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RESEARCH ARTICLE

COMPARISON BETWEEN TWO APPROACHES OF TAB BLOCK FOR POST HERNIATOMYANALGESIA: TRIANGLE OF PETITE VERSUS ANTERO –TO ANTERIOR SUPERIOR ILIAC SPINE

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ABSTRACT

Background: Inguinal hernia repair is associated with considerable postoperative discomfort. The perioperative management of pain following abdominal surgery can pose a challenge to anesthesia providers. Conventional practice has involved the use of opioids as well as neuraxial analgesic techniques. Unfortunately, these therapies are not without potential risks and side effects. **Aim of the study:** To compare the postoperative pain relief provided by Ultrasound guided TAP block technique with ultrasound guided ilioinguinal/iliohypogastric nerve blockade in adult undergoing unilateral hernia repair surgery regarding duration. **Patient and method:** A prospective randomized controlled study of 60 male patient undergone elective unilateral inguinal hernia repair, 30 of them under ultrasound guide TAP block anterior approach at the end operation using 20 ml of 0.25% plain bupivacaine was done. 30 of them under ultrasound guide TAP block posterior approach at the end operation using 20 ml of 0.25% plain bupivacaine was done. Then Vital signs, numerical pain score and analgesia requirement were recorded at recovery,2nd, 4th & 8th hours postoperatively. **Result:** By applying null hypothesis, using the t-student test of independent two samples, Pain score and Request for analgesia show no significant difference in both methods for postoperative pain relief. For vital signs there was no significant difference for both groups. **Conclusion:** There is no significant difference between both methods. Those two methods provide good analgesia with less postoperative other medication use without any significant side effect.

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INTRODUCTION

Intravenous regional anesthesia (IVRA) was first used by August Bier in 1908. This technique is easy, reliable and cost effective with a high success rate of 94–98%when used in short operative procedures of hand or forearm (Brill *et al*., 2004; Singh *et al*., 2010). The advantage of this method has fast return of motor and sensory function which enables patients for earlier discharge; however, this method has disadvantages such as tourniquet pain, insufficient muscle relaxation and postoperative analgesia (Jones, 1996). In order to avoid these disadvantages, many adjuvant drugs have been added to local anesthetics, many adjuvant drugs in literature, such as, NSAIDs, paracetamol, opioids and adrenergic receptor agonists (Sen *et al*., 2009). The anesthetic agent that is to be used in IVRA should ideally yield short onset, long lasting anesthesia with a low dose and minimal side effects (Mehrdad Noroozi *et al*., 2016).

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Its effects results from the local anesthetic in the first place but also in a later phase being related to nerve compression and ischemia (Choyce, 2005)

Technique: An intravenous cannula is placed in the upper extremity to be blocked as distally as possible; the patient should also have an intravenous cannula in the non-operative upper extremity for administration of fluids and other drugs. Traditionally, a double tourniquet is placed on the operative side; both cuffs should have secure closures and reliable pressure gauges (Ronald *et al*., 2015). After exsanguination of the arm, the proximal cuff is inflated to approximately 150 mm Hg greater than the systolic pressure, and absence of a radial pulse confirms adequate tourniquet pressure. The total dose of local anesthetic is based on the patient's weight, and it is injected slowly (3 mg/kg of 0.5% prilocaine or lidocaine, without epinephrine). The use of bupivacaine for intravenous regional anesthesia has been associated with local anesthetic toxicity and death and is not recommended (Davies, 1984). However, dilute solutions (0.125% levobupivacaine) of long-acting amides and the addition of adjuvants (tramadol,

