Original Research Article

Indian Journal of Anesthesia and Analgesia
2018;5(5): 829-835
DOI: http://dx.doi.org/10.21088/ijaa.2349.8471.5518.22

Comparison between Effects of 0.5% Bupivaciane with Dexmedetomidine and 0.5% Bupivaciane Alone in Brachial Plexus Block

Sagar S.[1], Vinayak N. Panchgar[2], Shivaraddi Bhandi[3], Harish Nadagoudar[4]

[1]Assistant Professor, Dept. of Anaesthesiology, Apollo Institute of Medical Sciences & Research, Hyderabad, Telangana 500033, India. [2]Assistant Professor [3]Senior Resdent [4]DNB Resident, Dept. of Anaesthesiology, Gadag Institute of Medical Sciences, Gadag, Karnataka 582103, India.

Abstract

Background: Brachial plexus block is a popular and widely employed regional nerve block of upper extremity which avoids the unwanted effect of anesthetic drugs. It has been proved in numerous studies that dexmedetomidine is harmless and effecient in several spinal, epidural and regional anaesthesia in humans. Less exploration of dexmedetomidine in regional blocks was the reason for study. *Objectives:* To determine efficacy of anesthesia and analgesia between dexmedetomidine-bupivacaine and bupivacaine-alone in brachial block. *Methods:* After institutional ethics committee approval and patient consent, 70 Upper limb surgery patients, aged 18-60 years; ASA physical statuses 1 and 2were randomized into 2 groups. Group I and II received bupivacaine 0.5%+ 1ml Normal saline, bupivacaine 0.5% 15ml+ 100 mcg dexmedetomidine+ 15ml lignocaine+adrenaline, respectively. A pre-anesthetic checkup was done for all patients, which will include a detailed history, general physical and systemic examination. *Results:* There was no statistical difference in the heart rate and BP at the start of the procedure. Mean heart rate recorded in study group at 15minutes, 30minutes, 45minutes, 60minutes and 90minutes was statistically significant compared to control group. Mean BP recorded was significantly lower in study group at 15 min, 45 min, 60 min durations in comparison to control group, however it was similar in both the groups at the end of the procedure. *Conclusion:* In conclusion, dexmedetomidine added to 0.5% bupivacaine and 2% lignocaine + adrenaline is an attractive and safe option for improving the duration and quality of supraclavicular brachial plexus block in upper limb surgeries.

Keywords: Dexmedetomidine; Bupivacaine; Brachial Plexus block; Upper Limb Surgeries.

Introduction

Brachial plexus block is a popular and widely employed regional nerve block of upper extremity which avoids the unwanted effect of anesthetic drugs used during general anesthesia and the stress of laryngoscopy and tracheal intubation [1]. It's a great anesthetic tool for relief of pain preoperatively, intraoperatively and postoperatively. Brachial block has undergone many changes and modifications since its inception by William Steward Halsted in 1885 who performed the block by exposing its roots [2].

There has always been a search for adjuvants to the regional block with drugs that prolongs the period of analgesia with reduced side effects [1]. Many drugs have been tried like opiods, clonidine, buprenorphine, dexamethasone, midazolam [3-6].

$Alpha_2$ adrenergic receptor agonists have been tried either alone or in combination with another drug to prolong anaesthesia in various methods of anaesthetic administration like epidural, intrathecal and regional nerve blocks. Dexmedetomidine is an $Alpha_2$ receptor agonist which is more selective for $Alpha_2$ receptor (8 times) as compared to clonidine [7]. It has been proved in numerous studies that dexmedeto-midine is harmless and efficient in

Corresponding Author: Vinayak N. Panchgar, Assistant Professor, Dept. of Anaesthesiology, Gadag Institute of Medical Sciences, Gadag, Karnataka 582103.
E-mail: vinayak.panchagar@gmail.com

Received on 01.02.2018, **Accepted on** 26.02.2018

several spinal, epidural and regional anaesthesia in humans including IV regional anaesthesia with lesser side effects [7-9].

In previous studies it has been proven that dexmeditomidine resulted in significant opoid sparing and reduced inhalational anesthetic requirements [10].

Our main aim was to study the effects of addition of dexmedetomidine to bupivacaine in supraclavicular brachial block and also to determine the duration of sensory and motor block and also the duration of postoperative analgesia following surgery.

Objectives

The aim of the present study is to evaluate the effect of 0.5% bupivaciane with dexmedetomidine and 0.5% bupivaciane alone in brachial plexus block.

