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ORIGINAL ARTICLE

Comparative Study between Intravenous and Intrathecal Dexmedetomidine in Spinal Anaesthesia in Patients Undergoing Elective Infra-Umbilical Surgery: the Effect on Postoperative Analgesia and Haemodynamic Parameters

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Background Management of postoperative pain is crucial to reduce central, peripheral, and immunological stress response. α2- adrenergic receptor agonists are commonly used as an adjuvant to intrathecal local anaesthetics to improve postoperative analgesia.

Objectives We compared intravenous versus intrathecal dexmedetomidine as an adjuvant to hyperbaric bupivacaine in patients undergoing elective infra-umbilical surgeries in supine position under spinal anaesthesia.

Methodology 100 patients randomized to two groups (50 each).for spinal anaesthesia with hyperbaric bupivacaine, intrathecally received 3.5ml [3ml (15mg bupivacaine) in both groups +0.5ml (saline versus 5µg dexmedetomidine in saline in Group A, B respectively)] .Slow intravenous infusion of 1µg/kg dexmedetomidine in 50ml saline versus 50ml saline was given to Group A and B respectively ten minutes before blockade.

Results Patients receiving intrathecal dexmedetomidine had a significantly longer motor and sensory blockade than those receiving intravenous dexmedetomidine (253.80±20.94 vs. 205.00±19.08; P<0.001) and (230.48±17.21 vs. 181.48±21.12; P<0.001) respectively. The intensity of pain was significantly lower in group B especially at t_6 and t_{12}. The total consumption of rescue analgesia was less in group B when compared to group A (100.30±21.63mg vs. 135.80±23.02mg respectively, P= 0.000). The incidence of hypotension was greater in the intravenous group with no statistical significance (34.0% vs 26.0%; P>0.05).

Conclusions The addition of 5 µg dexmedetomidine to 0.5% hyperbaric bupivacaine intrathecally can provide a better quality of postoperative analgesia with no significant side effects as compared to intravenous dexmedetomidine.

Keywords Dexmedetomidine; Intrathecal; Intravenous; Spinal Anaesthesia.
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INTRODUCTION

Spinal anaesthesia is a commonly used central neuraxial blockade often applied for lower abdominal, pelvic, and lower limb surgeries to reduce postoperative complications when compared to general anaesthesia. The most used local anaesthetic is hyperbaric bupivacaine, which acts by blocking voltage-gated sodium channel [1].

Postoperative analgesia is of major concern due to the relatively short duration of the local anaesthetic. Insufficient postoperative pain control can produce various effects on quality of life, prolong the recovery time and decrease patient satisfaction [2].

Opioids such as morphine and fentanyl are extensively added to local anaesthetics in neuraxial blockade to enhance the duration of postoperative analgesia. However, adverse effects, such as pruritus, urinary retention, postoperative vomiting, and respiratory depression, limit their usage [3,4]. Parenteral opioids can provide effective pain release, but may also cause systemic side effects as nausea and respiratory depression Non-steroidal anti-inflammatory drugs (NSAIDS), Patient's control analgesia (PCA) are further methods that can provide effective pain release, however their side effects cause limitations [4].

Dexmedetomidine (a highly selective α2-agonist) has been increasingly used as an adjuvant to local anaesthetic for spinal anaesthesia. This combination is associated with a lot of benefits, including reducing the use of analgesics, improving the intraoperative nerve blockade, shortening the onset time of the sensory or motor block, lowering the occurrence of shivering, prolonging the postoperative analgesia, and reducing the postoperative pain score [5].

α2-receptors are found in many sites throughout the body including central nervous system, spinal cord and peripheral tissues [6]. Dexmedetomidine can prolong spinal anaesthesia when given intravenously [7] or intrathecally by its actions on the substantia gelatinosa in the spinal cord and locus coeruleus in the brain [8]. It leads to sedation without respiratory depression and by lowering the secretion of catecholamines, it can decrease the stress response and the perioperative haemodynamic variations [9].

