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ORIGINAL ARTICLE

Effect of dexmedetomidine as adjuvant to lidocaine in intravenous regional anaesthesia for below elbow surgeries

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ABSTRACT

Background: Intravenous regional anaesthesia (IVRA) is safe, technically simple but it has several disadvantages as limited duration, lack of postoperative analgesia, and tourniquet pain.

Aim of the study: This study was a prospective comparative randomized controlled clinical study that was carried on to evaluate the effect of addition of dexmedetomidine to lidocaine on the characters of the produced intravenous regional anaesthesia for below elbow surgeries.

Patients and methods: Forty both sexes' patients, aged 18–50 years of ASA ps class I and II, undergoing hand and forearm surgeries were selected for this study. Patients were randomly divided into three equal groups: Lidocaine group (L group) received 10 ml of 2% lidocaine, Lidocaine/Dexmedetomidine group (L/D group) received 10 ml of 2% lidocaine plus 0.5µg/kg dexmedetomidine. The volume of the lidocaine with or without adjuvant increased to 40ml by normal saline. After that, the characters of the produced regional anaesthesia were recorded.

Results: Dexmedetomidine enhanced the onsets of sensory but not motor blocks, decreased the mean of surgical pain scores, decreased the intraoperative fentanyl consumption, delayed the onset of tourniquet pain, prolonged postoperative analgesia with minimal side effects.

Conclusion: Dexmedetomidine, when used as adjuvant to lidocaine for IVRA, significantly improve the quality of the produced regional anaesthesia.

Keywords: Dexmedetomidine, intravenous regional anesthesia, lidocaine.

INTRODUCTION

Intravenous regional anesthesia (IVRA) was first described by August Bier in 1908 (1). It has several advantages as being very simple, reliable and economic, wide safety margins, very high success rate and rapid onset (2). On the other hand, it has several disadvantages as short duration, tourniquet pain, great liability to local anaesthetic toxicity and very short postoperative analgesia after deflation of the tourniquet (3). In attempt to improve intra-operative and postoperative qualities of the IVRA, many adjuvant were added as muscle relaxants (4), opioids (5), ketamine (6), non-steroidal anti-inflammatory drugs (7), neostigmine (8), midazolam (9) Calcium

channel blockers(10) and dexmedetomidine (11)

Dexmedetomidine is highly selective toward the α_2 adrenoceptors. Nowadays, dexmedetomidine is commonly added to local anaesthetics to improve the quality of peripheral nerve block.

The aim of the current study was to evaluate the effect of addition of dexmedetomidine as adjuvant to lidocaine on the characters of the produced intravenous regional anaesthesia for below elbow surgeries.

PATIENTS AND METHODS

This study was prospective comparative randomized controlled clinical study that had been carried out at Zagazig University Hospitals from May, 2018 to February, 2019

after obtaining approval of Institutional Review Board (IRB) and informed consent from the patients. The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

This study included forty adult patients undergoing below elbow surgeries. **The inclusion criteria** were patients of the American Society of Anesthesiologists (ASA) physical Status class I and II, aged between 18 and 65 years and their body weight ranged from 75-95Kg, scheduled for unilateral minor operations on the forearm or hand (i.e. not need more than 60 min.). **The exclusion criteria** were patient refusal, uncooperative patients, difficult vein, crush injury, sickle cell disease, allergic reaction to the tested drugs, peripheral vascular and neurological diseases, muscle, hepatic and renal diseases beside cardiac conduction abnormalities.

All patients were visited for clinical evaluation to find out any exclusive criteria, to explain the technique of IVRA and to record the base line Heart rate, Mean arterial blood pressure, respiratory rate, and peripheral arterial oxygen saturation. No premedication was prescribed. In operating room, for safety, resuscitation equipments and emergency drugs were near to the patient. IV cannula and sphygmomanometer cuff were applied to the non-operated limb for fluids and drug administration and continuous measurement of blood pressure respectively. Also, ECG leads and pulse oximeter probe were applied to the chest and the big toe of one of the patient's lower limbs for continuous monitoring of heart rate, rhythm, and peripheral arterial oxygen saturation.

