings, and enhancing quality of life. Patients are more likely to adhere to a treatment plan as a result, which lowers morbidity and mortality. It also helps the patient avoid many of the unpleasant symptoms of opiate withdrawal.^[5-7]

A potent synthetic opioid that is comparable to morphine but generates more analgesia is called fentanyl. This potent pharmacologic substance is often 50–100 times more powerful. Analgesia comparable to around 10 mg of morphine can be achieved with a single dose of 100 micrograms of the drug.^[7,8]

On the other hand, fentanyl has highly distinct pharmacokinetics and characteristics. Its most frequent clinical uses are as a sedative in mechanically ventilated patients and as a severe pain reliever in patients with renal failure due to its predominantly hepatic clearance. Patients with persistent pain who have grown tolerant to opiates may also be treated with fentanyl by medical professionals.^[8,9] It is typically given as a sedative through drip due to its adaptability in titration circumstances. It may be necessary to administer high doses when employed as a sedative in individuals who need mechanical breathing. Additionally, fentanyl is offered as a pre-medication during surgery for treatments that are expected to be uncomfortable. The use of fentanyl for treating epilepsy is a final option. That is, in conjunction with specific neuroleptic drugs as part of therapeutic neuroleptanalgesia.^[9-12]

MATERIALS AND METHODS

A prospective study was undertaken at the Government General Hospital in Guntur by the anaesthesia department of the Guntur Medical College. The study included a total of 60 individuals. Who were hospitalised to the Government General Hospital in Guntur between January 2021 and December 2021 with plans to have lower limb surgery. Group A and Group B will be created out of all sixty patients at random. - Epidural study solution of 15 ml of 0.5% bupivacaine hydrochloride and 3 g/kg buprenorphine will be administered to Group A (n = 30). - Epidural study solution consisting of 15 ml of 0.5% bupivacaine hydrochloride and 1 g/kg fentanyl will be administered to group B (n = 30). The subsequent block features

must be noticed and noted: beginning of a sensory block maximum degree of sensory obstruction time to reach the highest sensory level, It's time to end the automobile blockade. Now is the time for two-segment regression, length of the analgesic, length of the motor block.

Inclusion Criteria:

1. Age group – 18-60 years both sex
2. ASA grade 1 and 2
3. Weight 30-80 kgs
4. Patients who give informed valid consent
5. Patients who are scheduled to undergo various lower abdominal and lower limb surgical procedures under epidural anaesthesia.

Exclusion Criteria:

1. Patients not willing to be a part of the study.
2. Having local skin infection along lumbar spine.
3. Spinal deformity.
4. Chronic backache.
5. Headache.
6. Drug addiction.
7. Neurological deficit.
8. Bleeding/clotting disorder.
9. Cardiovascular disease.
10. Systemic metabolic disorders such as severe hepatic or renal disease were excluded from the study

RESULTS

Table 1: Age distribution in years

	Group A		Group B		Total	
	N	%	N	%	N	%
21 – 30	3	10.0%	3	10.0%	6	10.0%
31 – 40	6	20.0%	11	36.7%	17	28.3%
41 – 50	13	43.3%	11	36.7%	24	40.0%
51 – 60	8	26.7%	5	16.7%	13	21.7%
Total	30	100.0%	30	100.0%	60	100.0%
Mean ± SD	44.20 ± 9.76		42.50 ± 8.31		41.20 ± 9.24	
Chi square test = 0.50, p = 2.30 , Not statistically significant						

The mean age of the participants in group A was 44.20 ± 9.76 . in group A 10% were aged 21-30 years, 20% were aged 31-40 years, 43.3% were aged 41-50 years, 26.7% were aged 51-60 years. The mean age of the participants in group B was 42.50 ± 8.31 years, in group B 10% belonged to 21-30 years, 36.7% were aged 31-40 years, 36.7% were aged 41-50 years, 16.7% were aged 51-60 years. There was no statistically significant difference was observed across the groups and thus both the groups stand comparable in terms of age.

