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COMPARATIVE EVALUATION OF DEXMEDETOMIDINE V/S NITROGLYCERINE WITH LIGNOCAINE IN BIER'S BLOCK FOR UPPER LIMB ORTHOPAEDIC SURGERIES

Anaesthesiology

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ABSTRACT

The analgesic effect of Dexmedetomidine and Nitroglycerine was evaluated when added to lidocaine in intravenous regional anaesthesia. We randomly assigned sixty patients undergoing upper limb orthopaedic surgeries into two groups, one of them receiving 1µg/kg dexmedetomidine and the other one receiving 200 µg nitroglycerine alongwith 0.5% lidocaine to total volume of 40 ml. Haemodynamic variables, VAS scores and analgesic requirements were recorded during the surgeries. After the tourniquet deflation, VAS score, time to first analgesic requirement, total analgesic consumption in the first 24 hours after surgery were recorded. Shortened sensory and motor block onset times were seen in nitroglycerine group (3.38±0.70 and 5.32±1.02 minutes respectively) as compared to dexmedetomidine group (4.50±0.91 and 8.18±1.53 minutes respectively) ($p<0.05$). Shortened VAS scores of tourniquet pain ($p<0.05$) and improved quality of anaesthesia were found in group LD (dexmedetomidine) ($p<0.05$). VAS scores were lower in group dexmedetomidine after tourniquet release and in the postoperative period ($p<0.05$). Postoperative analgesic requirements were significantly smaller in group LD ($p<0.05$). Mean duration of analgesia was longer in LD group (7.07±2.14 hours) as compared to LN (nitroglycerine) group (2.93±0.86 hours) ($p<0.05$). The sedation score was equal in both the groups in intraoperative period, however in the postoperative period, the median sedation score at 15 min and 30 min was higher in dexmedetomidine group as compared to nitroglycerine group ($p<0.05$). Surgeon satisfaction score in both the groups was equal, however the patient satisfaction score was significantly better in dexmedetomidine group ($p<0.05$). Hence, we conclude that dexmedetomidine group was accompanied by longer duration of analgesia and provides better quality of anaesthesia. Nitroglycerine group showed more rapid onset of sensory as well as motor blockade. In addition, dexmedetomidine group was associated with short-lived sedation in immediate postoperative period.

KEYWORDS

INTRODUCTION

In 1908, German Surgeon August K.G. Bier presented intravenous regional anaesthesia (IVRA) as a method to produce analgesia in an extremity especially for hand and forearm surgery[1]. He described complete anaesthesia after intravenous injection Prilocaine into a previously exsanguinated limb.

The main advantages of IVRA are its simplicity, reliability, and cost effectiveness.[2] Constraints of anaesthetic duration and tourniquet time limit the use of this technique to short procedures (approximately 20-60 minutes).[3] The rapid recovery of function make this technique ideally suited for surgeries performed in an ambulatory setting. There are few limitations associated with IVRA and those concerns regarding its use must be considered.[4] These concerns include, but are not limited to, local anaesthetic toxicity, delayed onset of action, poor muscle relaxation, tourniquet pain, and minimal postoperative analgesia.[4]

In an attempt to improve intra operative and postoperative qualities of IVRA, several adjuvants to the local anaesthetic can be added like opioids,[5] ketamine,[6] muscle relaxants,[7] nonsteroidal antiinflammatory drugs,[8,9] clonidine[10] and dexmedetomidine.[11]

α2 adrenergic receptor agonists have been the focus of interest for their sedative, analgesic and perioperative sympatholytic and cardiovascular stabilizing effect with reduced anaesthetic requirements.

Dexmedetomidine, is a potent α2 adrenoceptor agonist with strong analgesic effects. Nitroglycerine is a nitric oxide generator. By its vasodilator property, it can facilitate the distribution of local anaesthetics to the nerve trunk and enhance its analgesic effect. Nitroglycerine was used to improve the quality of IVRA in previous studies.[12,13]

A comparative study was carried out in our institute using IVRA with dexmedetomidine and nitroglycerine as adjuvants with lignocaine. The aim of the study was to compare the effects of adding dexmedetomidine or nitroglycerine to the local anaesthetic for IVRA on the intraoperative and postoperative analgesia during forearm and hand surgeries.

