Original Article

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Supraclavicular Brachial Plexus Block: A Comparative Clinical Study between Bupivacaine and Levobupivacaine

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Abstract

Background: The racemic bupivacaine is commonly used local anaesthetic drug brachial plexus block in as it provides longer duration of action but has risk of cardiotoxicity .The use of levobupivacaine in brachial plexus block seems promising considering its lower toxicity and the need of large volumes in this block. However there is possibility of unsatisfactory motor blockade.

Aim: To compare the efficacy and side effects of bupivacaine and levobupivacaine in brachial plexus block by supraclavicular approach. brachial plexus block

Methods: This study included 60 patients belonging to ASA grade I and II, either sex, between age group of 18 to 65 years and above, undergoing hand, forearm and arm surgery under supraclavicular block. These patients were randomly divided into two groups. The patients received 30 ml 0.5% bupivacaine (Group B) or 30 ml 0.5% levobupivacaine (Group L). Motor and sensory blocks were evaluated. Sensory and motor block onset times, durations of sensory and motor block and duration of postoperative analgesia were recorded.

Results: The two study groups were homogeneous with respect to age, sex, body weight and diagnosis type (type of fractures) and duration of surgery. The sensory and motor block onset times in Group B were significantly shorter than Group L ($p < 0.05$). The onset of sensory block was 9.33 $\pm$ 3.27 min. in group B whereas, it was 16.13 $\pm$ 2.83 min. in group L. The onset of motor block was 12.17 $\pm$ 2.18 in group B, whereas it was 20.00 $\pm$ 2.79 in group L. Sensory and motor block durations of Group B and L patients did not vary statistically significantly. In the present study, the mean duration of post operative analgesia was statistically insignificant between the two group (p= 0.766). It was 193.56 $\pm$ 23.51 in group B and 192.83 $\pm$ 23.54 in group L.

Conclusion:

30 ml 0.5% bupivacaine and levobupivacaine was enough to achieve adequate supraclavicular block. Bupivacaine leads to faster sensory and motor block onset compared to levobupivacaine; however it has similar duration of postoperative analgesia.

Keywords: Supraclavicular Block; Bupivacaine; Levobupivacaine; Cardiotoxicity.

Introduction

The techniques of peripheral neural blockade were developed early in the history of anaesthesia. The brachial plexus block is well-accepted component of comprehensive anesthetic care of upper limb surgeries. It is particularly useful in out-patient anaesthesia, for patients with full stomach, polytrauma and also patients with medical diseases like diabetes and those associated with cardiac, pulmonary, hepatic and renal impairments.

The supraclavicular approach to brachial plexus block provides the most reliable, uniform and predictable anesthesia for surgeries around elbow, forearm and hand [1]. The racemic bupivacaine is most commonly used local anaesthetic as it provides longer duration of action & favorable ratio of sensory to motor neural block [2].

The use of levobupivacaine in brachial plexus block seems promising considering its lower toxicity and the need of large volumes in this block. However there is possibility of unsatisfactory motor blockade, both in neuroaxis and brachial plexus block and there is lack of

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consensus regarding the use of levobupivacaine.

In this study, we investigated whether levobupivacaine with its inherent advantages-anesthetic potency, long duration of action; favorable toxicity profile is superior to bupivacaine in brachial plexus block.

Methods

After the institutional Ethics committee approval, written informed consent was obtained from all the patients. Sixty patients belonging to ASA physical status I&II of either sex and age more than 18 years under going elective orthopedic surgeries of upper limb were selected. The patients were randomly divided into two groups. The exclusion criteria was-uncooperative, obese patients, allergy and sensitivity to local anesthetic drugs, patients with cardiac, neurological, psychiatric disorders, altered coagulation profile and patients with contralateral phrenic nerve palsy and lung pathology. The patients and the observer were blinded to the study drugs.

After a detailed history taking, complete physical examination and routine investigations were undertaken for all patients. All the patients were pre-medicated with Tab. Ranitidine 150mg orally two hours prior to surgery and anxiolysis and sedation was given with Inj. Midazolam 0.02mg/Kg. Monitoring was done using electrocardiography, pulse-oxymeter and non-invasive blood pressure. Intravenous line was secured with 18G canula in large peripheral vein.

