Original Article

Comparison of Alkalinized and Non-Alkalinized Lignocaine in the Brachial Plexus Block

Anaesthesia Section

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ABSTRACT

Introduction: Interruption of pain is central to the anaesthetic practice. Regional anaesthesia is one of the widely practiced ways for mitigating surgical pain. Many long acting local anaesthetics have been tried, viz.. bupivacaine, ropivacaine … but they are limited by the drawbacks of delayed onset, the varying quality of the blockades and their unpredictable duration of action. The alkalinization of lignocaine has been shown to fasten the onset, potency and the duration of the block.

Type of study: Randomized single blind clinical trial.

Materials and Methods: After obtaining the institutional ethical committee clearance and their written informed consent, fifty patients who were aged between 25-50 yrs, of either sex, who belonged to ASA grade 1 and 2, who were posted for elective/ emergency surgery of the upper limb were enrolled for the study. Group 1 received 30ml of 1.5% lignocaine with adrenaline and Group 2 received 30 ml of 1.5% alkalinized lignocaine with adrenaline. A supra-clavicular block by a classical approach was made and the degree of the blockade was graded. The results were tabulated and analyzed by using appropriate statistical tests.

Conclusion: Alkalinization of lignocaine offers an earlier onset and it provides a good intensity and adequate depth and a satisfactory distribution of the regional block.

Key Words: Anesthesia, Local block

INTRODUCTION

The central axis of anaesthesia is predicated by the interruption of pain. Over a period of several years, many local anaesthetic agents have evolved viz, bupivacaine, ropivacaine, etc .The main drawbacks of these long acting drugs are their delayed onset of action, the varying quality of the blockade and their unpredictable duration of action. To overcome these drawbacks, the following have been tried viz. the addition of enzymes, oils, alkalinization, potassium, glycol and vaso-constricting agents, warming up of the local anaesthetic solutions, potentiation of the blockade by pain and muscular exercises.

Out of all the above, alkalinization has been shown to hasten the onset, the potency and the duration of the block [1]. Hence, in our study, an attempt was made to evaluate the effect of alkalinized lignocaine with respect to the onset and the degree and duration of the blockade in the supraclavicular brachial plexus block.

MATERIALS AND METHODS

After obtaining the institutional ethical committee clearance and their written informed consent, fifty patients in each group who were aged between 25-50 yrs, of either sex, who belonged to ASA grade 1 and 2, who were posted for elective surgery of the upper limb, were enrolled for the study. The exclusion criteria were renal, liver and neurological disorders and a history of an allergic reaction to local anaesthetics.

All the patients were investigated by assessing their haemoglobin levels, random blood sugar, serum electrolytes, serum creatinine, urine albumin, chest X-ray and ECG. The study population was randomly assigned into two groups by using a randomization table.

Group 1 received 30 ml of 1.5% lignocaine with adrenaline and Group 2 received 30 ml of alkalinized 1.5% lignocaine with adrenaline for the block.

30 ml of 1.5% lignocaine with adrenaline (for group 1) was prepared with 22.5 ml of 2% lignocaine, 1.5 ml of adrenaline (1 ml=100 microgram diluted), and 6 ml of normal saline. This solution was tested for pH, which revealed a pH of 6.38.

30 ml of alkalinized 1.5% lignocaine (for group 2) was prepared by adding 3.5 ml of 7.5% sodium bicarbonate 1.12 ml=1 mEq), 1.5 ml of adrenaline (1 ml=100 microgram diluted), 2.5 ml of normal saline and 22.5 ml of 2% lignocaine. Finally, the prepared solution was tested for pH, which revealed a pH of 7.25.

A supra-clavicular block by using a classical approach was made and the degree of the blockade was graded [2] as follows;

Proximally

- Grade-0 Difficulty in elevating the arm off the table (paresis)
- Grade-1 Inability to move the arm at all (paralysis).

