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Original Article

Anaesthesia Section

Comparison of Epidural Bupivacaine, Levobupivacaine and Dexmedetomidine in Patients Undergoing Vascular Surgery

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ABSTRACT

Introduction: Levobupivacaine is the s-isomer of racemic Bupivacaine. It is less cardio, neurotoxic and equally potent local anaesthetic compared to its racemate. It is known to cause less Depression of myocardial contractility. Dexmeditomidine when used via epidural route has synergistic effect with local anaesthetics. Majority of patients presenting for vascular surgery are elderly and have associated co-morbidities like diabetes, hypertension, and coronary artery disease. We intend to study safety and efficacy of epidural Levoupivacaine and Dexmedetomidine in this group of patients.

Materials and Methods: Sixty adult patients undergoing lower limb vascular surgery under lumbar epidural anaesthesia were randomly allocated to three groups. All groups were preloaded with 10ml/kg of crystalloid solution. B group was scheduled to receive 15 ml of racemic Bupivacaine, L-group was scheduled to receive 15ml of Levobupivacaine and LD-group received 15ml of Levobupivacaine with 0.5 mics/kg Dexmeditomedine. Time to onset of sensory block to T-10, maximum sensory level achieved, Bromage scale, time to two segment regression, time to total regression, sedation level achieved and patients assessment of quality of anaesthesia were assessed. Haemodynamic parameters were monitored throughout study period. Adverse effects were noted and treated appropriately.

Results: Baseline parameters were comparable among all the groups. Time to onset of sensory block to T-10 and maximum level of block achieved, was comparable among the groups. Time to two segment regression and time to total regression was significantly prolonged in LD group compared to other two groups. There was significant bradycardia noted in LD group which required treatment.

Conclusion: Levobupivacaine can be safely used in elderly high risk patients undergoing vascular surgery. Addition of dexmedetomidine prolongs the duration of anaesthesia and postoperative analgesia.

Keywords: Alpha blocker, Cardiotoxiciy, Epidural anaesthesia, Racemate, Vascular surgery

INTRODUCTION

Epidural anaesthesia forms an excellent mode of anaesthesia for patients undergoing lower limb vascular surgery. Bupivacaine, a long acting amide local anaesthetic has long been extensively used in this setting. Although it is generally well tolerated, concern has been raised over its relative cardiotoxicity. Cardiovascular collapse and death have been the most serious adverse effect of bupivacaine, likely due to its ability to depress the intracardiac conduction velocity and cardiac contractility. These effects of Bupivacaine have been atributed to its blocking properties of Na^+ and K^+ channels [1,2].

Bupivacaine contains an asymmetric carbon atom that gives it a chiral centre. It is a racemic mixture of two enantiomers: levo- or S(-) Bupivacaine and dextro- or R(+) bupivacaine. These have identical physical and chemical properties, but there is evidence of stereo specificity of action, in particular with relation to cardiotoxicity, Bupivacaine induced cardiotoxicity has been mostly related to the effects of its R(+) enantiomer, which exhibits a higher potency for blocking cardiac Na+ and K^+ channels. Studies in animal models with levo isomer, Levobupivacaine, showed that its lethal dose is 1.3 to 1.6 times greater than that of the racemic presentation. In human beings, Levoupivacaine has a less negative inotropic effect and would cause a smaller lengthening of the PR and QT intervals in the electrocardiogram, it characterizes the intoxication of the racemic formulation [3,4].

Alpha 2 agonists have sedative properties and analgesic action at peripheral, spinal and supraspinal levels, Dexmedetomedine, a highly selective alpha2 agonist, has synergistic action with local anaesthetics [5,6]. Majority of patients presenting for vascular surgery are elderly, have associated co-morbidities like hypertension, diabetes mellitus, ischemic heart disease.

In the present study we intend to compare the anaesthetic characteristics of racemic bupivacaine with its levoisomer and effect of adding dexmedetomidine to the block characteristics in this high risk group of population.