The brachial plexus block was performed under all aseptic precautions in supine position using supraclavicular approach by a trained anesthesiologist after eliciting paraesthesia. After negative aspiration, one of the following drug was administered –

Group B received 30 ml. of plain Inj. Bupivacaine 0.5% and *Group L* received 30ml. of plain Inj. Levobupivacaine 0.5%.

Assessment of Block

The sensory block was tested by sensation of pin prick and compared with same area on contralateral arm by using Hollmen scale [3].

The motor block was evaluated by movement at the fingers, wrist, elbow and shoulder joints by using Modified Bromage scale [4].

Onset of Sensory Block

This was defined as minimum of grade II of Hollmen scale in the distribution of any one of the four major nerves.

Onset of Motor Block

This was defined as minimum of grade I of modified Bromage scale.

Recovery from Sensory Block

This was the time at which the patient could perceive the normal sensation of pin prick in all the four major nerve distributions after the block placement (Grade I in Hollmen scale).

Recovery from Motor Block

This was the time at which the patient recovered completely from motor block and was able to do all movements of the limb (Grade 0 in modified Bromage scale).

Duration of Analgesia

It was taken from the time of onset of block to the first complaint of pain. Rescue analgesic was administered in the form of Inj. Diclofenac Sodium IM. In a dose of 1.5mg/kg.

Patients parameters like pulse rate, blood pressure, (systolic, diastolic and mean), $SpO_2$and pain score (VAS) monitored after administration of block and every 5 minutes intraoperatively and post operatively.

The characteristics of block like time for onset, as well as duration of motor and sensory block were noted using Hollmen's scale and Modified Bromage scale. Side effects such as nausea, vomiting, hypotension, bradycardia and sedation were also noted and recorded.

The collected data was compiled in Excel Sheet and master sheet was prepared. For analysis of the data SPSS software version 20th was used. Data was analyzed using chi-square test to check association between two techniques, chi-square test for trend and Fisher Exact test depending on type of data for comparison. $p<0.05$ statistically significant $p>0.05$ statistically not significant.

Results

The two study groups were homogeneous with respect to age, sex, body weight and diagnosis type (type of fractures) and duration of surgery (Table 1). ECG and SpO_2 were maintained throughout surgery in both groups.

The sensory block was assessed by Hollmen scale. In group B mean onset of sensory block was 9.33 ± 3.27min. While in group L, it was 16.13 ± 2.83.

this difference was statistically significant with earlier onset in group B.

The mean onset of motor block in group B was 12.17±2.18 min., while it was 20.00 ± 2.79 min. in group L. This difference was statistically significant with earlier onset in group B.

Table 1: Demography and baseline parameters

Parameter	Group B (n=30)	Group L (n=30)	p -value
Age (years)			
Mean + SD	31.17 ± 8.00	37.30 ± 7.88	0.948 NS*
21-30	6(20.0%)	6 (20.0%)	
31-40	15(50.0%)	14(46.7%)	
41-50	7(23.3%)	8(26.7%)	0.836**
>50	2(6.7%)	2(6.7%)	
Sex			
Male	20(66.7)	13(43.3)	0.076
Female	10(33.3)	17(56.7)	NS***
Weight (kgs) Mean + SD	57.60 ± 4.93	57.20 ± 4.79	0.751 NS*
Diagnosis			
#BBFA	16(53.3)	13(43.3)	
#Galleazzi	6(20.0)	7(23.3)	0.879
#Monteggia	3(10.0)	4(13.3)	NS****
#Radius & #ulna	5(16.7)	6(30.0)	

Value: Number (%) (Otherwise Mentioned)
Fracture
* Unpaired t test, two tailed p value>0.05 Not significant (@95%CL)
** Chi-Square test for trend, two tailed p value>0.05 Not significant (@95%CL)
***Fisher's exact test, p value>0.05 Not significant (@95%CL)
**** Chi-Square test, two tailed p value>0.05 Not significant (@95%CL)