In the present study, we compared the analgesic potency and duration of intravenous versus intrathecal dexmedetomidine as an adjuvant to hyperbaric bupivacaine in elective infra-umbilical surgeries under spinal anaesthesia.

METHODOLOGY

This prospective randomized comparative controlled double- blind clinical trial was authorized by the research ethical committee of the faculty of medicine, Ain Shams University (NO. FMASU MS 67/2021). Patient enrollment started in March 2021 and the study ended in September 2021. The principles of the declaration of Helsinki were followed. The study was conducted at the department of General surgery, Ain Shams University Hospitals.

The primary outcome was comparing the duration of post operative analgesia by identifying time to first analgesic request, in both groups. Time to first analgesic is defined as, the time from intrathecal injection till the patient's first request for analgesia.

The secondary outcomes were assessment of pain with the Visual Analogue Scale (VAS), comparing haemodynamic parameters, the undesirable side effects, assessment of motor blockade using modified Bromage scale and assessment of sedation using the Ramsay sedation score in both groups during the intraoperative and postoperative periods.

We enrolled 100 patients, aged 18-50 years old, of both sexes with American Society of Anaesthesiologists classification (ASA) I/II, who were scheduled for elective Infra-umbilical surgeries lasting 3 hours or less. All patients provided fully informed consent.

Exclusion criteria were refusal of patients, pregnant females, uncorrected coagulopathy, heart failure, neuropathy, uncontrolled hypertension, drug allergy to the study drugs, infection at the injection site or any other contraindications to spinal anaesthesia.

Patients were randomly assigned to one of the two groups using computer generated random numbers; Group A (n=50) to undergo spinal anaesthesia with 3ml of hyperbaric bupivacaine 0.5% with intravenous (I.V) 1µg/kg dexmedetomidine diluted in 50ml normal saline administered slowly IV (over 10 minutes), 10 minutes before the blockade, while patients in Group B (n=50) to receive 3ml of hyperbaric bupivacaine 0.5% plus intrathecal (I.T) 5µg dexmedetomidine diluted in 0.5ml normal saline, along with 50ml normal saline administered slowly IV (over 10 minutes) 10 minutes before the blockade.

After enrolling the patients, a sealed envelope containing the group allocation numbers was cracked open. A consultant anaesthesiologist with more than 5 years of experience in regional anaesthesia blind to the medications to be used, performed the spinal blocks.

Anaesthetic technique

Patients were fasted for at least 8 hours preoperatively. A wide bore I.V cannula (gauge 18 or bigger) was inserted, then 10ml of lactated Ringer's solution was administered. Standard monitoring including heart rate (HR), blood pressure (BP) and arterial oxygen saturation (SPO_2) were applied, and initial vital data were measured.

Dural puncture was performed using a 25-gauge Quincke needle in the sitting position at the L4-5 interspace or at L3-L4 interspace through a midline approach. Two syringes were prepared by an anaesthesiologist who was not involved in subsequent anaesthesia and data collection:

Patients in Group A received 3ml of bupivacaine 0.5% (15mg) plus 0.5ml of normal saline (Total volume: 3.5ml) along with 1µg/kg dexmedetomidine diluted in 50ml normal saline administered slowly I.V (over 10 minutes) 10 minutes before the blockade.

Patients in Group B received 3ml of bupivacaine 0.5% (15mg) plus intrathecal (I.T) 5µg dexmedetomidine diluted in 0.5ml normal saline (Total volume: 3.5ml) along with 50ml normal saline administered slowly I.V (over 10 minutes) 10 minutes before the blockade.

The patients were positioned in the supine position. The head was elevated by a pillow and oxygen was supplied to the patient via oxygen mask, set at 4L/min.