MATERIALS AND METHODS

After review and approval from institutional ethical committee and informed consent, 60 adult patients who were scheduled to undergo lower limb vascular surgery were included in the study. On the basis of pilot study data, present study was designed to be able to detect a 15% difference among study groups with regard to duration of sensory block and haemodynamic side effects. A power calculation based on these assumptions with an alpha of 0.05 and a beta of 0.8 resulted in the need for more than or equal to 20 patients in each treatment group. Patients with history of hypersensitivity to amide local anaesthetics, presence of a blood-clotting disorder, platelet count <100 000 mm^3, blood dyscrasia, refusal of or inability to receive an epidural block, cutaneous infections or anatomical malformation at the puncture site, severe respiratory, renal, hepatic disease, A-V or intraventricular conduction abnormalities, seizure disorders and patients with weight more than 110 kg or height less than 150 cm were excluded from the study.

Patients who were included in the study (n=60) were randomly divided into three groups using computer generated table of random numbers, group (n=20), group L (n=20) and group LD (n=20). All patients were premedicated with Diazepam 5 mg on the night before and on the morning of surgery. On arrival in the operating room, peripheral venous access was established with a wide bore cannula, inj Midazolam 1 mg iv was administered, preloaded with 10ml/kg of normal saline, Lidocaine 1% (3 mL) was used to infiltrate the skin and subcutaneous tissues at the L1-L2 or L2-3 interspace. The epidural space was identified in the lateral decubitus position by using an 18-gauge Tuohy needle and a loss of resistance technique, epidural catheter was inserted 3-4 mm into the epidural space and patient turned supine. 15 microgram of epinephrine was added to 3 mL study drug, after negative aspiration, it was administered as a "test dose". When there was no evidence of intravascular or subarachnoid injection (heart rate > 100 bpm, systolic blood pressure >90 mm Hg, or presence of sensory block) after 2 min, an additional 12 ml of the study drug was administered as slow injection at 1ml/min rate. Heart rate, Invasive blood pressure, Spo_2 and respiration were monitored throughout the study period. Hypotension, defined as more than 30% fall in systolic blood pressure as compared to the baseline, or SBP<90 mm of Hg was treated with a bolus of fluids, if not responding to the bolus, inj Ephidrine 6 mg incremental bolus was administered. Bradycardia defined as more than 30% fall in heart rate as compared to the baseline or< 40 bpm was treated with inj atropine 0.6 mg IV.

Adequate block to initiate surgery was defined as a sensory block bilaterally to dermatome T10. Sensory block was assessed bilaterally in the mid-clavicular line by using the blunt end of a 27-gauge dental needle.

Time taken to achieve this level of anaesthesia was noted. Block height, time to reach peak block, time to two-segment regression and total duration of sensory block was noted.

The onset, degree, and duration of motor block were measured in both legs by using a modified Bromage scale and scored as:

– zero- no paralysis, full flexion of hips, knees, and ankles;
– One- inability to raise extended leg, able to move knees;
– Two-inability to flex knees, able to flex ankles;
– Three-inability to move any portion of the lower limb [7]

Motor block was measured every 30 min postsurgery until the patient returned to a score of zero in both legs.

Haemodynamic parameters were monitored throughout study period, sedation level achieved, patients assessment of quality of anaesthesia, time to request of first dose of analgesia, any adverse effects associated were monitored and appropriately treated.

STATISTICAL ANALYSIS

Age, height, weight and block characteristics were analysed by analysis of variance (ANOVA) and Kruskal Wallis test. Serial data were analysed with repeated-measures analysis of variance. Chi-square test and Fisher-exact test was used for non-parametric data. Data was analysed using SPSS version 18.P less than 0.05 was considered significant.

RESULTS

Sixty patients were enrolled in the study. Adequate level of block was achieved in all the study patients with 15 ml of the study drug. Baseline characteristics.

There was no difference among the groups in terms of age, sex, weight, height. Duration of surgery was comparable among the groups [Table/Fig-1].

	Bupivacaine	Levo Bupivacaine	Levobupivacaine+ Dexmedetomedine	p-value
Age	63.90±7.033	67.95±7.937	67.25±7.772	.206
Height	165.60±7.148	166.05±5.799	167.70±5.602	.535
Weight	67.00±4.877	65.80±5.979	68.35±6.675	.42
Gender M/F	16/4	17/3	17/3	
Duration of Surgery	135.85±20.934	139.75±18.629	135.05±14.240	.683