ketorolac, clonidine) have been used to prolong sensory block and analgesia after tourniquet deflation (Atanassoff *et al*., 2005). The onset of anesthesia is usually within 5 minutes. When the patient complains of tourniquet pain, the distal tourniquet, which overlies anesthetized skin, is inflated, and the proximal tourniquet is released. Use of a single, wide cuff allows use of smaller inflation pressures during intravenous regional anesthesia. The postulated advantage is that the smaller pressures will decrease the incidence of neurologic complications related to high inflation pressures with the narrow double cuffs (Pedowitz, 1991). The tourniquet can be released safely after 25 minutes, the tourniquet was deflated after the operation done, by deflation for 10 sec and re-inflated for 1 min repeated for 3 times (Steve *et al*., 2013) but the patient should be closely observed for local anesthetic toxicity for several minutes after the tourniquet release. Slow injection of local anesthetic solutions at a distal site decreases the risk of toxicity (Ronald *et al*., 2015; Duggan, 1984). In general, approximately 3 mg/kg (40 mL of a 0.5% solution) of preservative-free lidocaine without epinephrine is used for upper extremity procedures. For surgical procedures on the lower limbs, 50 to 100 mL of a 0.25% lidocaine solution can be used (Ronald, 2015).

Safety: IVRA is a potentially dangerous technique, involving direct iv injection of local anesthetic; therefore the following must apply:

- All equipment is checked for leaks and other defects.
- The patient is prepared and starved as for any anesthetic procedure, with resuscitative drugs and equipment available.
- Full monitoring is applied throughout.
- Rapid injection is avoided (may force solution past the tourniquet).
- Injection near the antecubital fossa is avoided (solution may be forced into the systemic circulation).
- The technique is used with caution in patients with severe arteriosclerosis and hypertension, since the tourniquet may not completely compress their arteries. Similar caution has been suggested in obesity (Steve *et al*., 2013).

Lidocaine: Amide local anaesthetic agent .its p*K*a is 7.9. 65% protein bound, 95% of an injected dose undergoes hepatic metabolism and is excreted renally. Onset is rapid by all routes; usual duration of action for 1% solution is about 1 h, increased to 1.5–2 h if adrenaline is added (Steve *et al*., 2013).

Indications

- Local anaesthesia. Often combined with adrenaline, since lidocaine tends to produce local vasodilatation; 1:200 000 and 1:80 000 solutions are commonly available, the latter usually restricted to dental use (Steve *et al*., 2013).
- iv administration:
- depression of laryngeal and tracheal reflexes (e.g. during tracheal intubation/extubation). Commonly used to reduce the increase in ICP caused by laryngoscopy.
- class I antiarrhythmic drug in ventricular tachyarrhythmias.
- may be useful in treatment of chronic pain (Steve *et al*., 2013).

Many preparations are available, including: 0.25–0.5% solutions for infiltration anaesthesia and IVRA., 1–2% solutions for nerve blocks and epidural anaesthesia, 4% solution for topical anaesthesia of the mucous membranes of the mouth, pharynx and respiratory tract, 10% spray for topical anaesthesia, also available in 1–2% gel for urethral instillation, and 5% ointment for skin, rectum and other mucous membranes. A constituent of EMLA cream. In other countries (especially the USA), 5% hyperbaric solution has been used in spinal anaesthesia (Steve, 2013)

Dosage

- Depends on the block.
- 1–2 mg/kg iv 2–5 min before intubation/extubation.
- For ventricular arrhythmias: 1 mg/kg iv initially, then 4 mg/kg/min for 30 min, 2 mg/kg/min for 2 h and 1 mg/kg/min thereafter.
- Maximal recommended dose: 3 mg/kg without adrenaline, 7 mg/kg with adrenaline. Toxic plasma levels: above about 10 µg/ml (Steve *et al*., 2013)

Mechanism of action: Bind to fast sodium channels in the axon membrane from within, preventing sodium entry during depolarisation. The threshold potential is thus not reached and the action potential of the nerve not propagated (Steve *et al*., 2013).

Side effects: Hypotension, Hypertension, Tachycardia, Arrythmia (SVT), Bradycardia, Pulmonary oedema, Headache, Nausea, Abdominal pain, Increased joint pain, Chest pain and siezure (Raphael *et al*., 2002)

Paracetmol: Paracetamol was first synthesized in 1878 by Morse, and introduced for medical usage in 1883. However, due to misinterpretation of its safety profile, it enjoyed only limited use until the 1950s, when the chemically similar, and up until then preferred analgesic, phenacetin was withdrawn because of renal toxicity (Chhaya *et al*., 2013).