Material and Methods

After the approval from institutional ethics committee, 70 adult patients fitting to ASA I and II undergoing upper limb surgeries, under brachial plexus block were selected. This was a prospective randomized controlled study was done at KMC Hospitals, Mangalore and Govt. Wenlock Hospital. Pre-operative evaluation was done & consent was taken from patients. The study period was from January 2014 to July 2015.

Patients were distributed into two groups of 35 each: Group-I - Patients receiving 0.5% bupivacaine (15ml)+2% lignocaine with adrenaline (15ml)+ normal saline(1ml), Group- II - Patients receiving 0.5% bupivacaine (15ml)+2% lignocaine with adrenaline (15ml) + Dexmedetomidine (100mcg or 1ml).

The sample size was calculated based on the formula

$$\frac{2(Z_\alpha+Z_\beta)\ X\ \sigma^2}{d^2}$$

Z_σ = 1.96 (95% confidence level)

Z_β = 1.28 (90% power)

35 X 2 = 70 samples

Inclusion Criteria

1. Patients for surgeries on upper limb (i.e. elbow, forearm and hand surgeries.)
2. ASA(American Society of Anaesthesiologists) grade I and II
3. Between the ages of 18-60years, of either sex
4. Weight between 40-90 kgs

Exclusion Criteria

Patients with:

1. Patient refusal
2. Known hypersensitivity to local anaesthetics
3. Uncontrolled diabetes mellitus
4. Patients with local infection at the site of block
5. Patients on adrenoreceptor agonist and antagonist therapy
6. Preexisting peripheral neuropathy
7. Patients with bleeding disorder
8. Pregnant Women
9. ASA III and above

Methodology

After approval of institutional ethical committee, 70 consenting patients fulfilling the inclusion criteria was considered for our study. A pre-anesthetic checkup was done for all patients which includes a detailed history, general physical and systemic examination. Basic investigations was done (Hb%, complete blood counts, bleeding time, Clotting time, random blood sugar, serum urea, serum creatinine, if age is above 45yrs then ECG). Patients were kept nil per oral overnight.

Selected patients were divided into two equal groups randomly

Group I: Patients receiving 0.5% bupivacaine (15ml)+ 2% lignocaine with adrenaline (15ml) + normal saline (1ml).

Group II: Patients receiving 0.5% bupivacaine (15ml)+2% lignocaine with adrenaline (15ml)+ Dexmedetomidine (100mcg or 1ml)

On arrival in the operation theatre, baseline HR, BP and SpO_2 was recorded. An I.V. line was secured in the non operating limb and I.V. fluid (RL) was started. Under aseptic precautions all the patients were receiving brachial block via supraclavicular

approach by a skilled anesthesiologist different from the one assessing the patient intra and postoperatively. Both were blinded to the treatment groups. Neural localization was achieved using nerve stimulator with a 22G one inch short beveled, insulated (Teflon coated) stimuplex needle. Frequency of stimulation was fixed at 2Hz, while the power of current was at first set to deliver 2 mA and then gradually reduced to 0.6 mA. Following negative aspiration, the solution containing local anaesthetic combined with dexmedetomidine or normal saline as mentioned above was injected.

Sensory block will be evaluated by doing pinprick test by using 3-point scale.

0 Normal sensation

1 Loss of sensation of pinprick (analgesia)

2 Loss of sensation of touch (anasthesia)

Motor blockade will be done using Bromage three point score

1 Normal sensation

2 Reduced motor strength with capacity to move fingers only

3 Complete motor block with failure to move fingers

Sensory and motor blocks was checked every 3min till 30min once injection is given

Onset of action: Sensory & Motor blockade:

Sensory block: The time interval between the administrations of local anesthetic solution to loss of pin prick sensation.

Motor block: The time interval between administration of local anesthetic solution to loss of movements.

Duration of Blockade

Sensory Block: Time interval between loss of pin prick sensation to appearance of pin prick sensation.

Motor Block: Time interval between loss of movements to appearance of the movements

HR, Mean blood pressure (MBP) and saturation of Oxygen (SpO_2) was recorded at 0 min, 15 min, 30min, 45min, 60min and every half an hour thereafter till the end of surgery. Adverse events like hypotension (20% decrease in relation to baseline) and bradycardia (heart rate <50 beats per min) was corrected with appropriate measures. The onset and duration of sensory loss and motor blockade was studied. The loss of pinprick sensation was checked every 3 minutes till the onset of loss of sensation and then every hourly till 4hours post op and every 2nd hourly thereafter till the regain of sensation.