Another iv cannula was inserted into the most peripheral vein in the limb to be blocked. After that, the pre-checked double pneumatic tourniquet was applied to a well-padded proximal third of the arm of the limb to be operated.

Exsanguination of the limb was achieved by application of Esmarch bandage on the above heart raised limb. Immediately and after applying of Esmarch bandage, the proximal cuff of the pre-applied pneumatic tourniquet was inflated to a pressure of 100 mmHg above

the initial systolic pressure. After securing pneumatic tourniquet, Esmarch bandage was removed and the upper limb was lowered and checked for colour (pale colour) and arterial occlusion (absence of radial pulse) to be sure of the efficacy of the applied pneumatic tourniquet.

After that, the local anaesthetic mixture was slowly injected. When sensory block reached to the level of middle third of the arm, the distal cuff of pneumatic tourniquet was inflated to a pressure of 100 mmHg above the initial systolic pressure. Then, the proximal one was deflated.

The study participants were randomized using a computer-generated random numbers table into two equal groups. These two groups were Lidocaine group (L or control group) which received 10 ml of 2% preservative-free lidocaine and Lidocaine/ dexmedetomidine group (L/D group) which received 10 ml of 2% preservative-free lidocaine (lidocaine HCl; Hospira, Lake Forest, Illinois, USA) plus 0.5µg/kg dexmedetomidine (Precedex, Abbott Laboratories Inc., Abbott Park, IL).

The volume of the lidocaine with or without adjuvant increased to 40ml by normal saline. After local anaesthetic injection, the characters of the produced regional anaesthesia were assessed and recorded. These characters are the following:

I- Onset of sensory and motor block: It was the time (minutes) from the moment of local anaesthetic mixture administration to the moment of loss of sensation to pin prick at the middle third of the arm for the first and to the moment at which the patient was unable to flex his fingers, wrist, and elbow joints for the later ⁽⁹⁾

II- Analgesic potency of intravenous regional block: It was evaluated by assessing intra-operative surgical pain intensity, the total amount of supplemental systemic fentanyl which was needed to relief surgical pain and duration of tolerance to tourniquet pain.⁽⁹⁾

Intra-operative surgical pain intensity was evaluated by Visual Analogue Scale (VAS) and it was estimated at skin incision, every 5 minutes during the operation, and at skin closure. The mean of all these values were detected in each group.

Duration of tolerance to tourniquet pain was the time from the moment of tourniquet inflation to the moment at which the patient was unable to tolerate more the pain exerted by the inflated tourniquet on the applied area.

III- Sensory and motor block recovery (Offset) times:

These were the times from the moment of deflation of the tourniquet till the moment of return of pin prick sensation of the limb for the first and till the moment at which the patient can flex his fingers, wrist, and elbow joints for the later. These were assessed every 2 minutes.

IV- The time to ask for post operative analgesia (It was the time in minutes from the moment of tourniquet deflation to patient reporting pain intensity above 3 according to VAS) and **the amount of systemic Diclofenac sodium** which was needed to alleviate postoperative pain from the moment of deflation of tourniquet till the end of the first 24 hours postoperatively. VAS was assessed every 15 minutes.

Diclofenac sodium (75 mg im every 8 hours) was given to the patient if he was unable to tolerate postoperative pain i.e. VAS is more than 3.

V- The incidences of the various associated side effects:

The associated side effects as Local anaesthetic toxicity, bradycardia (heart rate decreases by > 30% of basal reading) ⁽⁹⁾, hypotension (mean arterial blood pressure decreases by > 30% of basal reading)⁽⁹⁾, hypopnea (respiratory rate < 8 breaths/min), hypoxemia ($SpO_2 < 90\%$ on room air) ⁽⁹⁾, and sedation (i.e. sedation score more than 2 intra or postoperatively) were detected and recorded. The sedation level was assessed by means of six points Ramsay agitation/sedation scale that is presented in **table 1**⁽¹²⁾. Bradycardia was treated with IV atropine (0.5 mg). Hypotension was treated with IV ephedrine (5 to 10-mg bolus). Hypoxemia was treated with O₂ supplementation via a face mask.