Table 2: Gender distribution

	Group A		Group B		Total	
	N	%	N	%	N	%
Male	19	63.3%	16	53.3%	35	58.3%
Female	11	36.7%	14	46.7%	25	41.7%
Total	30	100.0%	30	100.0%	60	100.0%
Chi square test = 0.60, p = 0.43 , Not statistically significant						

In the present study, in group A 63.3% were male, 36.7% were female and in group B 53.3% were male and 46.7% were female. Here it was observed that both the groups stand comparable in terms of age as no statistically significant difference was observed.

Table 3: Anthropometry details

	Group A	Group B	T value	95% CI	P value
Height	165.36 ± 8.43	167.70 ± 7.90	1.10	1.89 – 6.55	0.27
Weight	57.80 ± 10.17	57.66 ± 7.40	0.05	4.79 – 5.46	0.95
BMI	20.80 ± 2.28	20.46 ± 1.71	0.64	1.37 – 0.70	0.52

There was no significant difference in the mean height, weight, BMI across the groups and thus they stand comparable in terms of anthropometric measures.

Table 4: ASA status

	Group A		Group B		Total	
	N	%	N	%	N	%
I	19	63.3%	21	70.0%	40	66.7%
II	11	36.7%	9	30.0%	20	33.3%
Total	30	100.0%	30	100.0%	60	100%
Chi square test = 0.60, p = 0.43 , Not statistically significant						

Both Group A and Group B stand comparable in terms of ASA grading.

Table 5: Diagnosis

Diagnosis	Group 1	Group 2	Total
# LT Leg Both Bone	1	0	1 (1.7%)
# RT Leg Both Bone	1	0	1 (1.7%)
# Femur's Neck RT	1	0	1 (1.7%)
# RT Neck of Femur	1	0	1 (1.7%)
# RT Femur's Shaft	1	0	1 (1.7%)
# Shaft f Femur LT	1	0	1 (1.7%)
Bilateral Osteo Arthritis	0	1	1 (1.7%)
Chronic Pain RT Knee	1	0	1 (1.7%)
RT Femur Closed # Mid Shaft	1	0	1 (1.7%)
Both Bone Closed Communicated # Segmental	1	0	1 (1.7%)
# Shaft of RT Tibia Communicated	1	0	1 (1.7%)
Infected With Implant Non Union of LT Femur	1	0	1 (1.7%)
Non Union of LT Femur Infected With Mangled	2	0	2 (3.3%)
LT # Both Bone Leg	0	2	2 (3.3%)

LT # Midshaft Femur	0	1	1 (1.7%)
LT # Neck of Femur	0	1	1 (1.7%)
LT # Tibia Proximal Shaft	0	1	1 (1.7%)
LT # Shaft of Femur	0	2	2 (3.3%)
LT Complete Acl Tear	0	2	2 (3.3%)
LT Intertrochanteric #	3	0	3 (5.0%)
LT Intertrochanteric # Femur	2	2	4 (6.7%)
LT Lower 1/3rd # Tibia Shaft	1	0	1 (1.7%)
LT Nondisplaced	0	2	2 (3.3%)
# Neck of Femur LT Old Non United	2	0	2 (3.3%)
Shaft of Femur LT Segmental #	1	0	1 (1.7%)
LT Subtrochanteric #	0	2	2 (3.3%)
Medial Malleolar Nonunion RT	0	1	1 (1.7%)
RT # Both Leg Bone	0	1	1 (1.7%)
# Neck Of Femur RT	0	4	4 (6.7%)
RT # Tibia Shaft	0	2	2 (3.3%)
RT Bimalleolar #	3	0	3 (5.0%)
RT Intertrochanteric #	0	4	4 (6.7%)
RT Intertrochanteric # Femur	3	0	3 (5.0%)
RT Knee Acl Tear	0	1	1 (1.7%)
RT Tibial Plateau #	1	0	1 (1.7%)
RT Tibial Shaft # Middle 1/3rd	1	0	1 (1.7%)
Trimalleolar # of Lt Ankle	0	1	1 (1.7%)

Table 6: Baseline hemodynamic

	Group A ±	Group B	P value
PULSE	81.40 ± 8.87	82.50 ± 8.94	0.317
SBP	126.47 ± 7.05	127.23 ± 1.86	0.346
DBP	80.50 ± 7.65	82.90 ± 8.48	0.084
MAP	95.30 ± 6.61	97.90 ± 9.07	0.066
R.R	17.43 ± 0.86	25.00 ± 1.07	0.121

In the present study the mean of baseline haemodynamic parameters stand comparable in both the groups as no statistically significant difference was observed.