The motor blockade was assessed every 3 minutes till the loss of movements and then every hourly till 4hours post op and every 2nd hourly thereafter till the regain of movements. The time between the end of local anesthetic administration and request for first analgesic was recorded as duration of analgesia

Rescue analgesia diclofenac sodium 75mg IM was given when patient visual analogue score > 4

Visual analog scale:

Pain Intensity	Word Scale
0	No pain
1-2	Least pain
3-4	Mild pain
5-6	Moderate pain
7-8	Severe pain
9-10	Excruciating pain

Statistical Analysis

Descriptive statistics such as mean, SD and percentage was used. The data was analysed by using Students unpaired t test for continues data and Chi-square test for discrete data. A p-value of < 0.05 was taken as significant.

Results

This study was conducted on ASA1 and ASA2 patients within the age group of 18-60 yrs posted for various upper limb surgeries. The prospective randomized comparative study included a total of

Table 1: Comparison of Age and Weight between group I and II

Parameters	Group-I	Group-II	Mean difference	t-value	p-value	Remarks
Age (years)	39.31 ± 13.14	36.91 ± 11.89	2.40	0.80	0.43	NS
Weight (Kg)	58.91 ± 9.687	63.0 ± 8.957	4.09	1.83	0.071	NS

70 patients allocated into 2 groups with 35 patients in each group.

The mean age of the study population was 36.91 years as compared to 39.31 years in the control population. This was found to be statistically not significant (p = 0.426).

The mean weight of patients in study group was 63.0 as compared to 58.91 in the control group. There was statistically no difference in weight among the groups (p = 0.071) (Table 1).

In the study population, 14 (40.0%) were females and 21 (60.0%) were males as compared to 16 (45.7%) were females and 19 (54.3%) were males in the control population. This was found to be statistically not significant (p = 0.629) (Table 2).

The mean onset of sensory block was 11.40 minutes in the study population as compared to 17.31 minutes in the control population. This was found to be statistically significant (p<0.0001). The mean time of onset of motor blockade in the study population is 14.40 minutes as compared to 21.34 minutes in the control group. This was found to be statistically significant (p < 0.0001) (Table 3).

There was no statistical difference in the heart rate at the start of the procedure. Mean heart rate recorded in study population at 15minutes, 30minutes, 45minutes, 60minutes and 90minutes was statistically significant compared to control group. (Table 4).

There was no statistical difference in mean BP at the start of the procedure. Mean BP recorded was significantly lower in study group at 15 min, 45 min, 60 min durations in comparison to control group however it was similar in both the groups at the end of the procedure. (Table 5).

Table 2: Gender distribution

	Group		Total
	Control	Study	
Sex			
Female	16 (45.7%)	14 (40.0%)	30 (42.9%)
Male	19 (54.3%)	21 (60.0%)	40 (57.1%)
Total	35 (100.0%)	35 (100.0%)	70 (100.0%)

X^2= .233 p=0.629, NS

Table 3: Time of onset of sensory blockade and motor blockade

Parameters	Group-I	Group-II	Mean difference	t-value	p-value	Remarks
Time of onset of sensory block	17.31 ± 2.069	11.40 ± 2.032	5.91	12.07	<0.0001	Significant
Time of onset of motor block	21.34 ± 2.496	14.40 ± 2.032	6.94	12.76	<0.0001	Significant

Table 4: Intra-operative Mean Heart rate

Heart Rate At	Group-I	Group-II	Mean difference	t-value	p-value	Remarks
0 min	87.29 ± 8.62	83.03 ± 10.689	4.26	1.834	0.07	NS
15 min	85.26 ± 8.169	79.60 ± 11.133	5.66	2.424	0.018	Significant
30 min	84.74 ± 7.457	78.80 ± 11.36	5.94	2.587	0.012	Significant
45 min	85.77 ± 7.248	79.77 ± 9.613	6.0	2.948	0.004	Significant
60 min	85.94 ± 8.167	80.23 ± 10.304	5.71	2.571	0.012	Significant
90 min	87.58 ± 8.097	81.79 ± 8.587	5.79	2.902	0.005	Significant