Table 2: Onset, duration of sensory and motor block and duration of analgesia

	Time minutes	Group B (n=30)	Group L (n=30)	p-value
Onset time of sensory block (min)	< 120	0	0	p=1.00 NS**
Onset time of motor block (min)	121-150	6(20.0)	6(20.0)	
Duration of sensory block (min)	151-180	20(66.7)	20(66.7)	
Duration of motor block (min)	181-210	4(13.3)	4(13.3)	
Duration of analgesia (min)	211-240	0	0	
	Mean +SD	165.16 + 15.93	164.00 + 16.16	0.779 NS*

Value: Number (%) (Otherwise mentioned)
* Unpaired t test, two tailed p value>0.05 Not significant (@95%CL)
** Chi-Square test for trend, two tailed p value>0.05 Not significant (@95%CL)

Recovery

Recovery from motor block was, the time at which the patient had achieved grade 0 in modified Bromage scale of motor block. The mean time of recovery from motor block in group B was 165.16 ± 15.93 min. while it was 164.0 ± 16.16 in group L. This difference was statistically not significant. In both the groups, in majority of cases i.e. 20(66.7%) recovery from motor block was between 151-180 min.

Recovery from sensory block was the time at which the patient had achieved grade1 in Hollmen scale of sensory block. The mean time for the recovery of sensory block in group B was 174.90 ± 12.79 min., while it was 175.0 ± 12.41min. In group L this difference was statistically not significant .

Duration of Post-Operative Analgesia

Mean duration of post-operative analgesia in group B was 193.56 ±23 min while it was 192.83 ± 23.54 min. in group L. No significant statistical difference was observed.

Discussion

Brachial plexus block is close to the ideal anaesthesia technique for upper limb surgeries, as it

provides good intra-operative anaesthesia and also post- operative analgesia. Various local anesthetic agents were used in brachial plexus block like Lignocaine, Bupivacaine, Levobupivacaine and Ropivacaine. Lignocaine is very potent having earlier onset but has shorter duration of action, and high incidence of neurotoxicity. Bupivacaine is a popular local anaesthetic for brachial plexus block but is cardiotoxic. We compared bupivacaine with its levorotary isomer i.e. levobupivacaine in supraclavicular brachial plexus block. Levobupivacaine has potency similar to bupivacaine but levobupivacaine has less cardiovascular and central nervous system toxicity as compared to bupivacaine and thus levobupivacaine has a greater safety margin than bupivacaine [5].

This was a prospective, randomized, controlled clinical trial which included 60 patients belonging to ASA grade I and II, either sex, between age group of 18 years and above, undergoing elective upper extremity orthopaedic surgery under supraclavicular brachial plexus block.

In our study, onset of sensory block was 9.33 $\pm$ 3.27 min. in group B whereas, it was 16.13 $\pm$ 2.83 min. in group L. This difference was statistically significant ($p<0.0001$). These findings correlate with the findings of Cenk Ilham et al [6]. The sensory block onset time was 19.64 $\pm$ 10.70 min. in group B, whereas, it was 25.66 $\pm$10.72 min. in group L which was statistically significant ($p<0.036$).In another study, Dr. Charu J Pandya and colleagues [7]compared levobupivacaine 0.5% 0.8ml/kg and bupivacaine 0.5% 0.8ml/kg in supraclavicular brachial plexus block. They observed that the average sensory block onset time was less for levobupivacaine as compared with bupivacaine (10.5 min. Vs 18.7min.).

In the present study, onset of motor block was 12.17 $\pm$ 2.18 in group B, whereas it was 20.00 $\pm$ 2.79 in group L. this difference was statistically significant (0.0001). This means that the latency of motor blockade was more in the levobupivacaine group with earlier onset of motor blockade in the bupivacaine group. In both the groups there was no motor blockade as well as sensory blockade failure rates. Our findings were consistent with Cenk Ilham et al [6] who found that the motor block onset was statistically faster in group B (5.07 $\pm$ 4.07min.) than group L (9.2 $\pm$ 7.9 min). Their findings were statistically significant.

In this study, the duration of sensory block was 174.90 $\pm$ 12.79 min in group B and that in group L was 175.50 $\pm$ 12.41 min. As this difference proved to be statistically not significant ($p>0.05$), both bupivacaine and levobupivacaine had similar duration of sensory block. In the studies done by C.R. Cox et al [9], Baskan et al [10], H. Evten et al [11], Cenk et al [6] they observed no statistically significant difference in the sensory block duration between bupivacaine and levobupivacaine.