Table 7: Pulse Rate

	Group A	Group B	P value
5min	83.17 ± 8.84	81.80 ± 8.83	0.277
10min	83.14 ± 8.11	82.40 ± 8.08	0.364
15min	80.38 ± 7.25	80.00 ± 8.87	0.424
30min	79.28 ± 7.01	79.63 ± 7.81	0.422
45min	77.41 ± 7.37	77.80 ± 7.37	0.415
60min	79.90 ± 4.75	79.77 ± 7.63	0.466
75min	80.21 ± 5.04	80.33 ± 8.19	0.469
90min	81.52 ± 5.21	79.77 ± 8.97	0.096
105min	79.41 ± 4.72	79.70 ± 7.84	0.410
120min	78.62 ± 4.73	79.20 ± 7.17	0.358

135min	77.55 ± 6.33	83.97 ± 7.03	0.001
150min	76.59 ± 6.69	83.43 ± 7.29	0.000
165min	75.69 ± 4.32	81.43 ± 7.38	0.000
180min	80.00 ± 5.35	82.33 ± 8.22	0.082

In the present study no significant difference in the mean pulse rate was observed across the groups till 120 mins and the difference started appearing across the groups around 135 mins. The pulse rate started returning back to the levels of baseline at around 135 mins in group B whereas it was around 180 mins in case of group A.

Table 8: MAP

	Group A	Group B	P value
5min	94.76 ± 6.47	97.80 ± 9.16	0.025
10min	92.24 ± 7.10	94.43 ± 9.58	0.096
15min	88.59 ± 6.77	91.53 ± 8.72	0.028
30min	85.90 ± 6.45	88.97 ± 8.44	0.026
45min	84.69 ± 5.63	87.60 ± 8.87	0.019
60min	84.66 ± 5.45	87.63 ± 8.39	0.012
75min	85.03 ± 6.06	86.70 ± 8.42	0.116
90min	87.48 ± 5.99	87.03 ± 8.58	0.383
105min	86.17 ± 7.45	87.13 ± 8.49	0.287
120min	86.41 ± 7.86	90.33 ± 8.50	0.008
135min	89.93 ± 8.75	91.73 ± 8.62	0.162
150min	88.48 ± 9.60	90.73 ± 8.48	0.136
165min	90.28 ± 6.06	91.00 ± 9.94	0.336
180min	91.00 ± 5.48	91.73 ± 9.58	0.306

There was a statistically significant difference in the MAP across the groups till around 60 mins, where the MAP was significantly higher in the group B when compared to group A. after 60 mins no significant difference was observed across the groups.

Table 9: Respiratory rate

	Group A	Group B	P value
5min	17.62 ± 12.80	15.57 ± 1.48	0.193
10min	17.97 ± 12.95	15.43 ± 1.90	0.145
15min	17.97 ± 12.00	15.67 ± 1.32	0.151
30min	17.45 ± 12.47	15.67 ± 1.21	0.22
45min	18.24 ± 11.94	16.40 ± 1.25	0.202
60min	17.97 ± 12.95	15.67 ± 1.12	0.168
75min	18.07 ± 11.40	15.77 ± 1.19	0.138
90min	18.41 ± 11.56	15.90 ± 1.12	0.12
105min	18.55 ± 13.80	15.60 ± 1.28	0.124
120min	18.31 ± 10.96	15.70 ± 1.64	0.101
135min	18.41 ± 12.86	14.87 ± 1.43	0.069
150min	18.55 ± 14.95	14.67 ± 1.60	0.081
165min	15.83 ± 2.30	15.37 ± 1.25	0.267
180min	16.07 ± 2.07	16.23 ± 1.51	0.393

There was no significant difference in the respiratory rate across the groups over time and both the groups stand comparable. Though no statistically significant the respiratory rate was lower in group B when compared to group A.