MATERIAL AND METHODS

Present study was a prospective, randomized double blind study in which 60 patients in the age group of 18-65 years of either sex, with American Society of Anesthesiologists (ASA) class I, II, III admitted to Guru Nanak Dev Hospital attached to Govt. Medical College, Amritsar and scheduled for elective upper limb orthopaedic surgeries were included after approval of institution's ethical and scientific committee and taking informed consent. Unwilling patients, morbidly obese patients, patients on anticoagulation therapy, known case of sickle cell anaemia, Raunaud's disease, coagulation disorders and scleroderma were disqualified from inclusion in this study.

ALLOCATION OF GROUPS

- Patients were randomly divided into two groups of 30 each.
- Group I (LD): 0.5% lidocaine plus 1 µg/kg dexmedetomidine to total volume of 40 ml.
- Group II (LN): 0.5% lidocaine plus 200 µg nitroglycerine to total volume of 40 ml.

TECHNIQUE

As premedication, tablet Alprazolam 0.25mg was given one night prior to surgery. After the patients had been taken to the operation theatre, mean arterial blood pressure (MAP), peripheral oxygen saturation (Spo2) and Heart Rate (HR) were monitored. Before establishing the anaesthetic block, two cannulae were placed; one was in a vein on the dorsum of the arm to be operated and the other in the opposite hand for infusion of fluids .Patients were administered Injection Midazolam 1mg, Injection Butorphanol 1mg and Injection Glycopyrrolate 0.2mg intravenously through the cannula placed on opposite non operative arm prior to commencement of surgery. The arm to be operated was exsanguinated with an Esmarch bandage after elevating it for 3 minutes. A double pneumatic tourniquet was placed around upper arm, and the cuff was inflated to 100mmHg more than systolic BP to a minimum of 250 mmHg and the Esmarch bandage was removed after that. Absence of radial pulse and loss of pulse oximetry tracing was checked for confirmation of circulatory isolation of the arm. Patients were given 0.5% lignocaine diluted with saline to a total volume of 40 ml to which either 1 µg /kg dexmedetomidine or 200 µg nitroglycerine was added to establish IVRA. Following injection of the drug, I/V cannula was removed, pressure was held on the site for a few minutes and within 5-10 minutes, reliable surgical anaesthesia was

achieved with adequate muscle relaxation. The sensory block was assessed every 30 seconds interval by pricking the dermatomal sensory distribution of median, ulnar and radial nerves by using a 25G short bevelled needle. At the same time, motor function was assessed every 30 seconds interval by instructing the patient to flex and extend his/her wrist and fingers .Time of complete motor block was noted when the patient was unable to perform any of these movements. After the achievement of sensory and motor block, the distal tourniquet was inflated to 250 mmHg followed by release of proximal tourniquet and commencement of the surgery. BP,HR,SpO2 ,Visual Analog Scale(VAS) scores(0=no pain and 10=worst pain) and sedation score was monitored every 10 minutes during surgery, every 15 minutes for 30 minutes after tourniquet deflation and then at hourly intervals for 4 hours and then 4 hourly till 24 hours.

Sedation Score was assessed as:

1. Completely awake.
2. Awake but drowsy.
3. Asleep but responsive to verbal commands.
4. Awake but responsive to tactile stimuli.
5. Asleep and not responding to any stimulus.

Bradycardia (defined as heart rate<60/min) was treated with intravenous Atropine 0.6mg. Hypotension (defined as drop in systolic blood pressure more than 20% of the baseline value) was treated with intravenous Ephedrine. Injection Butorphanol 0.5 mg was administered intravenously if patient complained of pain during surgery (VAS>3). Injection Diclofenac sodium 75 mg was administered intramuscularly if patient complained of pain post operatively (VAS>3). Intraoperative Butorphanol and postoperative Diclofenac sodium consumption was recorded and number of doses of butorphanol and diclofenac were calculated. Onset and Regression time of motor and sensory blocks were noted. Any side effects or complications were noted. The data from the present study was systematically collected, compiled and statistically analyzed to draw relevant conclusions. The above-mentioned parameters and patient's characteristics were compared using appropriate statistical tests. The non-parametric data was analysed using the 'Chi-Square test' and the parametric data was analysed using the 'Unpaired "t" test' and the inter group comparison of parametric data was done using the 'Post Hoc' test. The 'p value' was determined to finally evaluate the levels of significance. The p-value of <0.05 was considered significant and the p-value of <0.001 was considered highly significant. The results were analyzed and compared to previous studies using latest software IBM SPSS 23 to draw relevant conclusions.