The duration of motor block was similar in both the groups which was statistically not significant ($p=0.779$); 165.1 $\pm$ 15.93 min in the bupivacaine group and 164.00 $\pm$ 16.16 min in the levobupivacaine group. Our results matched with the results in the studies done by C.R. Cox et al [9], Baskan et al [10], H. Evten et al [11], Cenk et al [6] and Dr. Charu J Pandya and Colleagues [7]. They all found no statistically significant difference between the motor block duration of the two drugs.

Moreover, our result showed that sensory block tended to last longer as compared to motor block. It is shown that Ropivacaine has a more selective action on pain transmitting A delta and C fibers rather than A beta fibers being large fibers. The minimal effective concentration of local anaesthetic for large (motor) fibers is greater than for small (sensory) fibers. Thus, motor function returns before pain perception and hence duration of motor block was shorter than the sensory block.

In the present study, the mean duration of post operative analgesia was statistically insignificant between the two group ($p=0.766$). It was 193.56 $\pm$ 23.51 in group B and 192.83 $\pm$ 23.54 in group L. Our results were consistent with the findings of Cenk et al [6] who had similar observations.

The immediate side effect like drowsiness, pruritus, respiratory depression, arrhythmias, hypotension, bradycardia, perioral numbness, convulsions, cardiac arrest were not seen in our study. Later complications associated with supraclavicular brachial plexus block techniques like haematoma, pneumothorax, recurrent laryngeal nerve palsy, phrenic nerve palsy, Horner's syndrome etc were also not observed in our study.

Local anaesthetic toxicity is an uncommon but well documented complication of regional anaesthesia. To reduce its occurrence, frequent aspiration and slow fractionalized injection with strict adherence drug dose schedule are recommended. Despite following these recommendations, cardiovascular toxicity may be unavoidable.

Several studies have been demonstrated and explained the mechanism of toxicity of bupivacaine [12,13]. Bupivacaine has been shown to have indirect depression of cardiac conduction (AV conduction, QRS complex) and contractility by blocking mainly inactivated state of sodium channels [13-15]. Studies

demonstrate dextro (R+) enantiomer has 2.4 times higher affinity for cardiac sodium channels and dissociates from it slowly as compared to levo (S+) enantiomer [13-15]. Levobupivacaine cause less rapid blockade of the cell firing in nucleus tractus solitarius (NTS) [13] which explains its lower CNS toxicity compared to racemic one. Also one more factor for difference in toxicity between two enantiomers can be explained on the basis of their pharmacokinetics. The protein binding of levobupivacaine is >97% as 95 against % in case of bupivacaine. That means <3% of levobupivacaine is free in plasma to have action on other tissues causing undesired toxic effect [16].

Thus levobupivacaine exhibits a wide margin of safety, less cardiodepressant activity which offers advantages over the currently used long acting agents like bupivacaine. Enough precautions were taken to use recommended dose and inject the anaesthetic in slow fractionalized doses. In our study, no side effect was observed related to local anaesthetic toxicity.

The major limitation of our study was that we did not use ultrasound guided blocks because of unavailability at the time of our study, this could have helped us to lower dosage and volume of local anaesthetic. Another limitation of the present study was the small number of cases. Though our results tend to suggest that levobupivacaine is a longer acting local anaesthetic with similar block quality and prolonged effect as that of bupivacaine, to obtain a definite result, study with enrolment of larger number of patients is required. Moreover, we included only patients with ASA I and II physical status only, a study of high risk patients to justify the safety of levobupivacaine has to be carried out.

Thus, we conclude that, 30ml of 0.5% bupivacaine and levobupivacaine was enough to achieve adequate motor and sensory supraclavicular block. Levobupivacaine has theoretical advantage of having less toxicity potential and being less cardiotoxic than bupivacaine. So, it may be safer drug in supraclavicular brachial plexus block; where accidental intravascular injection of large volume of local anaesthetic can occur and may be more detrimental especially in patients with cardiac disease.

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