Table 10: Onset of Analgesia in Min

	Group A	Group B	P value
T12	7.72 ± 3.25	6.17 ± 7.28	0.018
T10	11.62 ± 2.93	9.77 ± 1.69	0.007
T8	16.00 ± 2.89	12.58 ± 1.76	0.000
T6	18.55 ± 2.77	15.75 ± 1.80	0.013

At various sensory thresholds, there was a statistically significant difference in the onset of analgesia across the groups. When compared to group A, the length of onset of analgesia in group B was shown to be significantly shorter.

Table 11: Motor Bromage – Mean Duration of Analgesia

Dermatome level	Group A	Group B	P value
0	6.10 ± 2.55	6.00 ± 6.76	0.428
1	10.14 ± 2.64	9.17 ± 1.97	0.057
2	13.86 ± 2.63	13.30 ± 1.09	0.228
3	19.28 ± 3.08	17.70 ± 1.22	0.03

The mean duration of analgesia in the motor component was not significantly different across the groups at various levels and across various duration of time, the mean duration of onset at dermatome 3 was significantly lower in group B when compared to group A.

Table 12: VAS Score in Hours

	Group A	Group B	P value
1	0.50 ± 0.00	0.73 ± 0.47	0.004
2	0.50 ± 0.00	1.08 ± 0.72	0.000
4	0.60 ± 0.20	1.60 ± 0.20	0.000
6	0.83 ± 0.61	2.20 ± 0.36	0.000
8	1.18 ± 0.77	3.38 ± 1.56	0.000
10	2.18 ± 1.56	2.30 ± 2.69	0.391
12	2.50 ± 1.27	2.75 ± 2.74	0.269
14	3.37 ± 1.39	1.82 ± 3.48	0.000
16	3.14 ± 1.46	1.90 ± 3.75	0.000
18	0.68 ± 0.52	1.03 ± 0.90	0.035
20	0.75 ± 0.25	1.00 ± 0.00	0.000

There was a statistically significant difference in the mean VAS scores across the groups from the 1st hour itself and it was observed that the mean VAS score was lower in group A participants when compared to those in group B.

Table 13: Post-operative hemodynamic

	Group A	Group B	P value
Pulse	82.03 ± 4.41	80.90 ± 8.42	0.214
SBP	115.27 ± 6.46	112.93 ± 1.02	0.062

DBP	74.20 ± 7.97	72.33 ± 7.52	0.148
MBP	87.83 ± 6.16	86.87 ± 8.26	0.241
RR	16.30 ± 1.49	16.40 ± 1.25	0.39

There was no statistically significant difference in the post-operative mean haemodynamic parameters across the groups, thus the haemodynamic parameters stand comparable across the groups.

Table 14: Rescue analgesia given in minutes

	Group A	Group B	P value
Mean Duration of Analgesia	774 ± 174	463.2 ± 105	0.000

The mean duration at which rescue analgesia was needed in group A was at 774 ± 174 minutes and in group B it was 463.2 ± 105. A statistically significant difference was observed in the mean duration at which rescue analgesia was needed across the groups and it was observed that group B participants needed rescue analgesia at an earlier duration, when compared to those in group A.

Table 15: Side effects

	Group A	Group B	Total	P value
Nausea and Vomiting				
	13	2	15	0.0001*
Pruritus	0	10	10	0.0001*
Urinary retention	0	0	0	-

In group A majority had nausea and vomiting and in group B majority complained of pruritus. There was a statistically significant difference in the presentation of cases with side effects across the groups.

Table 16: Comparison of Mean time of onset of sensory block upto T10 in min

	Group A	Group B	P value
Sensory block onset time (mins)	7.10±0.84	4.97±0.81	<0.0001*

The mean time at onset of sensory block upto T10 was 7.10±0.84 mins in group A and it was 4.97±0.81 mins. The mean duration for onset of anaesthesia in group A was significantly higher in group A when compared to group B.