RESULTS

Both the groups were comparable with respect to age, sex, weight, baseline haemodynamic variables, duration of surgery and intraoperative and postoperative haemodynamic variables (Table 1). Sensory block onset and recovery was 4.50±0.91 minutes and 6.82±0.54 minutes respectively in group I (LD) and 3.38±0.70 minutes and 6.61±0.44 minutes respectively in group II (LN).Motor block onset and recovery was 8.18±1.53 minutes and 8.25±0.61 minutes respectively in group I (LD) and 5.32±1.02 and 7.98±0.48 minutes respectively in group II (LN).Sensory onset time was significantly lower in group II as compared to group I (p-value <0.05).Similarly, motor onset time was significantly lower in group II as compared to group I (p-value<0.05) (Table 2).VAS score was significantly higher at 15 min, 30 min, 1hr, 2hr, 3hr, 4hr, 8hr, 12hr, 16hr, 20hr and 24hr time intervals in group II (LN) as compared to group I (LD).The mean supplemental analgesic requirement, in intraoperative period, in group LN was 0.19+0.40 doses, which is significant (p-value<0.05) as compared to group LD in which no patient required supplemental analgesic intraoperatively. During postoperative period, mean rescue analgesia requirement in group I (LD) was 1.03±0.26 doses while in group II (LN), it was 2.30±0.66 doses, which was significantly higher in group II as compared to group I(LD)(p-value < 0.05). The mean sedation score during intra operative period in both the groups was equal which was 1 (completely awake). During postoperative period, significantly higher sedation was observed at 15min, 30 min and at 1hr interval in group I (LD) as compared to group II (LN).The maximum sedation score achieved in either group was 3 i.e. asleep but responsive to verbal commands. The mean duration of analgesia, based on the time of request of first dose of supplement analgesic, in group I (LD) and group II (LN) was 7.07±2.14 hr and 2.93±0.86 hr respectively, which was significantly longer in group I (LD) than in group II (LN), p-value being <0.05.The surgeon satisfaction score was equal in both the groups, which was 3(3-3) i.e. perfect. The patient satisfaction was significantly higher in group I (LD) as compared to group II (LN). Quality of analgesia, as determined by the number of rescue analgesic doses during intra operative and postoperative period, was significantly better in group I (LD) as compared to group II (LN) (Table 3).

Table 1Values are mean ± SD. There were no significant differences between groups.

		Group LD	Group LN	P- value
Age (yrs)		34.34±14.79	35.30±15.3	0.805
Sex (M:F)		24:6	23:7	0.754
Weight (kg)		61.97±12.53	61.80±12.59	0.45
Duration of surgery (mins)		52.30±10.74	52.33±10.98	0.49
Baseline hemodynamic variables	pulse	81.40±15.47	81.87±15.33	0.32
	Systolic BP	122.60±23.63	124.13±23.97	0.23
	Diastolic BP	80.27±15.28	80.67±15.37	0.37
	SpO2	99.87±18.24	99.97±18.25	0.08
Intraoperative Hemodynamic variables	pulse	84.45±1.41	84.46±1.65	0.92
	Systolic BP	124.80±1.39	125.37±1.38	0.98
	Diastolic BP	82.11±1.08	82.01±1.27	1.00
	SpO2	99.86±0.05	99.86±0.09	1.00
Post operative hemodynamic variables	pulse	85.36±1.30	85.35±1.31	0.99
	Systolic BP	124.65±1.36	123.87±1.46	0.82
	Diastolic BP	81.85±1.12	81.59±1.13	0.91
	SpO2	99.94±0.02	99.72±0.09	0.99

LD= dexmedetomidine; LN= nitroglycerine

TABLE 2

variable	Group LD	Group LN	p- value
Sensory block onset time (min)	4.50±0.91	3.38±0.70	0.00
Sensory block recovery time (min)	6.82±0.54	6.61±0.44	0.11
Motor block onset time (min)	8.18±1.53	5.32±1.02	0.00
Motor block recovery time (min)	8.25±0.61	7.98±0.48	0.06

