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RESEARCH ARTICLE

A COMPARITIVE STUDY OF CLONIDINE AND DEXMEDETOMIDINE AS ADJUVANT TO 0.25% BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK FOR POST OPERATIVE ANALGESIA AND SEDATION

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ABSTRACT

Background: Alpha -2 agonists are used in subarachnoid, epidural, regional blocks for their onset, duration and postoperative analgesia.[1-3]We compared the effects of adding clonidine and dexmedetomidine, as both are alpha -2 agonist, to a 35 ml solution of 0.25% bupivacaine in supraclavicular brachial plexus block.

Materials and Methods: Eighty patients of ASA 1 and 2 posted for upper limb orthopaedic surgeries were enrolled for a prospective, randomized, double-blind trial. Patients were divided into two groups, the Clonidine group C and the Dexmedetomidine group D. In group C (n = 40), 0.25% Bupivacaine + 1 µg/kg Clonidine; and in group D (n = 40), 0.25% Bupivacaine + 1µg/kg Dexmedetomidine, with the total volume of drug solution 35 cc. Drug solution were given for supraclavicular brachial plexus block using the peripheral nerve stimulator. The effects of adjuvants on duration of analgesia and sedation were compared.

Results: Demographic data and surgical characteristics were comparable in both the groups. Postoperative analgesia (POA) was longer in group D 637.50 ± 30.19 minutes than group C 294.38 ± 29.74, while Ramsay sedation score was higher in group D than group C.

Conclusion: Dexmedetomidine significantly prolonged the duration of postoperative analgesia along with high sedation scores.

INTRODUCTION

Upper limb orthopaedic surgeries are preferably performed in regional blocks such as brachial plexus block. The aim of postoperative pain relief is to provide subjective comfort in addition to inhibiting trauma induced nociceptive impulses to blunt autonomic and somatic reflex response to pain and subsequently enhance early restoration of functions.In the past two decade reappraisal of regional techniques has resulted ;

1) Interest in local block with controlled sedation so that the patient stays arousable during surgery.
2) Realization has grown that excellent post-operative pain relief can be provided easily and in no time to the patient with an appropriate regional blockade (Anibal *et al*., 1980)

Clonidine and Dexmedetomidine both are alpha 2 adrenoreceptor agonist, but dexmedetomidine is eight times more selective for alpha 2 adrenoreceptors than clonidine (Raimo *et al*., 1988). In the view of above goals, present study was done to compare the potentiation of postoperative analgesia and sedation of Clonidine and Dexmedetomidine with 0.25% bupivacaine as adjuvants.

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MATERIALS AND METHODS

After the approval of the Hospital Ethical Committee, patients were explained about the drug and only those who gave wilful written consent were included in the study. Eighty ASA physical status I and II patients, 18-60 years undergoing upper limb orthopaedic surgeries under supraclavicular brachial plexus block were enrolled in a prospective, randomized, double-blind trial. Exclusion criteria included infection at puncture site, bleeding disorder or patient on anticoagulant therapy, operation on shoulder joint, patients with abnormal psychological profile, history of opioid addiction, peripheral neuropathy & neurological deficit, history of convulsions, hepatic dysfunction, renal diseases, phrenic nerve palsy, pneumothorax and ischemic heart disease, failed blocks and patients who were supplemented intraoperatively with opioids are analgesics. Patients were randomly allocated in this double blind study (using a sealed envelope technique) into two groups. Clonidine group C (n = 40) received 35 ml solution of 0.25% bupivacaine with 1µg/kg clonidine. Dexmedetomidine group D (n = 40) received 35 ml solution of 0.25% bupivacaine and 1µg/kg of dexmedetomidine. The drug solutions were prepared by an anaesthesiologist not involved in the study. The anaesthesiologist performing the block and observing the patient was blinded to the treatment group. Data collection was done by the same anaesthesiologist who was unaware of the

group allocation. An 18 gauge (G) i.v. cannula was inserted in non-operated arm and lactated Ringer's solution was started.

The patients were administered brachial plexus block by supraclavicular route via the subclavian perivascular approach in supine position with arm adducted. Under strict aseptic precautions, the injection site was identified to be 1 cm behind the midpoint of the clavicle, (where the pulsation of the subclavian artery was felt) and infiltrated with 1 ml of 2% lignocaine subcutaneously. Neural localization was achieved by using Fisher and Paykel nerve stimulator, attached to a 22G 50 mm long stimulating needle. The location endpoint was a distal motor response, that is, the movement of the fingers and the thumb with an output current of 0.5 mA. During injection of the drug solution, negative aspiration was done after every 5 ml to avoid intravascular injection. Plexus block was considered successful when at least two out of the four nerve territories (ulnar, radial, median, and musculocutaneous) were effectively blocked for both sensory and motor block.