Mechanism of action: Although paracetamol was discovered over 100 years ago and has been widely used in medical practice for more than half the century, its mechanism of action has not been elucidated until now (Howard, 2009). It has analgesic and antipyretic properties similarly to NSAIDs, but contrary to them, it does not possess any anti-inflammatory activity. When applied in recommended doses, it does not induce typical for NSAIDs gastrointestinal side effects. However, it suppresses prostaglandin production likewise NSAIDs (Marta Jewiak, 2014). There is evidence for a number of central mechanisms, including effects on prostaglandin production, and on serotonergic, opioid, nitric oxide (NO), and cannabinoid pathways, and it is likely that a combination of interrelated pathways are in fact involved (Jahr, 2013).

Prostaglandin production: The enzyme responsible for the metabolism of arachidonic acid to the prostanoids (prostaglandins and thromboxanes), commonly referred to as cyclooxygenase (prostaglandin H2 synthetase -PGHS), and possesses two active sites: the COX and the peroxidase (POX) sites. The conversion from arachidonic acid to the prostanoids is in fact a two-stage process, requiring activity at the COX site to first produce the unsTable intermediate hydroperoxide, prostaglandin G2 (PGG2), which is then converted to prostaglandin H2 (PGH2) via POX. The enzymatic activity of COX relies on its being in the oxidized form and it is suggested that paracetamol interferes indirectly with this by acting as a reducing co-substrate at the POX site as seen in

Figure 1 (Bertolini *et al*., 2006). Figure 1: paracetamol in inhibition of prostaglandin production [17]

Serotonergic pathway: Serotonergic pathways are part of the descending pain system, originating in the brainstem nuclei, hypothalamus, and cortex, and interact with pain afferents in the dorsal horn. Serotonin receptors are present throughout the central nervous system, involved in a number of functions, including consciousness, mood, memory, and nausea and vomiting. It has become widely accepted that the activation of descending serotonergic pathways plays a key role in the action of paracetamol (Alloui *et al*., 2002).

Endocannabinoid enhancement: paracetamol is subject to deacetylation to p-aminophenol that in turn reacts with arachidonic acid affected by fatty acid amide hydrolase (FAAH), resulting in the formation of an active metabolite of the drug, the fatty acid amide N-arachidonoylphenolamine (AM404) (Hogestatt *et al*., 2005; Ottani *et al*., 2006). AM404 does not act directly on cannabinoid receptors, however, it increases activity of endocannabinoid system in an indirect way (Kelley *et al*., 2004). On one hand, this compound is a strong activator of the vanilloid receptor subtype 1 (TRPV1), being a ligand of receptors for cannabinoids CB1, and on the other hand, it leads to an increase in the endogenous pool of these compounds as an inhibitor of the endogenous cannabinoid (anandamide) reuptake (Zygmunt *et al*., 2000). Endogenous cannabinoids, e.g., anandamide, act antinociceptively both at the level of the spinal cord as well as the brain. The study on rats performed by Bertolini *et al*. presented that an earlier administration of the CB1 receptor inhibited AM404 activity and completely blocked analgesic action of paracetamol in the animals (Bertolini, 2006). Moreover, cannabinoids considerably lower body temperature through the activation of CB1 receptors in the pre-optic area (Marta Jewiak, 2014).

Pharmacokinetic: Paracetamol is almost exclusively metabolized by the liver. In adults, only 1–4 % is excreted in urine as unchanged paracetamol while the majority is excreted as paracetamol-glucuronide (47–62 %) or aracetamolsulphate (25–36 %). A smaller part (8–10 %) is oxidized by cytochrome P450 (including CYP2E1, but also CYP1A2 and possibly CYP3A4) into 3-hydroxyparacetamol and the toxic metabolite N-acetyl-p-benzoquinone- imine (NAPQI) [27].