The level of sensory blockade was assessed after 10 minutes by pin prick test with 27-gauge hypodermic needle and surgery started after sensory level fixation at T7. Then sensory level was assessed until recovery time.

Patients with failed spinal anaesthesia (due to failed lumber puncture or in adequate analgesia) were excluded from the sample size and proceeded to general anaesthesia.

The degree of motor block was assessed by using the modified Bromage scale, after administration of spinal anaesthesia by 10 minutes until recovery time (0= no motor nerve block,1= unable to lift the leg, 2= unable to bend the knee, 3= unable to bend the ankle).

Heart rate (HR), mean arterial blood pressure (MAP) and oxygen saturation (Spo_2) were monitored and recorded every 5 minutes in the first thirty minutes then every 10 minutes in the second thirty minutes, then once at 2 hours and 4 hours.

Sedation level was assessed using Ramsay sedation score 30 minutes after spinal anaesthesia, then in the postanaesthesia care unit (PACU) (1: patient anxious and restless; 2: patient co-operative, oriented and quiet; 3: patient responsive to commands; 4:responsive to a light snap of the eyebrows or a loud auditory stimuli; 5: unresponsive to light eyebrow snapping or loud auditory stimuli; 6: no response).

Side effects such as bradycardia, hypotension, nausea, vomiting, and shivering were documented and managed. Hypotension was defined as SBP <90mmHg or >20% decrease from baseline, and bradycardia was defined as HR <50 beats per minute. Hypotension was managed with 250ml lactated Ringer's solution and 3-6mg ephedrine I.V, and 0.01mg/kg I.V atropine was administrated to manage bradycardia.

In the PACU, pain was assessed by Visual Analogue Scale (VAS) and recorded at 1, 3, 6, 12 and 24 hours in the postoperative period (t_1, t_3, t_6, t_{12} and t_{24}). The scores of 1–3, 4–7 and 8–10 represent mild, moderate, and severe pain respectively. Diclofenac sodium 1mg/kg slow I.V infusion was given as a rescue analgesia if VAS was more than 3, it could be repeated after 8 hours with a maximum dose of 150mg per day. If pain persisted, I.V pethidine 50mg was given in between in order to keep VAS equal or less than 3. Duration of effective analgesia (The time interval between intrathecal injection till the patient's first request for analgesia), the number of requests, along with the total dosage of the administered diclofenac sodium and pethidine were recorded for 24hrs.

All data were recorded by an assistant who was blinded to the drugs given.

Sample Size calculation

Using PASS 11 program for sample size calculation, setting power at 80%, alpha error at 5%, reviewing results from a previous study by Sharma *et al.,* [10], showed that the time to request for first rescue analgesia (diclofenac) was prolonged in subarachnoid block, median [IGR] 5(6-7.5) hours than intravenous group, median [IGR] 4(2-4.5). Based on these results, a sample size of at least 50 patients per group was needed.

Statistical analysis

Data were collected, revised, coded, and entered to the Statistical Package for Social Science (IBM SPSS) version 23. The quantitative data were presented as mean, standard deviations and ranges when parametric and median, inter-quartile range (IQR) when data found non-parametric. Also, qualitative variables were presented as number and percentages. The comparison between groups regarding qualitative data was done by using Chi-square test and/or Fisher exact test when the expected count in any cell found less than 5. The comparison between two groups regarding quantitative data and parametric distribution was done by using Independent *t*-test while with non-parametric distribution was done by using Mann-Whitney test. The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the *p*-value was considered significant as the following: *P*-value >0.05: Non-significant (NS), *P*-value <0.05: Significant (S), *P*-value <0.01: Highly significant (HS).

RESULTS

A total of 100 patients were assessed for eligibility of the study and fulfilled all the inclusion criteria. They were randomized into two equal groups, group A and group B, each consisted of 50 patients. All the patients completed the study.

Table (1) presents the demographic data of all 100 patients enrolled in the study, including age, gender, ASA classification, and duration of surgery.