Sensory block (four nerve territories) was assessed by pin prick test using a 3-point scale:
0 = normal sensation
1 = loss of sensation of pin prick (analgesia)
2 = loss of sensation of touch (anaesthesia).

Motor block was determined by thumb abduction (radial nerve), thumb adduction (ulnar nerve), thumb opposition (median nerve), and flexion of elbow (musculocutaneous nerve) according to the modified Bromage scale (Sarkar *et al*., 2010) on a 3-point scale:

Grade 0: Normal motor function with full flexion and extension of elbow, wrist, and fingers
Grade 1: Decreased motor strength with ability to move the fingers only
Grade 2: Complete motor block with inability to move the fingers

Both sensory, motor blocks and sedation were assessed at 0,15, 30, 45, 60, 90,120, 150 and 180 min; and then hourly after the completion of injection, until they had resolved. Patients were asked to note the subjective recovery of sensation and movements which was then certified by an anaesthesiologist. Sedation score was assessed according to the modified Ramsay Sedation Scale (RSS) (Ramsay *et al*., 1974) from 1-6 as follows:

1 = Anxious, agitated, restless;
2 = Cooperative, oriented, tranquil;
3 = Responds to commands only;
4 = Brisk response to light glabellar tap or loud noise;
5 = Sluggish response to light glabellar tap or loud noise;
6 = No response.

Pain was assessed using visual analogue scale (VAS) 0-10. Protocol for rescue analgesia was identified to a VAS ≥ 5 with i/v tramadol 2mg/kg. The time between the complete sensory block and the first analgesic request was recorded as duration of post-operative analgesia (POA). The data was compiled and subjected to statistical analysis using Statistical Package for Social Sciences (SPSS), version 15. POA was subjected to Independent t-test for statistical analysis, while for sedation Chi –square test was applied. P-value < 0.05 was considered as statistically significant and P < 0.001 as highly significant.

RESULTS

The demographic data and surgical characteristics were comparable in both groups, (Table 1) The mean duration of post-operative analgesia (POA) for group D was 637.5.4 ± 30.19 min, while it was 294.38 ± 29.74 min for group C (Figure 1). POA was significantly longer in group D than group C (P < 0.001), (Table 2). The maximum modified Ramsay Sedation Score for group D was 4/6 in 28 patients, while that for group C score was 3/6 in 20 patients which was at highest at 75 min. Statistically high significant difference (P < 0.001) was seen at 30 min, which continued till 180 min. (Figure 2)

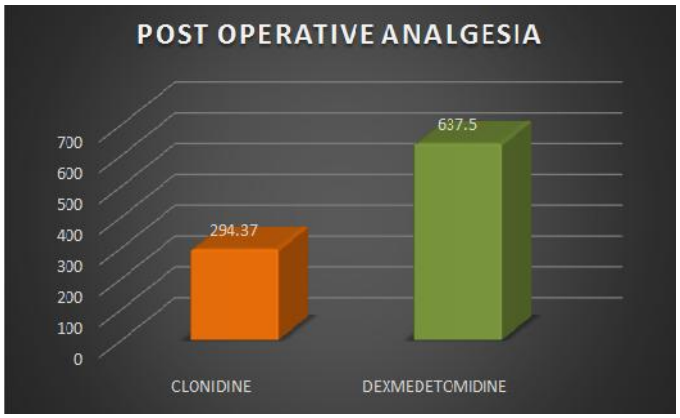

Figure 1

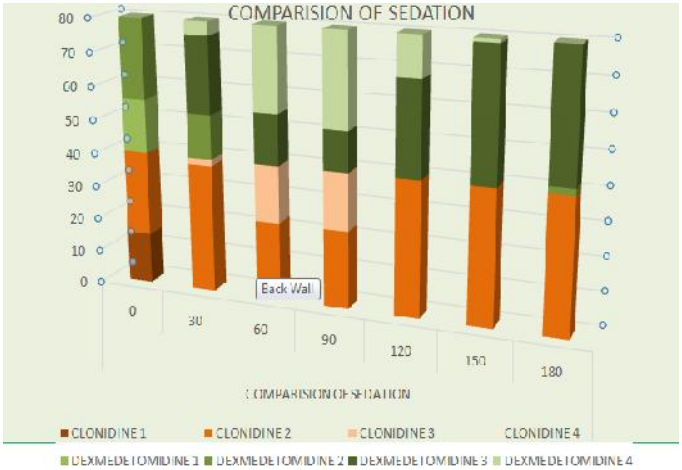

Figure 2

Table 1. Demographic and Surgical characteristics

	Group C (n = 40) Mean ± SD	Group D (n = 40) Mean ± SD
Age (Years)	32.1±8.9	31.4±9.3
Height (Cm)	164.3±10.2	165.5±11.1
Weight (Kg)	54.9±9.2	56.0±8.4
Gender (M/F)	27/13	24/16