Table (1): Ramsay agitation/sedation scale.⁽¹²⁾

At the end of the operation, the tourniquet was deflated by intermittent

deflation and re-inflation technique to avoid Ischemia-reperfusion shock. Tourniquet deflation was never done before passing 20 minutes after local anaesthetic mixture injection even if the operation had been finished before lapsing that time.

One hour after tourniquet deflation postoperatively, all patients were discharged to ward.

Statistical analysis:

It was done on the basis of the Gergers study ⁽¹⁰⁾, power of the test was 80% and confidence level was 95%, so the sample size was calculated to be 32 subjects, 16 patients for each group. For compensation for any dropped cases, the group size increased from 16 to 20 in each group. The sample size was calculated using Open Epi program.

The data were analyzed by using SPSS software program. The Values were presented as mean or median and standard deviation. Quantitative data were statistically analyzed by Student t-test. Ratios and % data were statistically analyzed by Chi-square test. In all tests, P value below 0.05 and 0.001 were considered statistically significant and highly significant respectively.

Results:

The demographic data (age, sex, height, and weight and ASA physical status classes), duration of surgery, tourniquet time and distribution of the various types of operations were presented in **table 2**. Statistically, these were comparable in the two studied groups.

The onset of each of sensory and motor blocks was 6.60 ± 1.5 and 10.5 ± 3.7 min respectively in L group and 3.8 ± 1.4 and 9.3 ± 3.2 min in L/D group. Statistically, the onset of sensory block in L/D was highly significant faster than that in L group ($P < 0.001$). Onset of motor block in L/D was comparable with that in L group (**Table 3**).

The mean of surgical pain scores was 2.4 ± 0.85 in L group and 0.7 ± 0.34 in L/D group. The mean of intraoperative fentanyl consumptions ($\mu\text{g}/\text{patient}$) was 15.5 ± 1.7 in L group and 2.80 ± 0.76 in L/D group.

Mean duration of tolerance to tourniquet pain (min) was 14.7 ± 3.8 in L group and 27.4 ± 4.06 in L/D group. Statistically, the mean of surgical pain scores and intraoperative

fentanyl consumptions ($\mu\text{g}/\text{patient}$) were highly significant less and the duration of tolerance to tourniquet pain was highly significant longer in L/D group than in L group (**Table 4**).

Sensory and motor block recovery times were 10.89 ± 1.77 and 13.35 ± 2.39 min. respectively in L group and 24.35 ± 3.39 and 32.80 ± 2.29 min. respectively in L/D group. Statistically, sensory and motor block recovery times in L/D group were highly significant longer than in L group (**Table 5**).

The time to ask for post operative analgesia was 52.50 ± 18.70 min. in L group and 127.8 ± 22.60 min. in L/D group. The consumed amount of diclofenac to relief pain in the 1st 24

hours postoperatively was 168.75 ± 58.97 mg/patient in L group and 82.5 ± 23.08 mg in L/D group. Statistically, the time to ask for post operative analgesia was highly significant longer and the consumed amount of diclofenac to relief pain in the 1st 24 hours post-operatively was significantly less in L/D group than those in L group (**Table 6**).

The associated side effects were bradycardia, hypotention and sedation that occurred after deflation of tourniquet at the end of operations. Each side effect occurred in 4 patients in L/D group and did not occur in L group (**Table 7**). Numerically but not statistically, the incidence of bradycardia, hypotention and sedation in L/D group were higher than in L group.

Table (1): Ramsay agitation/sedation scale ⁽¹²⁾.

Awake levels	Patient anxious or agitated or both	1
	Patient cooperative ,oriented and tranquil	2
	Patient responds to commands only	3
Asleep levels	A brisk response to a light glabellar tap	4
	A sluggish response to a light glabellar tap	5
	No response	6

Table (2): Patients demographic data, duration of surgery, tourniquet time and distribution of the various types of operations in the two studied groups.