[Table/Fig-1]: Demographic variables

	Bupivacaine	Levo Bupivacaine	Levo Bupivacaine+ Dexmedeto-medine	p-value
Time to onset to T-10	11.95±2.625	11.75±2.381	12.90±3.582	.416
Maximum sensory level achieved	T4-t6	T4-t8	T4-t6	.480
Time to maximum sensory level	19.00±4.425	22.40±3.719	21.35±5.071	.054
Maximum motor block achieved (Bromage score) 1/2/3	6/9/5	12/6/2	10/7/3	.523
Sedation score (Ramsay sedation score) 1/2/3/4	4/15/1/0	3/15/2/0	0/5/12/3	.001
Patients assessment of quality poor/fair/ good/excellent	0/5/9/6	0/4/11/5	0/2/7/11	.001

[Table/Fig-2]: Block intiation variables

Time to onset of sensory block to T-10 was comparable among the groups with average time to onset being 12 min [Table/Fig-2]. Maximum sensory level achieved was around T-4 to T-8 and was comparable among the groups. Time to maximum sensory block was also comparable among the groups [Table/Fig-2].

Maximum motor block was more in Bromage 1 in the Levobupivacaine and LD group compared to Bupivacaine which had more intense block with more patients achieving Bromage score of 2 and 3 though the findings have not reached statistical significance. (p=0.523, [Table/Fig-2]).

Patients in Levobupivacaine with Dexmedetomedine group had significantly better sedation levels achieved compared to the other two groups. Also, none of the patients had desaturation or respiratory depression requiring intervention in any of the groups. This indicates the active sedation inducing property of Dexmedetomidine. Patient's assessment of quality of anaesthesia was also better in LD group compared to the B and L groups accounting for sedative, anxiolytic and analgesic action of Dexmedetomedine [5,6] [Table/Fig-2].

	Bupivacaine	Levo Bupivacaine	Levo Bupivacaine+ Dexmedeto-medine	p-value
Two Segment Regression	100.25±18.387	92.00±17.652	131±19.708	.001
Time to Regression to S1	4.95±0.536	4.88±.705	6.63±.686	.001
Time to Return to Bromage 0	3.80±.410	3.85±.671	4.83±.922	.001
Time to First Request of Analgesia	330.50±46.394	325.00±47.848	496.00±30.505	.001

[Table/Fig-3]: Post-block characteristics

Post block characteristics

There was significant increase in the duration for two segment regression, regression to S1and motor block regression to Bromage 0 in LD group compared to other two groups which have comparable findings among themselves [Table/Fig-3].

Time to first request of analgesia was also significantly prolonged in LD group compared to other two groups [Table/Fig-3].

Baseline heart rates were comparable among the groups. From 15 min heart rate reduced significantly in LD group compared to the other two groups. None of the patients in groups B and L groups required atropine in contrast to 5 patients in LD group who required atropine, however all patients who required atropine responded to single dose of atropine and remained haemodynamically stable throughout the study period [Table/Fig-4].

Baseline systolic blood pressures were comparable among the groups [Table/Fig-5]. From 20 min post drug administration, mean systolic blood pressure decreased in LD group when compared to B and L groups and in LD group, though there was a initial fall in the blood pressure below the baseline, patients remained haemodynamically stable throughout the study period [Table/Fig-5].

Diastolic Blood Pressure

Baseline diastolic blood pressure was comparable among the groups, and there was no significant difference in DBP among the groups throughout the study period [Table/Fig-6].

Side Effects

None of the patients in any group had any significant side effects throughout the study period. Two patients in LD group complained

Time Min	Bupivacaine		Levobupivacaine		Levobupivacaine+ Dexmedetomedine		p-value
	Mean	SD	Mean	SD	Mean	SD	
0	72.65	5.932	71.90	10.770	71.95	9.265	.956
2	71.50	6.565	69.80	10.278	69.25	8.902	.698
5	68.30	15.634	68.75	9.124	67.25	8.765	.916
10	71.55	7.156	68.90	9.216	64.05	11.390	.045
15	71.90	6.357	69.65	9.522	63.95	11.852	.031
20	71.80	6.420	70.30	10.834	65.60	11.914	.133
25	71.85	6.327	71.25	12.315	62.65	12.762	.016
30	71.10	5.590	71.10	9.830	63.15	11.918	.013
40	71.35	6.401	72.05	9.350	64.95	11.546	.037
50	67.65	17.239	71.75	9.607	63.30	11.943	.143
60	71.75	5.270	71.85	9.235	65.20	12.003	.041
90	71.95	4.718	73.95	9.259	66.00	8.766	.006
Atropine Given	0		0		5		0.002