Table 5: Intra-operative Mean BP

BP At	Group-I	Group-II	Mean difference	t-value	p-value	Remarks
0 min	95.91 ± 7.386	94.20 ± 6.053		1.062	0.292	NS
15 min	94.34 ± 7.522	89.20 ± 6.033		3.155	0.002	Significant
30 min	91.77 ± 15.99	89.86 ± 6.761		0.652	0.516	NS
45 min	94.74 ± 7.163	90.0 ± 6.911		2.819	0.006	Significant
60 min	94.66 ± 7.889	90.26 ± 6.887		2.486	0.015	Significant
90 min	93.74 ± 7.706	91.53 ± 5.894		1.182	0.243	NS

Table 6: Comparison of various parameter duration between groups (Mean ± SD)

Parameter	Group-I	Group-II	Mean difference	t-value	p-value	Remarks
Duration of surgery	97.29 ± 12.128	97.60 ± 10.712	0.31	0.19	0.851	NS
Duration of tourniquet	86.83 ± 11.124	87.69 ± 9.908	0.86	0.34	0.735	NS
Duration of sensory blockade	369.89 ± 65.910	633.23 ± 57.549	263.34	17.81	<0.0001	Significant
Duration of motor block	317.49 ± 53.717	545.80 ± 50.267	228.31	18.36	<0.0001	Significant
Duration of analgesia	395.03 ± 49.090	683.00 ± 60.450	287.97	21.88	<0.0001	Significant

The mean duration of surgery in study group was 97.80 mins as compared to 97.29 mins in the control population. This was found to be statistically not significant (p=0.851).

Mean duration of tourniquet in study group is 87.69 mins as compared to 86.83 mins in control group. This was found to be statistically not significant (p=0.735).

The mean duration of sensory block was 633.23 min in study group as compared to 369.89 min in control group. This was found to be statistically significant (p= 0.00).

The mean duration of motor block in the study group was 545.80 mins as compared to 317.49 min in control group. This was found to be statistically significant (p= 0.00).

The mean duration of analgesia was found to be 683.00 min in the study group as compared to 395.03 min in control group. This was found to be statistically significant (p= 0.00). (Table 6).

Discussion

α-2-adrenergic receptor (α2AR) agonists have been the center of interest for their sedative, analgesic, perioperative sympatholytic, anaesthetic sparing, and hemodynamic-stabilizing properties [11]. Dexmedetomidine is a extremely selective α2AR agonist with a relatively increased ratio of α2/α1-activity (1620:1 as compared to 220:1 for clonidine) and it possesses all these properties but lacks respiratory depression [12,13], making it a useful and safe adjunct in diverse clinical applications [14].

Dexmedetomidine has also been used safely as an additive to peripheral nerve blocks. Several animal and human studies have been done.

A study conducted by Brumett et al. [15] proved that dexmedetomidine will increase the duration of analgesia and anesthesia when added to bupivacaine for sciatic nerve block in rats, devoid of any injury to the nerve. Another study conducted by the same authors showed that clinically applicable doses of dexmeditomidine increased block after adding to ropivacaine [16].

Sandhya A et al. [11] in 2014 conducted a study to assess the effect of addition of dexmedetomidine 100 μg to 0.325 % bupivacaine. This was compared with 0.325% bupivacaine + 1ml normal saline. Both groups received brachial plexus block via supraclavicular approach. They found that addition of bupivacaine lead to shorter onset times of motor and sensory blockade. The period of analgesia was significantly prolonged in the dexmedetomidine group. It was also noted that the intra-operative heart rate and BP was considerably lesser in the dexmedetomidine group compared to the other group.

The mechanism of action by which α-2-adrenergic receptor agonists will produce analgesia and sedation is multifactorial. These include vasoconstriction around the injection site, direct suppression of impulse propagation through neurons due to interaction with axonal ion channels or receptors, a decrease in local inflammatory mediators and an increase in anti- inflammatory cytokines through an α-2-adrenergic receptor mediated mechanism [13] .

In our study, we compared the addition of dexmeditomidine (100 μg/ 1ml) to 15 ml 0.5% bupivacaine + 15 ml 2% Lignocaine with adrenaline in the study group and normal saline (1ml) to 15 ml 0.5% bupivacaine + 15 ml 2% Lignocaine with adrenaline in the control group.

The outcome of the study showed that there were no noteworthy differences between the two groups with regard to age (mean) 36.91yrs in the study group as compared to 39.31 yrs in the control group. There was also no difference with regard to sex distribution, weight, baseline HR and BP in the groups as also with regard to the duration of surgery. This shows that the samples were equal in all the baseline variables expected.