Table (2) compares demographic variables between Group A (IV dexmedetomidine) and Group B (IT dexmedetomidine), confirming no statistically significant differences.

Table (3) compares mean arterial blood pressure (MAP) between both groups at various intra- and post-operative time intervals, showing no significant difference.

Table (4) displays side effects such as nausea, vomiting, shivering, hypotension, and bradycardia between the two groups, with no statistically significant differences.

Table (5) compares clinical parameters including sensory block level at 10 minutes, sensory and motor blockade recovery times, Bromage scale, and Ramsay sedation scores between both groups. The duration of sensory blockade was significantly prolonged in the intrathecal group B compared to the intravenous group A (230.48±17.21 vs. 181.48±21.12 minutes). Similarly, the duration of motor blockade was significantly longer in group B (253.80±20.94 minutes) compared to group A (205.00±19.08 minutes).

Table (6) compares postoperative pain using VAS scores at 1, 3, 6, 12, and 24 hours between both groups, showing significantly lower pain scores in Group B at 6 hours (t_6) and 12 hours (t_{12}).

Table (7) compares rescue analgesia data between both groups, including time to first analgesic request, number of requests, total diclofenac dose, and need for pethidine. The time to first rescue analgesia was significantly prolonged in the intrathecal group B (364.80±22.15min) compared to the intravenous group A (240.60±35.59min). Additionally, the total dose of diclofenac sodium required was lower in the intrathecal group B (100.30±21.63mg) compared to the intravenous group A (135.80±23.02mg).

Table 1: Demographic data:

		Total *No*= 100
Age (years)	Mean±SD	34.52±9.61
	Range	18–50
Gender (No., %)	Female	48(48.0%)
	Male	52(52.0%)
ASA classification (No., %)	I	55(55.0%)
	II	45(45.0%)
Duration of surgery (hrs)	Median (IQR)	1.83(1.33−2.25)
	Range	0.83–3

Table 2: Comparison between both groups regarding demographic data:

***No*= 50**		**Group A**	**Group B**	**Test value**	***P*-value**
		***No*= 50**	***No*= 50**		
Age (years)	Mean±SD	33.38±9.12	35.66±10.05	-1.188•	0.238
	Range	18–50	18–50		
Gender	Female	26(52.0%)	22(44.0%)	0.641*	0.423
	Male	24(48.0%)	28(56.0%)		
ASA classification	I	31(62.0%)	24(48.0%)	1.980*	0.159
	II	19(38.0%)	26(52.0%)		
Duration (hrs)	Median (IQR)	1.83 (1.33–2.25)	1.83 (1.33–2.25)	-0.288≠	0.774
	Range	0.83–3	0.83–2.83		

P-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant; *: Chi-square test; •: Independent *t*-test; ≠: Mann-Whitney test.

Table 3: Comparison between both groups regarding mean arterial blood pressure (MAP):

MAP (mmHg) *No*= 50		Group A *No*= 50	Group B *No*= 50	Test value	*P*-value	Sig.
Baseline	Mean±SD	94.22±6.75	92.06±6.45	1.636•	0.105	NS
	Range	78–103	77–103			
5 mins	Mean±SD	89.02±10.59	87.30±8.65	0.889•	0.376	NS
	Range	60–101	62–99			
10 mins	Mean±SD	86.76±8.50	85.60±8.86	0.668•	0.506	NS
	Range	69–99	65–99			
15 mins	Mean±SD	88.10±9.10	87.88±7.65	0.131•	0.896	NS
	Range	62–99	65–99			
20 mins	Mean±SD	86.92±6.70	85.64±6.19	0.992•	0.323	NS
	Range	71–99	74–96			
25 mins	Mean±SD	86.58±6.47	85.40±5.94	0.950•	0.344	NS
	Range	72–99	74–96			
30 mins	Mean±SD	86.50±6.31	86.36±5.95	0.114•	0.909	NS
	Range	72–97	75–97			
40 mins	Mean±SD	86.78±6.34	86.28±6.20	0.399•	0.691	NS
	Range	74–97	75–98			
50 mins	Mean±SD	88.02±6.13	86.28±6.20	1.411•	0.161	NS
	Range	76–101	75–98			
60 mins	Mean±SD	90.04±5.23	88.82±6.64	1.019•	0.311	NS
	Range	77–98	76–100			
2 hours	Mean±SD	90.52±5.19	89.54±6.33	0.846•	0.399	NS
	Range	78–98	78–101			
4 hours	Mean±SD	91.00±5.05	91.18±6.07	0.161•	0.872	NS
	Range	79–98	79–102			