Distally

- Grade-0 No motor block
- Grade-1 Paresis
- Grade-2 Paralysis (of hand was determined by checking for inability in flexing the wrist against gravity and movements)

The person who performed the procedure was blinded with regards to the drug which was used. The study drug which was to be used was selected based on a computer generated randomization. The onset and the spread of the sensory and motor blockade were assessed every minute after injecting the local anaesthetic solution.

The sensory block was assessed by checking for a pin prick sensation in the axillary, median and the ulnar distribution areas.

The heart rate, ECG and SpO_2 were monitored continuously and the noninvasive blood pressure was recorded every 5 minutes. The patients were watched for bradycardia (Heart rate <60), convulsions, drowsiness and any other complications.

The onset of the sensory and the motor blockade was determined proximally and distally to allow the differentiation of their onset in the mantle and core fibers. Proximally, the onset of analgesia and anaesthesia was determined over the lower deltoid muscle in the distribution of the axillary nerve and distally, over the thumb and the little fingers in the distribution of the median and ulnar nerves respectively.

The onset of the motor blockade was determined proximally in the deltopectoral group of muscles and distally in the muscles of the hand to allow the differentiation of the onset in the mantle and core fibers.

Definitions

Analgesia: The time of injection to the time of onset of the loss of pain on pinprick

Anaesthesia: The time of injection to the time of onset of the loss of touch and pressure by pin prick.

Paresis: Grade 1, The time of injection to the time of onset of the loss of the motor power (partial motor block)

Paralysis: Grade 2, The time of injection to the time of complete motor loss (complete motor block)

Penetration time: The time of injection to the time of the proximal paresis.

Intra-neural-diffusion: The time of the proximal paresis to the time of the complete distal sensory block.

Duration of sensory blockade: The time in minutes from the onset of analgesia to the recurrence of the pain to pinprick at the proximal and the distal levels was noted.

Duration of motor blockade: The time in minutes from the onset of the paresis to the recurrence of the motor movements at both the proximal and the distal groups.

RESULTS AND OBSERVATION

Both the groups (50 in each group) were compared for any difference in height, weight, age, sex, distribution, time of onset, duration and the degree of the blockade. The statistical analysis consisted of the 1) Two tailed student t test and the 2) Z test. A p value of less than 0.05 ($P < 0.05$) was considered as statistically significant, In the Z test, a value of more than 1.96 ($Z>1.96$) was considered to be significant.

[Table/Fig-1] shows the sex distribution of the study subjects. [Table/Fig-2] shows their age, height and weight (mean ± S.D). [Table/Fig-3] shows the onset of the sensory blockade (mean ± S.D in minutes). [Table/Fig-4] shows the onset of the motor blockade (mean ± S.D in minutes). [Table/Fig-5] shows the duration of the sensory and the motor blockade (mean ± S.D in minutes). [Table/Fig-6] shows the rate of penetration and diffusion (mean in minutes).

DISCUSSION

Brachial plexus blocks are widely used for upper limb surgeries. A supraclavicular approach for a brachial block results in a homogenous blockade. Lignocaine is an amide local anaesthetic which is widely used because of its shorter latency, but it has the disadvantage of a shorter duration of action.

Morrisson D H suggested that a volume of 0.05 ml/2.45 cm^2 of height [3], to give this volume with 2% lignocaine with adrenaline toxic limits would exceed. With a 1% lignocaine solution, the dose would be highly inadequate to provide good analgesia and motor blockade. Hence, in our study, we used an optimal concentration of 1.5% lignocaine with adrenaline to give an adequate volume dosage within the therapeutic range.