TABLE 3

variable	Group LD	Group LN	p- value
Intra operative VAS score	0.92±0.73	2.20±0.91	0.01
Intra operative supplemental analgesia requirement (doses)	0	0.19± 0.14	0.02
Intra operative sedation score	1±0	1±0	
Post operative VAS score	2.09±1.00	2.70±0.90	0.04
Post operative rescue analgesic requirement(doses)	1.03±0.26	2.30±0.66	0.00
Post operative sedation score	1.26±0.58	1.00±0.00	0.03
Mean duration of analgesia(hours)	7.07±2.14	2.93±0.86	0.00
Surgeon satisfaction score	3.00±0.55	3.00±0.55	
Patient satisfaction score	4.73±0.97	4.50±0.96	0.03

DISCUSSION

The main result of our study was that addition of Dexmedetomidine to lidocaine for IVRA provided better quality of analgesia with reduced intraoperative and postoperative analgesic consumption and longer duration of postoperative analgesia without causing any side effects. These improvements were associated with more but short lived sedation. However, the addition of Nitroglycerine to lidocaine improved the speed of motor and sensory onset of anaesthesia.

It is proposed that the site of action of IVRA is both the nerve endings and the major nerve trunks, possibly reaching the nerve trunk via small venules within the nerve core.[14]

The precise mechanism by which α 2 blockers exert their analgesic effects remains unknown. Activation of postsynaptic α 2 receptors in substantia gelatinosa of the spinal cord is the presumed mechanism by which α 2 blockers produce analgesia[15]. However, the analgesic effects of Dexmedetomidine in the clinical setting have been investigated primarily in regard to its opioid sparing action. α 2 blockers enhance peripheral nerve blocks of local anaesthetics by selectively blocking A delta and C fibres. These may produce a peripheral analgesic effect by releasing encephalin like substances. Finally, alpha 2 adrenergic receptors located at nerve endings may have a role in the analgesic effect of the drug by preventing norepinephrine release[16].

Intravenous administration of Dexmedetomidine produces abrupt

hypertension and bradycardia until the central sympatholytic effect predominates, resulting in moderate decreases in both mean arterial pressure and heart rate from baseline, and it also has a sedative effect[17]. The ability of Dexmedetomidine to blunt the central sympathetic response explains its sedative, proanaesthetic and proanalgesic effects at 0.5-2 µg/kg dose given intravenously. It also minimizes opioid induced muscle rigidity, lessens postoperative shivering, causes minimal respiratory depression and has hemodynamic stabilizing effects.[18]

Alpha2 adrenergic agonists produce sedation, and electroencepha lographic studies confirm the increase in stage I and II sleep. The hypnotic response probably is mediated by activation of α 2 adrenoceptors in the locus ceruleus, which are coupled via a pertusis-sensitive G protein to a change in conductance through an ion channel[19].

NTG is metabolized to nitric oxide (NO) in the cell.[20,21] NO produces an increase in the intracellular concentration of cyclic guanosine monophosphate, which produces pain modulation in the central and peripheral nervous system.[20,22] NO generators also induce anti-inflammatory effects and analgesia by blocking hyperalgesia and the neurogenic component of inflammatory oedema by topical application.[23] Another possible mechanism includes an analgesic effect through the direct stimulation of peripheral fibres mimicking the actions of locally applied acetylcholine.[20] All these mechanisms might contribute to the analgesic effects of NTG added to lidocaine in IVRA.

Sensory and motor block onset times were statistically shorter in group II (LN) in our study. This is advantageous for the surgeon as well as for the anaesthesiologist. This beneficial effect of NTG might depend on a strong vasodilatory effect that promotes distribution of lidocaine to nerves. This result corresponds to the findings of Selda Sen et al, who reported shortened sensory and motor block onset times using the same dose of NTG as is used in the present study.[24]

CONCLUSION

The results of this study suggest that the addition of Dexmedetomidine to lidocaine in IVRA improves the quality of the block, decreases the intraoperative and postoperative analgesic consumption and provides longer duration of analgesia without any side effects. On the other hand, addition of Nitroglycerine to lidocaine in IVRA shortenes the sensory and motor onset times. In addition, dexmedetomidine group showed higher sedation in immediate postoperative period as compared to nitroglycerine group, but this sedation was transitory.

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