Aim of the Study: To evaluate the effect of paracetamol on sensory and motor block onset time, sensory and motor recovery and postoperative analgesia, when added to lidocaine in IV regional anesthesia (IVRA).

Patients and methods: After the study proposal was approved by the Iraqi scientific council of anesthesiology committee, and informed consent was obtained from the patients to participate in the study. The study include 60 patients were randomly enrolled in double blinded prospective clinical study in Baghdad in Al Yarmouk teaching hospital from 1st of February 2016 to 1st of December 2016.

The inclusion criteria

- ASA I and II patients
- Upper limb (forearm and hand) elective surgery
- The expected time of surgery was 20 – 60 min planned for intravenous regional anesthesia.

Exclusion criteria

- Patient refusal
- Trauma patients .
- Any contraindicated to tourniquet .
- History of allergy or any contraindication to drugs used in the study
- Psychological problems
- Patients body weight below 65 kg.

The patients were divided into 3 groups

Group 1: patients received IVRA 0.5% lidocaine solution with total dose of 200mg diluted with normal saline to 40 ml total volume.

Group 2: patients received IVRA 200mg lidocaine solution with 300mg paracetamol 10 mg / ml (total volume 40 ml).

Group 3: patients received IVRA 0.5% lidocaine solution with total dose of 200mg diluted with normal saline to 40 ml total volume with 300 mg paracetamol iv in the other cannula (systemic).

All patients were prepared for intravenous regional anesthesia by putting intravenous cannula in both limbs, monitoring of pulse rate, oxygen blood saturation, noninvasive blood pressure, double pneumatic tourniquet applied to the arm proximal to the operative site, exsanguination of the operative limb done and inflation of proximal tourniquet to a value equal to 150 mm Hg above the systolic BP. Conformation of circulatory isolation of the operative hand was verified by absence of radial pulse, inspection and loss of pulse oximetry tracing in the ipsilateral hand fingers. Then injection of the study drugs given. The sensory loss onset and motor loss onset time has been recorded in all groups, and the sensory recovery with motor recovery time also recorded . Assessment of onset of sensory block evaluated by pinprick of the skin in the thenar eminence (median nerve), hypothenar eminence (ulnar nerve) and first web space (radial nerve) and Assessment of onset of motor block evaluated by asking the patients to move his fingers. The distal tourniquet inflated after establishment of the sensory block followed by deflation of the proximal one. The tourniquet was deflated after the operation done in not less than 30 min, by deflation for 10 sec and re-inflated for 1 min repeated for 3 times. The pain intensity had been recorded according to visual analogue scale score (VAS) before the tourniquet inflation, just after proximal tourniquet inflation and 10 min after distal tourniquet inflation, and after 5 min of tourniquet deflation. Within the first six hours postoperatively the first time analgesic request was observed and recorded and the patients given 75mg diclofenac intramuscularly. In this study the age of the patients, gender, height, weight, BMI, PR, MAP, onset of sensory and motor loss, recovery of sensory and motor, and VAS score, all had been recorded and analyzed to see the relationship between the three groups.

Statistical analysis:

- Patients were randomly allocated by simple randomized method.
- Each patient assigned a serial identification number, the data were analyzed using the Statistical Package for Social Sciences (SPSS) version 20 and Microsoft excel 2010.

- The categorical data presented as means and percentage tables.
- The ANOVA test has been used to detect the correlation between either the age, sex, height, weight, BMI, onset of sensory and motor loss, recovery of sensory and motor, and VAS score
- The correlation was significant when p value was less than 0.05.