[Table/Fig-4]: Haemodynamic variables: Pulse rate

Time Min	Bupivacaine		Levobupivacaine		Levobupivacaine+ Dexmedetomedine		p-value
	Mean	SD	Mean	SD	Mean	SD	
0	134.85	12.930	134.75	11.187	136.90	11.580	.813
2	121.20	11.624	128.40	10.787	122.05	13.500	.147
5	115.25	11.470	122.35	13.035	115.75	11.369	.189
10	112.35	12.253	118.35	13.926	111.35	8.580	.167
15	112.35	12.253	114.00	16.902	105.50	11.478	.126
20	111.90	15.117	118.70	11.685	106.75	11.350	.017
25	116.75	14.223	113.55	15.753	107.75	10.361	.116
30	117.25	15.248	118.00	9.793	105.65	9.832	.002
40	125.60	7.970	121.30	8.386	105.95	10.107	.001
50	125.25	9.414	120.85	8.969	107.60	10.884	.001
60	124.80	8.192	121.80	9.871	108.20	10.807	.001
90min	123.75	8.602	121.10	8.961	106.05	11.993	0.001

[Table/Fig-5]: Haemodynamic variables: systolic blood pressure

Time Min	Bupivacaine		Levobupivacaine		Levobupivacaine+ Dexmedetomedine		p-value
	MEAN	SD	MEAN	SD	MEAN	SD	
0	70.60	8.029	74.45	5.969	73.90	6.632	.174
2	66.65	7.220	70.60	5.103	69.25	5.702	.122
5	65.50	7.970	68.80	5.217	67.75	5.702	.259
10	64.20	8.108	68.55	5.216	66.20	6.313	.127
15	63.75	8.071	67.25	5.600	63.70	7.109	.194
20	63.75	8.012	67.10	4.689	64.60	6.116	.238
25	63.90	7.225	65.00	6.325	65.35	6.310	.772
30	64.40	6.151	66.90	4.930	64.45	6.117	.300
40	63.40	5.725	65.90	5.046	64.75	6.942	.419
50	64.20	5.791	66.25	5.543	64.15	6.667	.458
60	64.25	5.418	66.75	5.486	64.15	6.869	.227
90	64.85	6.055	67.90	5.025	64.15	6.869	.122

[Table/Fig-6]: Haemodynamic variables: Diastolic '+' blood pressure

of dryness of mouth. Number of patients requiring Vasopressor did not differ among the groups of patients [Table/Fig-7].

	Bupivacaine	Levobupivacaine	Levobupivacaine+ Dexmedetomedine	p-value
Nausea	0	0	0	
Vomiting	0	0	0	
Dizziness	0	0	0	
Dry Mouth	0	0	2	
Vassopressor Administered	3	2	5	0.244

[Table/Fig-7]: Side effects

DISCUSSION

This study demonstrates that epidural levobupivacaine is a suitable anaesthetic for use in vascular surgery. Levobupivacaine provided adequate sensory block for surgery in all patients. Levobupivacaine and Bupivacaine showed equivalent efficacy for the time taken to reach sensory block adequate for surgery. Sensory block to T10 was achieved within 15 minutes of administering the epidural injection in all groups and the maximum spread of sensory block was observed within 30 minutes.

Levobupivacaine is the second local anaesthetic to be studied clinically as a pure single isomer. Ropivacaine was the first single enantiomer local anaesthetic to be approved. However, studies suggest that Ropivacaine is less potent than Bupivacaine when administered by epidural injection [8].

Cardiovascular toxicity by local anaesthetics results in either direct myocardial depression, or arrhythmogenicity. Studies have shown that Levobupivacaine has a reduced potential to cause cardiotoxic effects. In a study which involved 14 volunteers (the intentional intravascular administration at10 mg/minutes until mild CNS symptoms developed) Levobupivacaine produced significantly less effects on myocardial function than racemic Bupivacaine. Changes in stroke index, acceleration index, and ejection fraction were less marked with Levobupivacaine. Mild CNS symptoms developed at a larger dose level of Levobupivacaine (56.1 mg) than with racemic Bupivacaine (47.9 mg). Evidence suggests that Levobupivacaine may provide a greater safety margin than Bupivacaine from direct depression of myocardial contractility in humans, same findings have also been observed in animal studies [3,4].