	L Group (n=20) Mean \pm SD	L/D Group (n=20) Mean \pm SD	T-tests P-value
	Age (years).	29.56 \pm 4.23	32.91 \pm 4.15
Weight (kg).	86.43 \pm 5.12	84.62 \pm 6.28	0.531
Height (cm).	170.4 \pm 5.67	168.23 \pm 6.4	0.383
Sex ratio (Male/ Female ratio).	12/8	10/10	0.281
ASA ps classes (Class I/II ratio).	17/3	18/2	0.817
Duration of surgery (min.).	46.75 \pm 5.2	48.35 \pm 5.1	0.647
Tourniquet time (min).	53.10 \pm 7.4	57.70 \pm 5.6	0.112
Distribution of the various types of operations [N (%):			
- Carpal tunnel release.	8 (40 %)	8 (40%)	0.934
- Ganglion excision.	4 (20%)	4 (20%)	0.906
- Fracture fixation.	2 (10%)	2 (10%)	0.804
- Tendon repair.	0 (0.0%)	1 (5.0%)	0.596
- Foreign body removal.	4 (20%)	3 (15%)	0.676
- Plate and screw removal.	1 (5.0%)	0 (0.0%)	0.596
- Tendon lengthening.	0 (0.0%)	1 (5.0%)	0.596
- Nerve repair.	1 (5.0%)	1 (5.0%)	0.596

Data are expressed as Mean \pm Standard Deviation (SD) or numbers (%).

n = Group number. N = number of each operation type in each group.

L group = lidocaine alone (Control) group.

L/D group = Lidocaine/Dexmedetomidine group.

ASA ps classes = American Society of Anesthesiology physical status classes.

P > 0.05 = non significant difference

Table (3): Onset of sensory and motor block after establishment of IVRA in the two studied groups.

	L group (n=20) Mean \pm SD	L/D Group (n=20) Mean \pm SD	T-tests
			P-value
Onset of sensory block (min).	6.60 \pm 1.5	3.8 \pm 1.4	<0.001
Onset of motor block (min).	10.5 \pm 3.7	9.3 \pm 3.2	>0.050

Data are expressed as Mean \pm Standard Deviation (SD).

n = Group number.

L group = lidocaine alone (Control) group.

L/D group = Lidocaine/Dexmedetomidine group.

P < 0.001 = highly significant difference.

Table (4): Analgesic potency of intravenous regional block in the two studied groups.

	L Group (n=20) Mean \pm SD	L/D Group (n=20) Mean \pm SD	T test
			P-value
Intra-operative surgical pain score (VAS values).	2.4 \pm 0.85	0.7 \pm 0.34	<0.001
Total fentanyl consumption during surgery (μ g/patient).	15.5 \pm 6.7	2.80 \pm 5.6	<0.001
Duration of tolerance to tourniquet pain (min).	14.7 \pm 3.8	27.4 \pm 4.06	<0.001

Data are expressed as Mean \pm Standard Deviation (SD).

n = Group number.

L group = lidocaine alone (Control) group.

L/D group = Lidocaine/Dexmedetomidine group.

P < 0.001 = highly significant difference.

Table (5): Sensory and motor block recovery (Offset) times from IVRA in the two studied groups.

	L Group (n=20) Mean \pm SD	L/D Group (n=20) Mean \pm SD	T test
			P-value
Sensory block recovery time (min).	10.89 \pm 1.77	24.35 \pm 3.39	<0.001
Motor block recovery time (min).	13.35 \pm 2.39	32.80 \pm 2.29	<0.001

Data are expressed as Mean \pm Standard Deviation (SD).

n = Group number.

L group = lidocaine alone (Control) group.

L/D group = Lidocaine/Dexmedetomidine group.

P < 0.001 = Highly significant difference.

Table (6): The time to ask for postoperative analgesia and the consumed amount of diclofenac sodium to relief pain in the 1st 24 hours postoperatively in the two studied groups.

	L Group (n=20) Mean ± SD	L/D Group (n=20) Mean ± SD	T test P-value
	Time to ask for postoperative analgesia (min).	52.50±18.70	127.8±22.60
The consumed amount of diclofenac sodium during the 1 st 24 hrs postoperatively (mg/patient).	168.7±58.97	82.50±23.08	<0.001

Data are expressed as Mean ± Standard Deviation (SD).

n = Group number.

L group = lidocaine alone (Control) group.

L/D group = Lidocaine/Dexmedetomidine group.

P< 0.001 = Highly significant difference.