RESULTS

In analysis of the study data, there was no statistical significant relationship among the groups regarding the age, sex, height, weight and BMI as the P value was more than 0.05 as shown in Table 1. The sensory onset mean time for the all groups was 6.16 min with range of 4 – 8 minute, the fast group to achieve the sensory block was group 2 (5.3 min), the sensory recovery mean time was 5.95 min among the study groups and the slowest one to return the sensation was group 2 (7.8 min), the sensory onset and sensory recovery mean of all the study groups was shown in Table 2 and Figure 3: The motor onset mean time for the study groups was 10.7 min with range of 7-14 minute, the fast group to achieve the motor block was group 2 (8.3 min), the motor recovery mean time was 7.23 min among the study groups and the slowest one to return the motor was group 2 (8.75 min), the motor onset and motor recovery mean of all the study groups was shown in Table 3 and Figure 4: The onset time for sensory loss analysis ; there is a significant relationship between the group 1 and 2 (p value < 0.001), group 1 and 3 (p value 0.001), and group 2 and 3 (p value 0.023) and this mean that group 2 was statistically significant faster than both groups as shown in Table 4. The sensory recovery time was also shows a significant relationship between group 1 and 2 (p value < 0.001) and group 2 and 3 (p value < 0.001) but not between group 1 and 3 (p value 0.994), that is mean the group 2 is significantly slower for sensory recover than both groups as shown in Table 4. The motor loss onset time was shows a significant relationship between group 1 and 2 (p value < 0.001) and group 2 and 3 (p value < 0.001) but not between group 1 and 3 (p value 0.507), that is mean the group 2 is significantly faster to obtain motor loss than both groups as shown in Table 4. The motor recovery time was also shows a significant relationship between group 1 and 2 (p value < 0.001) and group 2 and 3 (p value < 0.001) but not between group 1 and 3 (p value 0.088), that is mean the group 2 is significantly slower for motor recover than both groups as shown in Table 4

DISCUSSION

Intravenous regional anesthesia (IVRA) technique is easy, reliable and cost effective with a high success rate of 94–98%when used in short operative procedures of hand or forearm (Brill, 2004; Singh, 2010). Many adjuvant drugs have been added to local anesthetics, such as, NSAIDs, paracetamol, opioids and adrenergic receptor agonists (Sen *et al*., 2009). In this study the mean age was 29.38 years, group 1 was 29.55, group 2 38.75 and group 3 was 28.85 . there were no statistical significant differences were seen among the groups regarding the age of the patients as p value between group 1 and 2 was 0.998, between group 1 and 3 was 0.932 and between group 2 and 3 was 0.891. In comparison of these results with other studies, Ayman Salah *et al*. (2013) (Egypt 2013), Haitham A. Azeem *et al*. (Egypt 2013), Noroozi *et al*. (Iran 2016), Huseyin Sen (Istanbul 2009) and Deogaonkar *et al*. (India 2016) show that there were no significant differences among the study groups regarding the age, and this was compatible with our study. Male was 53% and female was 47% in the study sample, group 1 male was 55% female was 45%, group 2 male was 60% female was 40%, and group 3 male was 45% female was 55%, there was no significant differences among the groups regarding the sex as p value between group 1 and 2 was 0.985 between group 1 and 3 was 0.899 and between group 2 and 3 was 0.726. During the comparison of this study with other studies Ayman Salah *et al*. (2013) Haitham A. Azeem *et al*. (2012) M. Noroozi *et al*. (2016) Huseyin Sen *et al*. (2009) and Deogaonkar *et al*. (2016) show that there were no significant differences among the study groups regarding the sex, and this was compatible with our study. The mean weight for the study groups was 80.41 kg, group 1 was 79.5 group 2 was 81.45 and group 3 was 80.3, there was no significant differences among the groups regarding the weight as p value between group 1 and 2 was 0.724 between group 1 and 3 was 0.971 and between group 2 and 3 was 0.907 .

In comparing this result with other studies; Ayman Salah *et al*. (2013), Haitham A. Azeem *et al*. (2012), M. Noroozi *et al*. (2016), Huseyin Sen *et al*. (2009) and Deogaonkar *et al*. (2016) show that there were no significant differences among the study groups regarding the weight, and this was compatible with our study. In the height mean was 173.8 cm, group 1 was 172.1 cm group 2 was 175.45 cm and group 3 was 173.85, there were no significant differences among the groups as p value between group 1 and 2 was 0.277, group 1 and 3 0.807 and group 2 and 3 was 0.859. In comparing this result with other studies;, M. Noroozi *et al*[5] and Huseyin Sen *et al*. (2009) show that there were no significant differences among the study groups regarding the height, and this was compatible with our study.