Table 17: Comparison of Mean Time for Maximum sensory block

	Group A	Group B	P value
Time for Maximum sensory block in mins	13.10±1.03	8.27±0.69	<0.0001*

The mean time for maximum sensory block in group A was 13.10±1.03 mins and in group B it was 8.27±0.69 mins. The mean duration required for maximum sensory block in group A was significantly higher when compared to group B.

Table 18: Mean time for onset of Motor block in Minutes

	Group A	Group B	P value
Mean Time for Onset of motor block in minutes	9.53±1.14	6.43±1.04	<0.0001*

The mean time for onset of motor block in group A was 9.53±1.14 minutes and in group B it was 6.43±1.04 minutes. The mean time of onset of motor block was significantly lower in group B when compared to group A.

Table 19: Mean Time for Maximum motor block (Bromage 3)

	Group A	Group B	P value
Time for Maximum motor block (Bromage 3)	19.00±1.62	19.03±1.56	0.942

The Mean Time for Maximum motor block (Bromage 3) in group A was 19.00±1.6 mins and in group B it was 19.03±1.56 mins. No statistically significant difference was observed in the mean values across the groups.

Table 20: Comparison of 2 Segment Regression time in minutes

	Group A	Group B	P value
2 Segment Regression time in minutes	106.67±7.81	195.33±11.44	<0.001*

The mean 2 segment regression time in group A was found to be 106.67±7.81 mins and in group B it was 195.33±11.44. The mean time for 2 segment regression was significantly higher in group B when compared to group A.

Table 21: mean duration of Regression time to S1

	Group A	Group B	P value
mean duration of Regression time to S1	243.00±11.03	391.83±13.61	<0.001*

The mean duration of regression time to S1 in group A was 243.00±11.03 mins and in group B it was 391.83±13.61 mins. There was a statistically significant difference across the groups in the mean time of regression to S1 the time taken for regression to S1 in group B was significantly higher in group B when compared to group A.

Table 22: mean duration of regression to Bromage 0

	Group A	Group B	P value
Mean duration of regression to Bromage 0	230.17±12.70	332.83±14.42	<0.001*

The mean duration of return to Bromage score 0 in group A was 230.17±12.70 mins and in group B it was 332.83±14.42 mins. There was a statistically significant difference in the mean duration of return to Bromage score 0 across the groups. The time required in group B was significantly higher when compared to group A.