C = Bupivacaine + Clonidine, D = Bupivacaine + Dexmedetomidine, M= Male, F =Female. There was no significant difference between groups

Table 2. Duration of analgesia in Groups C (Bupivacaine + Clonidine) and D (Bupivacaine + Dexmedetomidine)

	Group C (n = 40) (X ± SD)	Group D (n = 40) (X ± SD)	P-value Sig. (2 tailed)
Duration of analgesia (min)	294.38 + 29.74	637.50 + 30.19	0.000

Table 3. Comparision of sedation

	0	30	60	90	120	150	180
Clonidine 1	15	-	-	-	-	-	-
Clonidine 2	25	38	23	23	40	40	40
Clonidine 3	-	2	17	17	-	-	-
Clonidine 4	-	-	-	-	-	-	-
Dexmedetomidine 1	16	-	-	-	-	-	-
Dexmedetomidine 2	24	13	-	-	-	-	2
Dexmedetomidine 3	-	23	15	12	28	39	38
Dexmedetomidine 4	-	4	25	28	12	1	-
Asymp. Sig. (2-sided)	0.074	0.000	0.000	0.000	0.000	0.000	0.000

Sedation scores were compared at the start of surgery and intraoperatively at 30, 60, 90, 120, 150 180 min. Chi-square test used for statistical analysis showed significant difference among scores in both groups.

DISCUSSION

Dexmedetomidine, the pharmacologically active d-isomer of Medetomidine is a highly specific and selective α2 adrenoceptor agonist with α2:α1 binding selectivity ratio of 1620:1 as compared to 220:1 for clonidine, thus decreasing the unwanted side effects of α1 receptors, (Carollo *et al*., 2008; Hall *et al*., 2000). Presynaptic activation of α2 adrenoceptor in central nervous system (CNS) inhibits the release of norepinephrine, terminating the propagation of pain signals and their postsynaptic activation inhibits sympathetic activity. High selectivity for α-2A receptors mediates analgesia, sedation, and anxiolysis. Various randomized control trial done so far shows encouraging results for its use in intravenous sedation, as adjuvants in spinal, (Kanazi *et al*., 2006; Konakci *et al*., 2008), epidural (Yazbek-Karam *et al.,* 2006) caudal anaesthesia (El-Hennawy *et al*., 2009). Studies by Brummett *et al*. (2008, 2010) showed that dexmedetomidine enhances duration of bupivacaine anaesthesia and analgesia of sciatic nerve block in rats without any evidence of histopathological damage to the nerve (Brummett *et al*., 2008; Brummett *et al.,* 2010).

In another study, dexmedetomidine added to ropivacaine increased the duration of sciatic nerve blockade in rats, most likely due to the blockade of hyperpolarization-activated cation current (i.e., a direct effect on the peripheral nerve activity) (Brummett *et al*., 2011) Kosugi *et al*. (2010) examined the effects of various adrenoceptor agonists including dexmedetomidine, tetracaine, oxymetazoline and clonidine, and also an α2 adrenoceptor antagonist (atipamezole) on compound action potential (CAP) recorded from frog sciatic nerve, and found that CAPs were inhibited by α2 adrenoceptor, (Kosugi *et al*., 2010). Masuki *et al*., 2005 suggested that dexmedetomidine induces vasoconstriction via α2 adrenoceptors in the human forearm possibly also causing vasoconstriction around the site of injection, delaying the absorption of local anaesthetic and hence prolonging its effect Masuki *et al*. (2005). Esmaoglu *et al*. (2010) reported prolongation of axillary brachial Brachial plexus block when dexmedetomidine was added to levobupivacaine.Yoshitomi et al., demonstrated that dexmedetomidine as well as clonidine enhanced the local anaesthetic action of lignocaine via peripheral α-2A adrenoceptors (Yoshitomi *et al.,* 2008). To conclude, in our study we found that both clonidine and dexmedetomidine when added to Bupivacaine for supraclavicular brachial plexus block significantly prolonged duration of analgesia which ruled out the need for any additional analgesics. The added advantage of conscious sedation, makes them potential adjuvant for nerve blocks. Dexmedetomidine had longer post-operative analgesia and higher sedation as compared to clonidine without any significant adverse side effects.

Limitation of the Study

Since 'pain' is a subjective phenomenon associated with a wide variability of responses among the individuals, it is difficult to standardize the variable. What may be tolerable for one person may be intolerable for another. Pre-operative anxiety and apprehension may have led to some discrepancy in sedation scores.