The present study assessed the effect of the addition of 3.5 ml of 7.5% sodium bicarbonate to 22.5 ml of 1.5% lignocaine. Normal saline was added to make the volume final to 30 ml, which produced the lignocaine strength of 1.5%. Fresh adrenaline was added to the solution to make the lignocaine 1.5% with adrenaline 1:200000 (5 microgram/ml) solution. The present study was aimed at bringing

Male	Female	Total
Group 1	18	7 25
Group 2	16	9 25

[Table/Fig-1]: Sex distribution
P value > 0.05 statistically not significant

	Age (years)	Height (cms)	Weight (kgs)
Group 1	34.08 ± 6.54	158.96 ± 4.57	57.44 ± 5.56
Group 2	31.04 ± 7.87	160.52 ± 5.09	58.20 ± 6.33

[Table/Fig-2]: Mean age, height and weight distribution ± S.D (Standard Deviation)
P > 0.05 statistically not significant

	Analgesia		Anaesthesia		
	Group 1	Group 2	Group 1	Group 2	Z value
Axillary	6.24 ± 0.091	3.12 ± 0.63	8.24± 1.23	4.4 ± 0.71	6.1495
Median	7.48 ± 0.74	4.08 ± 0.69	11.52 ± 1.85	6.36 ± 0.99	6.0715
Ulnar	9.04 ± 1.42	4.52 ± 0.75	14.68 ± 1.6	9.00 ± 1.44	6.0753

[Table/Fig-3]: Onset of sensory blockade ± SD (in minutes)
P < 0.0001 statistically very highly significant

	Paresis		Paralysis		
	Group 1	Group 2	Group 1	Group 2	Z value
Proximal	4.88 ± 0.69	2.88 ± 0.45	7.16 ± 0.99	3.72 ± 0.61	6.1835
Distal	6.92 ± 0.92	4.00 ± 0.52	17.08 ± 1.98	10.24 ± 1.52	6.0978

[Table/Fig-4]: Onset of motor blockade (in minutes)
P< 0.0001 statistically very highly significant

	Sensory	Motor
Group 1	131 ± 11.9	38 ± 9.19
Group 2	132 ± 19.58	139.2 ± 18.01
Z value	0.4902	0.3523

[Table/Fig-5]: Duration and sensory and motor blockade ± S .D (in minutes)
P > 0.05, statistically not significant

	Penetration	Diffusion
Group 1	4.88	9.8
Group 2	2.28	6.72

[Table/Fig-6]: Rate of penetration and diffusion (in minutes)

the pH of lignocaine towards the pka value without changing the preparation.

The alkalinization of lignocaine hydrochloride led to a higher pH of > 6 and thus it was less dependent on the buffering capacity of the tissues. On injecting the alkalinized solution, the free base was liberated, the carbon dioxide rapidly diffused into the axon interior and the pH fell, which helped in the dissociation of the local anaesthetic to the active cationic form. This effect resulted in ion trapping, hence favouring the rapid movement of the local anaesthetic into the axon.

A tenfold increase in the degree of the block was observed with alkalinized lignocaine as compared to the hydrochloride salt by Catchlov et al [4] in vivo solutions.

In our study, the mean onset time of analgesia in the shoulder was 3.12 minutes, in the hand ulnar nerve area, it was 4.08 minutes and in the median nerve area, it was 4.52 minutes. In the control group, the mean onset time of analgesia in the shoulder was 6.24 minutes, in the hand ulnar region, it was 9.04 minutes and in the median region, it was 7.48 minutes.

The complete sensory blockade with the study group in the shoulder occurred within 4.4 minutes, in the hand (median), it occurred within 6.3 minutes and in the hand (ulnar), it occurred within 9 minutes . In the control group, a complete sensory blockade in the shoulder occurred after 8.24 minutes, in the hand (median), it occurred after 11.52 minutes and in the hand (ulnar), it occurred after 14.68 minutes.

The onset time for the motor block in the study group was only 2.28 minutes (proximal) and 4 minutes (distal). Complete paralysis occurred within 3.72 minutes (proximal) and 10.34 minutes (distal). In the control group, the onset time (paresis) was 4.88 minutes (proximal) and 6.92 minutes (distal). A complete motor blockade in the proximal area took more than 7.16 minutes and in the distal area, it took 17.08 minutes.

The average duration of the sensory blockade in the study group was 132 minutes as compared to 131 minutes in the control group and that of the motor blockade in the study group was 139.2 minutes and it was 138 minutes in the control group. So, in this study, the duration was nearly the same and it was statistically insignificant.