P-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant; •: Independent *t*-test.

Table 4: Comparison between both groups regarding side effects:

No= 50		Group A *No*= 50	Group B *No*= 50	Test value	*P*-value	Sig.
Nausea (NO., %)	No	48(96.0%)	49(98.0%)	0.344*	0.558	NS
	Yes	2(4.0%)	1(2.0%)			
Vomiting (NO., %)	No	49(98.0%)	50(100.0%)	1.010*	0.315	NS
	Yes	1(2.0%)	0(0.0%)			
Shivering (NO., %)	No	50(100.0%)	50(100.0%)	–	–	–
	Yes	0(0.0%)	0(0.0%)			
Hypotension (NO., %)	No	33(66.0%)	37(74.0%)	0.762*	0.383	NS
	Yes	17(34.0%)	13(26.0%)			
Bradycardia (NO., %)	No	45(90.0%)	46(92.0%)	0.122*	0.727	NS
	Yes	5(10.0%)	4(8.0%)			

P-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant; *: Chi-square test.

Table 5: Comparison between both groups regarding other parameters:

No= 50		Group A	Group B	Test value	*P*-value	Sig.
		No= 50	*No*= 50			
Sensory blockade after 10 mins	Th2	0(0.0%)	5(10.0%)	5.263*	0.022	S
	Th3	0(0.0%)	3(6.0%)	3.093*	0.079	NS
	Th4	7(14.0%)	17(34.0%)	5.482*	0.019	S
	Th5	9(18.0%)	7(14.0%)	0.298*	0.585	NS
	Th6	20(40.0%)	10(20.0%)	4.762*	0.029	S
	Th7	7(14.0%)	8(16.0%)	0.078*	0.780	NS
	Th8	7(14.0%)	0(0.0%)	7.527*	0.006	HS
Recovery time of sensory blockade in minutes	Mean ± SD	181.48±21.12	230.48±17.21	-12.716•	0.000	HS
	Range	151–210.25	203.9–256.93			
Modified Bromage scale after 10 mins	Bromage 1	43(86.0%)	47(94.0%)	1.778*	0.182	NS
	Bromage 2	7(14.0%)	3(6.0%)			
Recovery time of motor blockade in minutes	Mean±SD	205.00±19.08	253.80±20.94	-12.181•	0.000	HS
	Range	140–236	221–286			
Ramsay sedation score after 30 mins	Median (IQR)	3(3–4)	2(2–2)	-5.674≠	0.000	HS
	Range	2–4	2–4			
Ramsay sedation score in PACU	Median (IQR)	1(1–2)	2(2–2)	-6.139≠	0.000	HS
	Range	1–2	1–3			

P-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant; *: Chi-square test; •: Independent t-test; ≠: Mann-Whitney test.