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REFERENCES

Anibal G, Witcher T. 1980. Mixture of Local Anaesthetics Bupivacaine and Chloroprocaine. Anaesth. Analg. 59: 683.

Brummett CM, Amodeo FS, Janda AM, Padda AK, Lydic R. 2010. Perineuraldexmedetomidine provides an increased duration of analgesia to athermal stimulus when compared with a systemic control in a ratsciatic nerve block. *RegAnesth Pain Med* 35:427-31.

Brummett CM, Hong EK, Janda AM, Amodeo FS, Lydic R. 2011. Perineural dexmedetomidine added to ropivacaine for sciaticnerve block in rats prolongs the duration of analgesia by blockingthe hyper polarization-activated cation current. Anesthesiology, 115:836-43.

Brummett CM, Norat MA, Palmisano JM, Lydic R. 2008. Perineural administration of dexmedetomidine in combination with bupivacaine enhances sensory and motor blockade in sciatic nerve block without inducing neurotoxicity in rat. Anesthesiology, 109:502-11.

Carollo DS, Nossaman BD, Ramadhyani U. 2008. Dexmedetomidine: A review of clinical applications. CurrOpinAnaesthesiol, 21:457-61.

El-Hennawy AM, Abd-Elwahab AM, Abd-Elmaksoud AM, El-Ozairy HS, Boulis SR. 2009. Addition of clonidine ordexmedetomidine to bupivacaine prolongs caudal analgesia in children. *Br J Anaesth*, 103:268-74.

Elliott S, Eckersall S, Fliqelstone L. 1997. Does addition of clonidine affect duration of analgesia of Bupivacaine in inguinal hernia repair. *Br J Anaesth*, 79:446-9.

Esmaoglu A, Yegenoglu F, Akin A, Turk CY. 2010. Dexmedetomidine added to levobupivacaine prolongs axillary brachial plexus block. AnaesthAnalg 111:1548-51.

Hall JE, Uhrich TD, Barney JA, Arain SA, Ebert TJ. 2000. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. AnesthAnalg 90:699-705.

Kanazi GE, Aouad MT, Jabbour-Khoury SI, Al Jazzar MD, Alameddine MM, Al-Yaman R, *et al.* 2006. Effect of low-dose dexmedetomidine or clonidine on the characteristics of bupivacaine spinal block. Acta Anesthesiol Scand, 50: 2227.

Konakci S, Adanir T, Yilmaz G, Rezanko T. 2008. The efficacy and neurotoxicity of dexmedetomidine administered via the epidural route. *Eur J Anaesthesiol* 25:403-9.

Kosugi T, Mizuta K, Fujita T, Nakashima M, Kumamoto E. 2010. High concentrations of dexmedetomidine inhibit compound action potentials in frog sciatic nerve without alpha (2) adrenoceptor activation. *Br J Pharmacol* 160:1662-76.

Masuki S, Dinenno FA, Joyner MJ, Eisenarch JH. 2005. Selective alpha2- adrenergic properties of dexmedetomidine over clonidine in the human forearm. *J ApplPhysiol*, 99:587-92.

Popping DM, Elia N, Marret E, Wenk M, Tramèr MR. 2009. Clonidine as an adjuvant to local anaesthetic for peripheral nerve and plexus blocks: A meta-analysis of randomized trials. Anesthesiology 111:406-15.

Raimo V, Juha M, Veijo S, Leena N, Virtanen R. 1988. Characterisation of selectivity, specificity and potency of medetomidine as α2 adrenoceptor agonist. *Eur J Pharmacol*, 150:9-14.

Ramsay MA, Savage TM, Simpson BR, Goodwin R. 1974. Controlled sedation with alphaxolone-alphadolone. *Br Med J*, 2:656-9.

Sarkar DJ, Khurana G, Chaudhary A, Sharma JP. 2010. A comparative study on the effects of adding fentanyl and buprenorphine to localanaesthetica in brachial plexus block. *J ClinDiagn Res*, 4:3337-43.

Singelyn FJ, Gouveineur J, Robert A. 1996. A minimum dose of clonidine added to mepivacaine prolongs duration analgesia after brachial plexus block. AnesthAnalg 83:1046-50.

Yazbek-Karam VG, Aouad MA. 2006. Perioperative uses of dexmedetomidine. *Middle East J Anesthesiol*, 18:1043-58.

Yoshitomi T, Kohjitani A, Maeda S, Higuchi H, Shimada M, Miyawaki T. 2008. Dexmedetomidine enhances the local anesthetic action of lidocaine via an alpha-2A adrenoceptor. AnesthAnalg 107:96-101.

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