In the present study, onset, duration of sensory block were comparable between B and L groups which have a similar tolerability profile with comparable rates of adverse events. Our findings are in agreement with study by Dan J Kopacz et al., who studied the effects of epidural Levobupivacaine 0.75% with Racemic Bupivacaine for Lower Abdominal Surgery. They did not notice any difference in the onset, duration and tolerability profile among the two groups [9]. In the present study we noticed less intense motor blockade in L and LD groups compared to Bupivacaine group, which is in agreement with study by Dan J Kopacz et al.. Pasquale De Negri et al., studied the effect of Epidural Bupivacaine, Levobupivacaine, and Ropivacaine on postoperative analgesia and motor blockade and found that Bupivacaine had significantly higher motor block compared to the other groups. Study by B Locatelli et al., also showed higher motor blockade in Bupivacaine group compared to Levobupivacaine [10,11]

There has been a constant search for adjuvants for local anaesthetics to improve quality of regional anaesthesia. Alpha2 adrenoceptor agonists are now being used with great interest in anaesthesia practice for their sympatholytic, sedative, analgesic, and anaesthetic-sparing effects. Clonidine has been used extensively for this purpose. Dexmedetomidine is a more selective alpha 2 agonist with a greater selectivity for the apha2 receptors than the alpha1 receptor. It was introduced in clinical practice in the United States in 1999 and approved by the FDA only as a short-term (<24 hours) sedative Dexmedetomidine is shorter acting drug than clonidine and has a reversal drug, Atipamezole, for its sedative effect [5,6].

At the spinal cord level, stimulation of alpha receptors at the substantiagelatinosa of the dorsal horn leads to inhibition of the ring of nociceptive neurons and inhibition of the release of substance P. Alpha 2-adrenoceptors located at the nerve endings have a role in the analgesic mechanisms by preventing Norepinephrine release. The spinal mechanism is the principal mechanism for the analgesic action of Dexmedetomidine. There are studies which show evidence for both a supra spinal and peripheral sites of action [5,6].

Bajwa et al., studied the effect of Clonindine and Dexmedetomidine on patients undergoing surgery under epidural anaesthesia. They noticed an increase in time to two segment regression, sensory and motor block duration and also increase in time to first request of analgesia and better sedation in the Dexmedetomidine group, in the present study we have similar findings. They noticed decrease in HR and SBP in Dexmedetomidine group of patients which is similar in the present study. Patients in Dexmedetomedine group responded to treatment with atropine and Ephedrine and remained haemodynamically stable throughout the study period [12].

Sandip Sinha et al., studied the effect of Dexmedetomidine on paravertebral block using ropivacaine and noticed significant increase in duration of sensory, motor block and also sedation levels, they also noticed bradycardia and hypotension in the Dexemeditomidine group which responded to treatment. Our findings are similar to their study, though blood pressure decreased from baseline in Dexmedetomidne patients, they remained haemodynamically stable throughout the study period [13].

XZ Zeng et al., studied the effect of adding 0.5mics/kg of dexmedetomidine to 0.75% levobupivacaine epidurally in patients undergoing nephrectomy, they noticed that duration of sensory and motor blockade was prolonged in Dexmedetomidine group compared to placebo. Sedation levels, muscle relaxation was significantly better in dexmedetomidine group compared to placebo, our findings are similar to Zeng et al., as we noticed increase duration of sensory blockde, better sedation levels in demedetomidine group [14].

Studies have used dose of 0.5-2mics/kg of Dexmedetomidine, in the present study we have used a lower dose i.e. 0.5 mics/kg as majority of our study population was elderly with associated co-morbidities and a pilot study with 1mics/kg was associated with excessive hypotension. None of the patients in our study had any other significant side effects like, nausea vomiting, rhythm abnormalities or respiratory depression [12-14].

LIMITATIONS

Limitations of our study are that we did not use blinding techniques, we did not measure the plasma concentration of local anaesthetics during the study period. We did not record the PR interval, QT and corrected QT interval in ECG or mechanical parameters like stroke index, acceleration index, ejection fraction during the study period which might have demonstrated differences among the drugs.

CONCLUSION

Above study shows that Levobupivacaine is a safe alternative to Bupivacaine for patients undergoing lower limb vascular surgery, with epidural block characteristics similar to Bupivacaine. Addition of Dexmedetomidine prolongs the duration of anaesthesia and provides excellent sedation and postoperative analgesia and hence, is a good adjuvant to regional anaesthesia in patients undergoing vascular surgery.

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