DISCUSSION

Pain is a complex, subjective experience that has proven challenging to quantify in a repeatable manner. Fast pain is a sensory discriminative feature that describes the location and nature of the stimulus, whereas slow pain is a motivational emotional component that leads to an unpleasant aspect.^[12,13] In clinical practise, providing pain treatment that is satisfactory is a constant challenge. It has been discovered that post-operative pain is worse right away and progressively becomes better over the course of the following 24 hours. The persistence of pain has served as a persistent catalyst for the development of medications and other pain-relieving techniques. It is crucial to address post-operative discomfort not just for humanitarian reasons but also to avoid physical morbidity.^[13,14] The tissue is damaged as the anaesthetic wears off, and the pain-inducing chemicals released during the surgery significantly lower the nociceptors' normally high threshold, making harmless stimulation painful. Additionally, axon damage plays a part in nociception. Both pharmaceutical and nonpharmacological methods can be used to treat pain. However, despite the expanding selection of tools at our disposal, effective pain management is still difficult to achieve.^[15,16] One of the most often utilised techniques for lower limb surgery is epidural anaesthesia because it effectively blocks sensory and motor functions. Additionally, it lessens the negative physiological effects after surgery, including autonomic hyperactivity, cardiovascular stress, elevated metabolic rate, pulmonary dysfunction, and immune system malfunction. Bupivacaine is the medication used for regional anaesthesia the most frequently in modern anaesthesiology practise.^[17,18] To improve the quality of analgesia and lengthen the duration of action, numerous adjuvants have been explored. Spinal anaesthesia is preferable over epidural anaesthesia because it allows for top-up anaesthetic and analgesic dosages and allows for targeted block levels to be obtained without significant hemodynamic changes.^[18,19] Modern anaesthesia practises frequently include epidural anaesthesia, particularly for patients having lower body procedures. A topical anaesthetic with desirable properties, such as prolonged sensory blockade and shorter motor blockade duration, was created to satisfy this aim.^[19,20] Epidural Bupivacaine was typically used for post-operative analgesia. 0.5% epidural bupivacaine, 0.25 for sensory and autonomic blockade, and 0.125 for autonomic blockade cause the motor, sensory, and sympathetic blocking. Epidural and intrathecal opioids are currently used to give intraoperative and postoperative analgesia. In 1976, Taksh and Rudy showed that the spinal cord has opioid receptors that can generate potent analgesia.^[20-22] It has now been established that epidural opioid administration is preferable than intravenous and intramuscular opioid injections. At Guntur Medical College, Guntur, researchers compared the hemodynamic, sedative, and analgesia-potentiating effects of epidurally administered fentanyl and buprenorphine when combined with bupivacaine. The study was titled "A Comparative Study of Epidural Bupivacaine with Buprenorphine and Bupivacaine with Fentanyl in Lower Limb Surgeries." Following informed agreement, 60 ASA class I and II patients scheduled for various elective lower limb procedures were at random divided into two groups: group A received buprenorphine with buprenorphine (A), group B received fentanyl with buprenorphine (B). An epidural catheter was inserted and secured 3 cm inside the epidural space, and a test dose of 3 ml of 2% lignocaine with adrenaline was administered, with any intravascular or intrathecal catheter placement being observed. The epidural space was found using a loss of resistance approach to air under strictly adherent aseptic conditions. The research drug was then administered into the patient in 16 ml, and several parameters were recorded.

CONCLUSION

We compared the effectiveness of epidural injections of buprenorphine versus injections of fentanyl with 0.5 percent bupivacaine for lower limb surgeries. The results showed no significant hemodynamic or respiratory effects in either group. The post-operative analgesia lasted substantially longer in the buprenorphine group. Buprenorphine users are more likely to experience nausea and vomiting than fentanyl users, while both of these adverse effects can be treated with antiemetics such Ondansetron. Because of this, buprenorphine performs better than fentanyl when administered epidurally in terms of providing effective long-term postoperative analgesia. For preoperative analgesia, buprenorphine, fentanyl, and 0.5% bupivacaine can be injected epidurally in a single dose.