Table 6: Comparison between both groups regarding VAS score:

VAS score		Group (A)	Group (B)	Test value	*P*-value	Sig.
No= 50		*No*= 50	*No*= 50			
1 hour (t_1)	Median (IQR)	0(0–0)	0(0–0)	0.000	1.000	NS
	Range	0–0	0–0			
3 hours (t_3)	Median (IQR)	2(1–2)	0.5(0–1)	7.566	0.000	HS
	Range	1–2	0–1			
6 hours (t_6)	Median (IQR)	5(5–6)	3.5(3–4)	-6.256≠	0.000	HS
	Range	3–8	2–7			
12 hours (t_{12})	Median (IQR)	3(3–4)	2(2–3)	-5.060≠	0.000	HS
	Range	2–6	1–5			
24 hours (t_{24})	Median (IQR)	2(2–2)	2(1–2)	-0.795≠	0.426	NS
	Range	1–3	1–4			

P-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant; ≠: Mann-Whitney test.

Table 7: Comparison between both groups regarding rescue analgesia:

***No*= 50**		**Group A**	**Group B**	**Test value**	***P*-value**	**Sig.**
		***No*= 50**	***No*= 50**			
1st rescue analgesia in minutes	Mean±SD	240.60±35.59	364.80±22.15	-20.947•	0.000	HS
	Range	120–270	300–450			
Number of requests	1	0(0.0%)	31(62.0%)	61.026*	0.000	HS
	2	20(40.0%)	19(38.0%)			
	3	30(60.0%)	0(0.0%)			
Dose of diclofenac sodium (mg)	Mean±SD	135.80±23.02	100.30±21.63	7.947•	0.000	HS
	Range	95–150	70–150			
Number of patients requesting pethidine	No	35(70.0%)	43(86.0%)	3.730*	0.053	NS
	Yes	15(30.0%)	7(14.0%)			

P-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant; *: Chi-square test; •: Independent *t*-test.

DISCUSSION

This prospective randomized study compared intravenous versus intrathecal dexmedetomidine in spinal anaesthesia in patients undergoing elective infraumbilical surgeries. Our results showed that the duration of analgesia, duration of sensory and motor blockade in group B were significantly longer than those of group A. As regards sedation, Ramsay sedation score was higher in group B during the recovery period. Both groups showed no statistically significant difference as regard heamodynamic stability with comparable incidence of side effects.

Prolongation of the subarachnoid block after IV administration of dexmedetomidine is by supraspinal action [11]. This action occurs due to the stimulation of the adrenoceptors in the locus coeruleus [12]. Hyperpolarization of noradrenergic neurons which suppresses neuronal firing in the locus coeruleus leads to inhibition of the descending medulospinal noradrenergic pathway and inhibition of norepinephrine release results in hypnotic and supraspinal analgesic effects of dexmedetomidine [13].

In our study, the duration of sensory blockade was significantly prolonged in the intrathecal group B as compared with the intravenous group A (230±17.21 vs 181.48±21.12). Such finding was also documented by a related study [10].

Also, the duration of motor blockade in our study was significantly prolonged in the I.T group B as compared with the I.V group A (253.80±20.94 vs 205.00±19.08). Our finding coincides with Sharma *et al.,* [14]. Also, Alshwadfy *et al.,* found that the duration for motor block regression to Bromage 1 (min) was (140.17±29.23 - 164.17±45.32 - 230.17±58.93) in control, I.V and I.T group respectively [15].

We observed comparable VAS scores in both groups especially after 6hr. and 12hr. (at t_6 and t_{12}) which was less in the intrathecal group B as compared to the intravenous group A. This effect of dexmedetomidine may be due to inhibition of pain receptors at the spinal cord and through its action on supra-spinal site and peripheral tissues after systemic absorption. This observation is in line with the findings presented by Khosravi *et al.,* [16] and Liu *et al.,* who performed their study on 90 patients divided equally into 3 groups, found also that at 5hr postoperatively, the VAS scores of the intrathecal group were lower than those of the intravenous group and the control group, and the difference was statistically significant (P<0.001) [17].

The time to rescue analgesia was significantly prolonged in the intrathecal group B as compared to the intravenous group A (364.80±22.15min vs 240.60±35.59min). Similar results were observed by Alshwadfy *et al.,* who found that the time to first request of analgesia (min) was (262.83±29.82 - 376.17±69.55 - 345.50±54.29) in control, IT and IV group respectively [15].