The mean BMI for the study sample was 26.63 kg/m^2, and for group 1 was 26.82 kg/m^2, group 2 was 26.45 kg/m^2 and group 3 was 26.6 kg/m^{2,} there were no significant differences among the groups regarding the BMI as p value between group 1 and 2 was 0.840 and group 1 and 3 was 0.968 and group 2 and 3 was 0.988, there were no data in other studies to compare the BMI with this study. In analysis of the sensory onset, the mean sensory onset for the group 1 was 7.15 min, group 2 was 5.3 min and group 3 6.05 min, during the analysis of these data, there were a significant differences seen between the groups, as the group 2 was the fast on to achive the sensory as compared with group 1 (p value was < 0.001) and between group 2 and 3 (p value was 0.023, while the group 3 was faster than group 2 (p value was 0.001). In comparing these results with other studies, in Ayman *et al*. (2013) the paracetmol when added to lidocaine in IVRA show a significant decrease in sensory onset time, in Haitham *et al*[30] study they uses a multi dose of paracetmol and seen that there was a significant decrease in the onset time of sensory block when use 300 mg paracetmol as compared with lower doses, in Noroozi *et al*[5] there was a significant decrease in sensory block onset when adding paractmol to the lidocaine in IVRA, and these was compatible with this study. In Sen *et al*. (2009) and Deogaonkar *et al*. (2016) they shows that the paracetmol when added with lidocaine as admixture in IVRA was faster than of the iv paracetmol and faster than the control but there was no significant difference among the groups in either adding the paracetmol to the lidocaine or when used as an iv, and this was incompatible with our study.

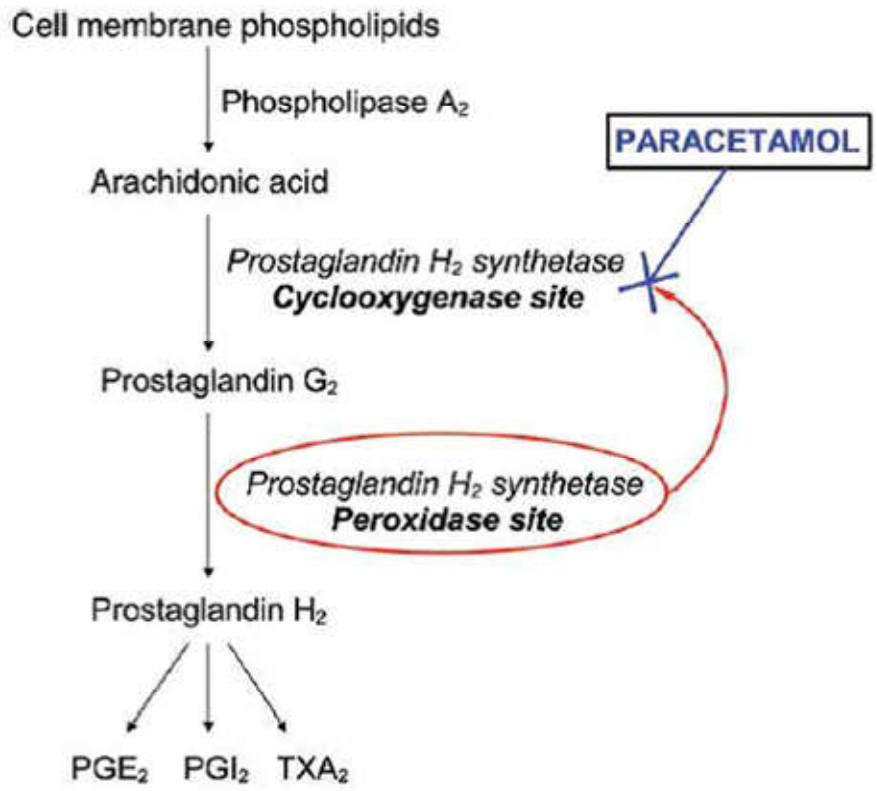

Figure 1. Paracetamol in inhibition of prostaglandin production (Chhaya, 2013)