REFERENCES

1. Cousins, M. J., Bridenbaugh, P. O., Carr, D. B., & Horlocker, T. T. (Eds.). (2009). Cousins and Bridenbaugh's neural blockade in clinical anesthesia and pain medicine. Lippincott Williams & Wilkins.
2. Kumar, P., & Singh, S. (2020). Comparative study of outcome in epidural bupivacaine with buprenorphine and bupivacaine with fentanyl in lower limb surgeries. *IJMA*, 3(1), 106-113.
3. Dhakshinamoorthy, M., Srinivasan, S. K., & Sittaramane, S. (2017). Comparative Study of the Effect of Buprenorphine and Fentanyl as an Adjunct to Bupivacaine in Epidural Anesthesia for Lower Abdominal and Lower Limb Surgeries. *International Journal of Scientific Study*, 5(1), 22-26.
4. Wheatley, R. G., Schug, S. A., & Watson, D. (2001). Safety and efficacy of postoperative epidural analgesia. *British journal of anaesthesia*, 87(1), 47-61.
5. Downing, J. W., Leary, W. P., & White, E. S. (1977). Buprenorphine: a new potent long-acting synthetic analgesic. Comparison with morphine. *British Journal of Anaesthesia*, 49(3), 251-255.
6. Justins, D. M., Francis, D., Houlton, P. G., Reynolds, F., & Marx, G. F. (1983). A Controlled Trial of Extradural Fentanyl in Labour. *Survey of Anesthesiology*, 27(1), 31.
7. D'Angelo, R., Gerancher, J. C., Eisenach, J. C., & Raphael, B. L. (1998). Epidural fentanyl produces labor analgesia by a spinal mechanism. *The Journal of the American Society of Anesthesiologists*, 88(6), 1519-1523.
8. Bartels, K., Mayes, L. M., Dingmann, C., Bullard, K. J., Hopfer, C. J., & Binswanger, I. A. (2016). Opioid use and storage patterns by patients after hospital discharge following surgery. *PloS one*, 11(1), e0147972.
9. Chou, R., Gordon, D. B., de Leon-Casasola, O. A., Rosenberg, J. M., Bickler, S., Brennan, T., ... & Wu, C. L. (2016). Management of Postoperative Pain: a clinical practice guideline from the American pain society, the American Society of Regional Anesthesia and Pain Medicine, and the American Society of Anesthesiologists' committee on regional anesthesia, executive committee, and administrative council. *The journal of pain*, 17(2), 131-157.
10. Suner, Z., Kalayci, D., Sen, O., Kaya, M., Unver, S., & Oguz, G. (2019). Postoperative analgesia after total abdominal hysterectomy: is the transversus abdominis plane block effective?. *Nigerian Journal of Clinical Practice*, 22(4), 478-478.
11. Horn, R., & Kramer, J. (2019). Postoperative pain control.
12. Rawal, N. (2016). Current issues in postoperative pain management. *European Journal of Anaesthesiology| EJA*, 33(3), 160-171.

13. Triffterer, L., Marhofer, P., Lechner, G., Marksz, T. C., Kimberger, O., Schmid, W., & Marhofer, D. (2017). An observational study of the macro-and micro-haemodynamic implications of epidural anaesthesia in children. *Anaesthesia*, 72(4), 488-495.
14. Strandness, T., Wiktor, M., Varadarajan, J., & Weisman, S. (2015). Migration of pediatric epidural catheters. *Pediatric Anesthesia*, 25(6), 610-613.
15. Antibas, P. L., do Nascimento Junior, P., Braz, L. G., Doles, J. V. P., Módolo, N. S., & El Dib, R. (2014). Air versus saline in the loss of resistance technique for identification of the epidural space. *Cochrane Database of Systematic Reviews*, (7).
16. Harrison, G. R., & Clowes, N. W. B. (1985). The depth of the lumbar epidural space from the skin. *Anaesthesia*, 40(7), 685-687.
17. Afshan, G., Chohan, U., Khan, F. A., Chaudhry, N., Khan, Z. E., & Khan, A. A. (2011). Appropriate length of epidural catheter in the epidural space for postoperative analgesia: evaluation by epidurography. *Anaesthesia*, 66(10), 913-918.
18. Wolfe, R. C., & Spillars, A. (2018). Local anesthetic systemic toxicity: Reviewing updates from the American Society of Regional Anesthesia and Pain Medicine Practice advisory. *Journal of PeriAnesthesia Nursing*, 33(6), 1000-1005.
19. Li, J., Duan, R., Zhang, Y., Zhao, X., Cheng, Y., Chen, Y., ... & Zhao, S. (2018). Beta-adrenergic activation induces cardiac collapse by aggravating cardiomyocyte contractile dysfunction in bupivacaine intoxication. *PLoS one*, 13(10), e0203602.
20. Prabhakar, A., Lambert, T., Kaye, R. J., Gagnard, S. M., Ragusa, J., Wheat, S., ... & Kaye, A. D. (2019). Adjuvants in clinical regional anesthesia practice: A comprehensive review. *Best Practice & Research Clinical Anaesthesiology*, 33(4), 415-423.
21. Barrington, M. J., & Uda, Y. (2018). Did ultrasound fulfill the promise of safety in regional anesthesia?. *Current Opinion in Anesthesiology*, 31(5), 649-655.
22. Preuss, C. V., Kalava, A., & King, K. C. (2019). Prescription of controlled substances: benefits and risks.