Another study compared I.V dexmedetomidine 0.5µg/kg immediately after spinal anaesthesia and intrathecal dexmedetomidine 3µg, they found that the duration of sensory block in the intrathecal group was much longer. Our findings are in line with those of this research [18].

However, contradictory result was observed by Elgebaly as the requirement of first rescue analgesic was significantly earlier in the intrathecal group as compared to the intravenous group (270.15±25.00 vs 371.25±88.54min). This may have occurred as the I.V dexmedetomidine

was given as loading dose [1µg/kg] followed by maintenance infusion [0.4µg/kg/h] throughout the study interval [19].

Regarding the total dose of diclofenac sodium required in our study, the patients in the intrathecal group B required less rescue analgesic as compared to the I.V group A (100.30±21.63 vs 135.80±23.02). Also, number of patients requiring pethidine was less in group B compared to group A (14% versus 30%). This result coincided with a previous study which found that the median dose of diclofenac and tramadol consumption over a period of 24 hours was less in intrathecal group as compared to intravenous group (median [IQR]: 150[75–150]mg vs 195[150–225]mg, *P*= 0.000) respectively [10].

Dexmedetomidine can reduce blood pressure and heart rate due to its binding to α2 receptors in the locus coeruleus, decreasing the release of norepinephrine and inhibiting sympathetic activity [20]. Our findings revealed that mean arterial blood pressure (MAP) was not significantly different between the two groups in most of the study periods. 17 cases (34%) in group A versus 13 cases (26%) in group B, developed mild hypotension, all were managed with I.V infusion of 250ml lactated Ringer's solution. There was no statistical difference between the two groups.

Many have noted bradycardia as a prominent side effect with I. V dexmedetomidine, this may be due to the use of a bolus dose of 1µg/kg followed by infusion greater than 0.4µg/kg/hr [20-23]. However in our study, only 5 cases (10%) in Group A versus 4 cases (8%) in Group B, developed bradycardia with no significant difference between both groups. Cases were mild, and effectively managed with 0.01mg/kg I.V atropine. As we administered a small dose of dexmedetomidine by slow intravenous infusion over 10 min, this could possibly explain their low incidence following I.V dexmedetomidine. This finding agrees well with observations of Kaya *et al.,* [24].

Our study showed that the Ramsay sedation score during surgery was significantly higher in the intravenous group as compared with the intrathecal group (3 vs 2 respectively) however, the level of sedation during recovery room was higher in the intrathecal group than the intravenous group (2 vs 1 respectively). Such finding was also documented by Abdallah *et al.,* [11].

The occurrence of sedation can be explained by the binding of drug to α2 receptors in the locus coeruleus and its central effects on brain and brain stem. Moreover, the drug can be rapidly absorbed into the cerebrospinal fluid (CSF) and exerts its effects on α2 receptors in the spinal cord [16].

The incidence of nausea, vomiting, and shivering did not differ significantly between the two groups. The incidence of nausea was 4% in the intravenous group and 2% in the intrathecal group. Moreover, the incidence of vomiting was 2% in the intravenous group, in contrast to 0% in the intrathecal group. None of the patients had shivering in the postoperative period in either group. This was consistent with the results of other studies [14,17].

LIMITATIONS

The duration of analgesia was largely influenced by patients' subjective experience of pain and their request for rescue analgesia, rather than objective measures. Furthermore, this study did not explore the potential dose-dependent effects of intravenous or intrathecal dexmedetomidine.

CONCLUSION

We concluded that the addition of 5ug intrathecal dexmedetomidine as an adjuvant in spinal anaesthesia in infraumbilical surgeries, produces along and better quality of postoperative analgesia with minimal side effects than 1ug/kg intravenous dexmedetomidine.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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