Table (7): The incidences of the various associated side effects in the two studied groups.

	Groups					
	L Group (n= 20)		L/D Group (n= 20)		Chi-square test	
	N	%	N	%	X ²	P-value
Haemodynamic changes:						
Bradycardia.	0	0	4	20	0.536	0.765
Hypotension.	0	0	4	20	0.536	0.765
Patient with sedation score more than 2 intra and postoperatively.	0	0	4	20	0.349	0.349

Data are expressed as numbers (%).

n = Group number.

N = number of each associated side effect in each group.

L group = lidocaine alone (Control) group.

L/D group = Lidocaine/Dexmedetomidine group.

P> 0.05 = non significant difference

DISCUSSION

Intravenous regional anesthesia (IVRA) is a simple, reliable, and cost-effective technique which has success rates of 94 - 98%.⁽⁸⁾ The disadvantages of IVRA are slow onset, limited duration for surgery, poor muscle relaxation, tourniquet pain, lack of postoperative analgesia, and local anaesthetic toxicity.⁽¹³⁾

The present study demonstrated that, addition of dexmedetomidine as adjuvant to lidocaine, enhanced the onset of sensory but not motor block of IVRA.

These findings were in agreement with some workers. Nasr and Waly⁽¹⁴⁾ and

Elramely and Elmoutaz⁽¹⁵⁾ reported that, addition of dexmedetomidine as adjuvant to lidocaine for IVRA enhanced the onset of its sensory block, but on the contrary the onset of motor block was not affected. Gerges⁽¹⁰⁾, Abdelkader et al⁽¹⁶⁾, Nilekani et al⁽¹⁷⁾ and Mahmoud et al⁽¹⁸⁾ reported that, addition of dexmedetomidine as adjuvant to lidocaine for IVRA enhanced the onset of its sensory block. In contrast, Subramanya et al⁽¹⁹⁾ reported that, addition of dexmedetomidine as adjuvant to lignocaine for IVRA leads to earlier onset of both sensory and motor blocks. The controversy between the present study finding

and Subramanya et al. finding was attributed to the premedication that they gave. Subramanya et al used 0.015 mg/kg midazolam intravenously for premedication to all their patients but no premedication was given to the patients of the present study. Kohno et al. ⁽²⁰⁾ reported that, systemic benzodiazepine induces attenuation of motor tonus at the ventral horn of the spinal cord.

In the present study, the detected rapid onset of sensory block of IVRA in dexmedetomidine added group means that, it has synergistic effect to sensory blockade of lidocaine in peripheral nerve blocks. The mechanism by which dexmedetomidine enhances the sensory blockade of local anaesthetics in peripheral nerve blocks is unclear. It is postulated that, dexmedetomidine has a local anaesthetic effect with rapid onset of sensory blockade.

In the present study, it was found that, addition of dexmedetomidine as adjuvant to lidocaine for IVRA, increased its analgesic potency during surgery. The signs which indicated that were a decrease in the mean of surgical pain scores, an increase in the duration of tolerance to tourniquet pain and a decrease in the intra-operative fentanyl consumption.

These findings were in agreement with the reported findings of some workers. Gerges ⁽¹⁰⁾ Nasr and Waly ⁽¹⁴⁾ , Abdelkader et al ⁽¹⁶⁾ , Nilekani et al ⁽¹⁷⁾ , Mahmoud et al ⁽¹⁸⁾ and Subramanya et al. ⁽¹⁹⁾ reported that, addition of dexmedetomidine as adjuvant to lidocaine for IVRA increased the duration of tolerance to tourniquet pain and decreased the intra-operative fentanyl consumption.

The mechanism by which α_2 -adrenergic receptor agonists produce analgesia and sedation is not fully understood but is likely to be multifactorial. Peripherally, α_2 agonists produce analgesia by reducing release of norepinephrine and causing α_2 receptor-independent inhibitory effects on nerve fiber action potentials ⁽²¹⁾. Centrally, α_2 agonists produce analgesia and sedation by inhibition of substance P release in the nociceptive pathway at the level of the dorsal root neuron and by activation of α_2 adrenoceptors in the locus coeruleus ⁽¹¹⁾.