With alkalinization, the efficacy of the blockade improved significantly in the patients as compared to that in the control group. In the study group, 21 out of 25 patients achieved complete motor blockade (distal paralysis) and the depth of the sensory blockade was adequate, which reduced the need of adjuvants intraoperatively. In the control group, only 8 patients out of 25 achieved complete distal paralyses.

In 1986, Radha Sukani and Alon P Winnie [5] carried out a study which compared alkalinized and non-alkalinized lignocaine in the brachial plexus blockade by using a supra-clavicular technique. They found that alkalinized lignocaine reduced the latency by 45% as compared to non alkalinized lignocaine and that it produced a complete motor block in almost twice as many patients as non alkalized lignocaine (54% vs 31%). The duration of anaesthesia which was provided by the two agents was virtually identical, as was the motor blockade.

In 1995, Giorgio and Capogna et al [1] found that using alkalinized lignocaine in the axillary brachial plexus block produced a faster onset and an earlier peak effect time and the onset of the motor block. Similar results were observed in a study which was conducted by A V Gormley W F et al in 1996.

The present study confirmed the results of Radha Sukani and Alon Winnie, W P Gormly et al and Giorgio Capogna et al regarding the onset, the degree of the blockade and its duration.

However, a study which was conducted by D G Ririe et al [6] on the effect of alkalinization of lignocaine on the median nerve block, observed an increased rate of motor block without any change in the onset or extent of the sensory block. In another study which was conducted by Ruby et al [7] to study the effect of the alkalinization of lignocaine hydrochloride on brachial plexus blocks, it was observed that alkalinization reduced the latency of the sensory and motor blockade and that it also increased the duration of the blockade.

Mark et al [8] observed that alkalinization did not have any significant effect and that it provided no clinical advantage.

In a recent study which was done by Jaichandran V et al, it was found that alkalinization also decreased the pain on injection of the local anaesthetic for the peribulbar block [9].

No drug related or technique related complications were observed during the study.

CONCLUSION

The alkalinization of lignocaine offers an earlier onset and it provides good intensity and adequate depth and a satisfactory distribution of regional block. There was not much difference in the duration of the blockade. The need of a intraoperative adjuvant was significantly reduced with the use of alkalinization.

REFERENCES

[1] Capogna G, Celleno D, Costantino P, Muratori F, Sebastini M, Baldassini M. Alkalinization improves the quality of lidocaine fentanyl epidural anaesthesia for caesarean section. *Can J Anaesth* 1993; 40:425-30.
[2] AV Gormley WF, Hill DA, Murray JM, Fee JP. The effect of the alkalinization of lignocaine on axillary plexus anaesthesia. *Anaesthesia* 1996;51(2):185-88.
[3] Morrison DH Editorial. Alkalinization of local anaesthetics. *CSA* 1995; 42 (12):1076-79
[4] Catchlov, Richard FH. The influence of CO2 and pH on the local anaesthetic action. *J Pharmacol.Exp.Ther* 1972; 181 : 298-309.
[5] Sukani R, Winnie AP. The clinical pharmacokinetics of alkalinized local anaesthetics. A subclavian perivascular model. *Anaesth Analg* 1987; 66:739-45.
[6] Ririe DG, Walker FO, James RL, Butterworth J. Effect of the alkalinization of lidocaine on the median nerve block. *Br J Anaesth* 2000; 84: 163-68.
[7] Mehta R, Verma DD, Gupta V, Guruwara AK. To study the effect of alkalinizatiion of lignocaine hydrochloride on the brachial plexus block *Indian J Anaesth* 2003;47 :283-86.
[8] Chow MYH, Sia ATH, Koay CK, Chan YW. Alkalinization of lignocaine does not hasten the onset of the axillary plexus block. *Anaesth Analg* 1998; 86:566-8.
[9] Jaichandran V, Vijaya L, George RJ, Indermohan B. Peribulbar anesthesia for cataract surgery : Effect of Lidocaine warming and alkalinization on injection pain and motor and sensory blockade. *Indian J Ophthalmol*. 2010 Mar-Apr; 58(2):105-8.

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