Table 1. Demographic analysis among the study groups

Variable	Groups		Mean	Std. Error	P value
Age	1	2	29.75	1.19	0.998
		3	28.85	1.31	0.932
	2	1	29.55	1.19	0.998
		3	28.85	1.41	0.891
	3	1	29.55	1.31	0.932
		2	29.75	1.41	0.891
Sex	1	2	M 12 / F 8		0.985
		3	M 9 / F 11		0.899
	2	1	M 11 / F 9		0.985
		3	M 9 / F 11		0.726
	3	1	M 11 / F 9		0.899
		2	M 12 / F 8		0.726
Height	1	2	175.45	2.01	0.277
		3	173.85	2.18	0.807
	2	1	172.10	2.01	0.277
		3	173.85	2.26	0.859
	3	1	172.10	2.18	0.807
		2	175.45	2.26	0.859
Weight	1	2	81.45	2.08	0.724
		3	80.30	2.03	0.971
	2	1	79.50	2.08	0.724
		3	80.30	1.91	0.907
	3	1	79.50	2.03	0.971
		2	81.45	1.91	0.907
BMI	1	2	26.45	0.50	0.840
		3	26.60	0.55	0.968
	2	1	26.82	0.50	0.840
		3	26.60	0.51	0.988
	3	1	26.82	0.55	0.968
		2	26.45	0.51	0.988

Table 2: Sensory onset time and recovery among the study groups (in minutes)

	Onset	SD	Recovery	SD
Group 1	7.15	0.933	5.05	0.825
Group 2	5.3	0.864	7.8	1.005
Group 3	6.05	0.825	5	0.561
All groups	6.16	1.152	5.95	1.545

Table 3. Motor onset time and recovery among the study groups (in minutes)

	Onset	SD	Recovery	SD
Group 1	12.1	1.02	6.1	1.020
Group 2	8.3	0.801	8.75	1.069
Group 3	11.7	0.978	6.85	1.089
All groups	10.7	1.951	7.23	1.533

Table 4. Analysis of Sensory and motor onset and recovery among the study groups

Variable	Groups		Mean	Std. Error	p value
Sensory onset	1	2	5.3	0.284	< 0.001
		3	6.05	0.279	0.001
	2	1	7.15	0.284	< 0.001
		3	6.05	0.267	0.023
	3	1	7.15	0.279	0.001
		2	5.3	0.267	0.023
Sensory recovery	1	2	7.8	0.291	< 0.001
		3	5	0.223	0.994
	2	1	5.05	0.291	< 0.001
		3	5	0.258	< 0.001
	3	1	5.05	0.223	0.994
		2	7.8	0.258	< 0.001
Motor onset	1	2	8.3	0.290	< 0.001
		3	11.7	0.316	0.507
	2	1	12.1	0.290	< 0.001
		3	11.7	0.283	< 0.001
	3	1	12.1	0.316	0.507
		2	8.3	0.283	< 0.001
Motor recovery	1	2	8.75	0.331	< 0.001
		3	6.85	0.334	0.088
	2	1	6.1	0.331	< 0.001
		3	6.85	0.341	< 0.001
	3	1	6.1	0.334	0.088
		2	8.75	0.341	< 0.001