The present study demonstrated that, addition of dexmedetomidine as adjuvant to lidocaine for IVRA led to prolongation of sensory and motor block recovery times after tourniquet deflation.

These findings were in accordance with some reported findings. Gerges ⁽¹⁰⁾ , Nasr and Waly ⁽¹⁴⁾ and Elramely and Elmoutaz ⁽¹⁵⁾ , Abdelkader et al ⁽¹⁶⁾ , Nilekani et al ⁽¹⁷⁾ , Mahmoud et al ⁽¹⁸⁾ and Subramanya et al ⁽¹⁹⁾ reported that addition of dexmedetomidine as adjuvant to lidocaine for IVRA led to prolongation of the duration of post-operative analgesia and lowering the VAS score after tourniquet release.

The detected prolonged sensory block recovery times after release of tourniquet in dexmedetomidine added group may be attributed to the more stay of the combined lidocaine/dexmedetomidine than lidocaine alone in the operating limb⁽²¹⁾.

In the present study, the associated side effects were bradycardia, hypotension and sedation. Each of these side effects occurred in 4 patients in dexmedetomidine added group and did not occur in lidocaine alone group.

These results were in agreement with some workers and in disagreement with other workers.

Regarding to the associated hemodynamic changes, Nasr and Waly ⁽¹⁴⁾ and Elramely and Elmoutaz ⁽¹⁵⁾ , reported some bradycardia after deflation of the tourniquet in dexmedetomidine added group. On the contrary, Abdelkader et al ⁽¹⁶⁾ and Nilekani et al ⁽¹⁷⁾ and Subramanya et al ⁽¹⁹⁾ and Gupta et al ⁽²²⁾ reported that, addition of each of dexmedetomidine as adjuvant to lidocaine for IVRA did not lead to any hemodynamic changes.

Regarding to the associated sedation, Nasr and Waly ⁽¹⁴⁾ , Elramely and Elmoutaz ⁽¹⁵⁾ , Subramanya et al. ⁽¹⁹⁾ and Sheth et al ⁽²³⁾ reported that addition of dexmedetomidine as adjuvant to lignocaine for IVRA was associated with short-lived sedation.

α_2 -adrenergic receptors at the nerve endings are thought to play a role in the analgesic effect of the drug by preventing norepinephrine release ⁽²⁴⁾. The actions of dexmedetomidine as found to be mediated via postsynaptic α_2 -

adrenoceptors activate G-proteins, thereby increasing conductance through potassium channels. Studies in mice have demonstrated that the α_{2A} -adrenoceptor subtype is responsible for relaying the sedative and analgesic properties of dexmedetomidine (25). Thus, α_2 -agonists are an attractive option as an adjuvant in pain management because of their potentiating effects at central and peripheral sites (26).

Tourniquet deflation can lead to an abrupt introduction of dexmedetomidine into the systemic circulation. Acute intravenous administration of dexmedetomidine is known to produce hypotension, bradycardia and also sedation (27 & 28).

The detected each of bradycardia and hypotension in 4 patients of L/D group might be related to the postsynaptic activation of central α_2 -adrenoceptors, leading to decreased sympathetic activity that decrease the blood pressure and slower HR (29).

The detected sedation in 4 patients of L/D group was attributed to the central sedative effect of dexmedetomidine (α_2 agonists) by inhibition of substance P release in the nociceptive pathway at the level of the dorsal root neuron and by activation of α_2 adrenoceptors in the locus coeruleus (10).

Limitations of this study were lack of patients and surgeons assessment of the quality of IVRA and lack of control groups received systemic dexme-detomidine as adjuvant to lidocaine IVRA to compare their central versus peripheral sites of action.

CONCLUSION

Dexmedetomidine when added as adjuvant to lidocaine significantly improve the quality of the produced intravenous regional anaesthesia with minimal associated side effects.

Recommendation: Addition of dexmedetomidine as adjuvant to lidocaine is recommended to improve the quality of intravenous regional anaesthesia.

Conflict of Interest: No any financial or personal relationships with other people or organizations that could inappropriately influence the current study.

Financial Disclosures: No any specific financial interests, relationship and affiliations relevant to the subject of the manuscript.

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