In our study, the mean onset of sensory block for the study group was 11.4±2.032 as compared to 17.31±2.069 in the control group (p=0.00). This was found to be statistically significant. This finding correlates with the findings of Sandhya A et al. [11],

mean onset time for sensory blockade 13.2±1.8,. This also confirms the findings of Saritha et al. [1], Amany et al. [13], Aliye Esmaoglu, et al. [7] that the addition of dexmedetomidine in the brachial plexus block shortens the mean onset of sensory blockade.

Sandhya A et al. [11] showed that study group receiving 30 ml of 0.325% bupivacaine + 1ml (100µg) dexmedetomidine had complete motor block at 16.2±1.8 min and control group receiving 0.325% bupivacaine+1ml normal saline had complete motor block at 22.7±2.8 min concluding that time taken for complete motor blockade was earlier in study group than control group.

In the present study, study group received 15 ml 0.5% bupivacaine+15 ml 2% lignocaine+ adrenaline with 1ml (100 µg) dexmedetomidine. Control group received 15 ml 0.5% bupivacaine+2% lignocaine+adrenaline with 1ml normal saline. Study group had complete motor blockade at 14.4±2.032 min. Control group had complete motor blockade at 21.34±2.496 min. this was found to be statistically significant (p = 0.00). This compares with the study done by of Saritha et al. [1], Amany et al. [13], Aliye Esmaoglu, et al. [7].

Rachana Gandhi et al. [8] conducted a study in 2012 to assess the effects of dexmedetomidine 30 µg (2ml) + 38 ml 0.25% bupivacaine. This was compared with 0.25% bupivacaine (38 ml) + normal saline (2ml). It was noted that onset of motor block in the study group (11.2±2.1 min) was earlier compared to onset of sensory blockade (21.4±2.5 min). It was also noted that onset of both motor block (8.5±1.4 min) and sensory block (18.4±2.5 min) in the control group was earlier compared to study group.

In the present study, study group received 15 ml 0.5% bupivacaine + 15 ml 2% lignocaine+ adrenaline with 1ml (100 µg) dexmedetomidine. Control group received 15 ml 0.5% bupivacaine + 2% lignocaine + adrenaline with 1ml normal saline. The onset of sensory blockade(11.4±2.032) was earlier compared to motor blockade (14.4±2.032min) in the study group. This finding correlates with the findings of Sandhya A et al. [11]., Saritha et al. [1], Amany et al. [13], Aliye Esmaoglu, et al. [7].

When the local anesthetic infiltrates the epineural barrier , the first fibers which will be encountered are motor, and therefore motor fibers are blocked first and thereafter sensory fibers will be blocked. Hence if the local anesthetic drug reaches at a mantle bundle in a concentration which is considerably above the critical motor concentration, topographical assembly of the nerve fiber is of little or no significance, despite the fact that the drug solution reaches the motor fibers first, blockade will not occur because of the insufficient concentration of the drug solution and the large size of the fiber, but as soon as the solution will reach the smaller pain fibers, blockade takes place as they have a lesser minimal effective concentration, and leads to analgesia [17].

The duration of sensory blockade in the study group was 633.23±57.549 min and 369.89±65.910 min for control population. This was found to be statistically significant (p= 0.00). This is in accordance with the results of earlier studies by Sandhya A et al. [11], Saritha et al. [1], Amany et al. [13], Aliye Esmaoglu, et al. [7].

The duration of motor blockade in the study group was 545.80±50.267 min and 317.49±53.717 min for control population. This was found to be statistically significant (p= 0.00). This is in accordance with the results of earlier studies by Sandhya A et al. [11], Saritha et al. [1], Amany et al. [13], Aliye Esmaoglu, et al. [7].

The mean duration of analgesia was 683.00 ± 60.450 in the study group as against 395.03 ± 49.090 min in the control group (p = 0.00). Similar results were found in the studies of Sandhya A et al. [11], Saritha et al. [1], Amany et al. [13], Aliye Esmaoglu, et al. [7]. There was a significant prolongation of duration of analgesia by 1.5–2 times after the addition of dexmedetomidine in brachial plexus block.