In sensory block recovery after the tourniquet deflation, the mean time in group 1 was 5.05 min, group 2 was 7.8 min and group 3 was 5 min, and this mean that the group 2 was the last one to return of the sensory with a significant differences between group 1 and group 3 (as p values between group 2 and them were < 0.001), while there was no significant differences between group 1 and 3 (p value 0.994). In the Aymen *et al*. (2013) and Haitham *et al*. (2012) shows that the IVRA paracetmol with lidocaine take more time significantly to return of sensory block than control group, and this was compatible with this study. The Sen *et al*. (2009) and Deogaonkar *et al*. (2016) shows that the IVRA paracetmol groups take more time significantly then the iv paracetmol and control but there was no significant difference when the paracetmol when used iv from the control group, and this was compatible with this study. The mean onset of motor block for group 1 was 12.1 min, group 2 was 8.3 min and group3 was 11.7 min, the group 2 was significantly the fast one to achieve the motor block as the p value between group 2 and group 1, group 2 and 3 were less than 0.001, but there was no significant difference between group 1 and 3 (p value 0.0507). In aymen *et al*. (2013), haitham *et al*. (2012) and Noroozi *et al*. (2016) they show that the paracetmol group was significantly faster than the control group in achieving the motor block which was compatible with this study. In Sen *et al*. (2009) and Deogaonkar *et al*. (2016) they shows that the IVRA paracetmol was significantly the fastest among the both control group and iv paracetmol, and these was compatible with this study.

The mean motor recovery time in group 1 was 6.1 min, group 2 was 8.75 and group 3 was 6.85, the group 2 was significantly the longest in recovery of motor function among both groups (p value less than 0.001), and the group 3 was longer than group 1 but not statistically significant (p value was 0.088).

In the comparison of these results with other studies, Aymen *et al*. (2013), Haitham *et al*. (2012), Sen *et al*. (2009) and Deogaonkar *et al*. (2016) all shows the IVRA paracetmol was significantly the longest as compared with control group, and longest from the iv paracetmol (in Sen *et al*. (2009) and Deogaonkar *et al*. (2016)), and this was compatible with this study. In the analysis of patient satisfactory for the pain relief before inflation and after deflation, the study shows that there was no significant differences among the three groups as the p value were more than 0.05. In Haitham *et al*. (2012) and Sen *et al*. (2009) there was no significant differences among the groups in VAS score at pre and post-operative time, and this was compatible with this study. In aymen *et al*. (2013), noroozi *et al*. (2016) and Deogaonkar *et al*. (2016), they shows that the lidocaine paracetmol group VAS score was significantly different at pre and post-operative score than the control group, and this was not compatible with this study. The first time requirement of analgesia in the group 1 was 1.9 hours and the group 2 was 5.6 hour and for group 3 was 4.05 hour, and this show that the group 2 had the significantly more time for the first time requirement for the analgesia (p value < 0.001) and the group 1 had significantly the fastest time for the analgesia requirement as compared with other groups (p value < 0.001). When compared with Aymen *et al*. (Ayman Salah, 2013) shows that the IVRA paracetmol had a long time for the first time analgesic requirement and this was compatible with this study. In sen *et al*. (Sen *et al*., 2009) and Deogaonkar *et al*. (2016) the systemic and IVRA paracetmol and lidocaine only groups had no significant differences regarding the first time requirement for analgesia, and this was not compatible with this study. There were no significant differences among the groups regarding the pulse rate or mean arterial blood pressure as p value among the groups in all time were more than 0.05, and this was compatible with Haitham *et al*.

(Reda, 2012) and Noorozi *et al*. (2016) but not compatible with Aymen *et al*. (2013) as they show that there were a significant differences among the groups regarding the pulse rate and MAP

Conclusion

- IVRA Paracetmol cause a decrease in sensory block onset time and motor block onset time than dose lidocaine alone or IVRA lidocaine with systemic paracetmol. .
- IVRA Paracetmol cause an increase in sensory recovery and motor recovery than dose lidocaine alone or IVRA lidocaine with systemic paracetmol.
- Systemic paracetmol with IVRA lidocaine cause a decrease in sensory onset time than lidocaine alone.
- There was no difference in VAS score between IVRA lidocaine alone, IVRA lidocaine with paracetmol or IVRA lidocaine with systemic paracetmol

Recommendation

It is better to add paracetmol to the lidocaine in IVRA to decrease the onset time and delay the sensory and motor recovery.

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