Stable hemodynamic was noted in the patients during the study except significant lower heart rate in study group at 15min, 30min, 45min, 60min and 90min as compared to the control group. There was no statistical difference in mean BP at the start of the procedure. Mean BP recorded was significantly lower in study group at 15 min, 45 min, 60 min durations in comparison to control group however it was similar in both the groups at the end of the procedure.

From this study we would like to conclude that dexmedetomidine can be safely used as an adjunct with local anesthetic in peripheral nerve blocks; however, further clinical trials are required in larger study population to determine the exact dosage and the safety profile of the drug.

Conclusion

In conclusion, dexmedetomidine added to 0.5% bupivacaine and 2% lignocaine + adrenaline is an attractive and safe option for improving the

duration and quality of supraclavicular brachial plexus block in upper limb surgeries.

Reference

1. Swami SS, Keniya VM, Ladi SD, Rao. Comparision of dexmedetomidine and clonidine as an adjuvant to local anaesthesia in supraclavicular brachial plexus block:A randomized double blind prospective study.Indian Journal of Anaesth, 2012; 56:243-9.
2. Halsted WS. Practical comments on the use and abuse of cocaine; suggested by its invariably successful employement in more than a thousand minor surgical operations. NY Med J 1885;42:294.
3. Sigh S, Aggarwal A. Arandomized controlled double-blinded study of the efficacy of clonidine added to bupivacaine as compared with bupivacaine alone used in supraclavicular brachial plexus block for upper limb surgeries. Indian J Anaesth 2010;54:552-7.
4. Biradar PA, Kaimar P, Gopalakrishna K. Effect of dexamethasone added to lidocaine in supraclavicular brachial plexus block: A prospective, randomised, double-blind study. Indian J Anaesth 2013;57:180-4.
5. Kim M.H , Lee Y.M. Intrathecal midazolam increases the analgesic effects of spinal blockade with bupivacaine in patients undergoing haemorrhoidectomy. Br J Anaesth 2001;86:77-9.
6. Candido K.D, Franco C.D . Khan M.A, Winnie A.P, Raja S.D. Buprenorphine Added to the Local Anesthetic for Brachial Plexus Block to Provide Postoperative Analgesia in Outpatients. Reg Anesth and Pain Med, 2001 Jul;26(4):352–6.
7. Aliye Esmaoglu, Fusun Yegenoglu, Aynur Akin, Cemil Yildirim Turk.Dexmedetomoidine Added to Levobupivacaine Prolongs Axillary Brachial Plexus block. Anaesth Analg. 2010:111(6):1548-51.
8. Gandhi R, Shah A,Patel I.Use of dexmedetomidine along with bupivacaine for brachial plexus block. National Journal of Medical Research. 2012 Jan- March;2(1):67-69.
9. F.W. Abdallah, R. Brull. Facilitatory effects of perineural dexmedetomidine on neuraxial and peripheral nerve block: a systematic review and meta-analysis. Br J Anaesth. 2013 Jun;110(6):915-25. doi: 10.1093/bja/aet066.
10. Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anesthetic requirement. Indian J Anaesth, 2011;55:352-7.
11. Khan ZP, Ferguson CN, Jones RM. Alpha-2 and imidazoline receptor agonists. Their pharmacology and therapeutic role. Anaesthesia 1999;54:146-165.
12. Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: A review of clinical applications. Curr Opin Anaesthesiol 2008;21:457-61.
13. Shukry M, Miller JA. Update on dexmedetomidine: Use in nonintubated patients requiring sedation for surgical procedures. Ther Clin Risk Manag 2010;6: 111-21.
14. Taghinia AH, Shapiro FE, Slavin SA. Dexmedetomidine in aesthetic facial surgery: Improving anaesthetic safety and efficacy. Plast Recon str Surg 2008;121:269-76.
15. Brummett CM, Norat MA, Palmisano JM, Lydic R. Perineural administration of dexmedetomidine in combination with bupivacaine enhances sensory and motor blockade in sciatic nerve block without inducing neurotoxicity in rat. Anesthesiology 2008; 109:502–11.
16. Brummett CM, Padda AK, Amodeo FS, Welch KB, Lydic R. Perineural dexmedetomidine added to ropivacaine causes a dose-dependent increase in the duration of thermal antinociception in sciatic nerve block in rat. Anesthesiology 2009;111:1111–9.
17. Winnie a P, Tay CH, Patel KP, Ramamurthy S, Durrani Z. Pharmacokinetics of local anesthetics during plexus blocks. Anesth Analg. 1977;56(6):852-61.