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A Prospective Study of Analgesic Results of Intravenous Dexmedetomidine in Patients Undergoing Meshplasty for Inguinal Hernia Repair under Spinal Anaesthesia

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ABSTRACT
This research work was designed to study the sedative and analgesic effect following the administration of intravenous dexmedetomidine as an adjuvant to intrathecal hyperbaric bupivacaine. Patients having any contraindications to spinal anaesthesia (i.e. inability to maintain stillness during needle puncture, raised intracranial pressure, skin/soft tissue infection at the site of needle puncture, severe hypovolemia, coagulopathy, preexisting neurological disease), known allergy to study drug, heart block/dysrhythmia and patients on treatment with a-adrenergic antagonists were not included in the study. The average baseline values of haemodynamic data for both groups were statistically similar. The number of patients who experienced low blood pressure and slow heart rate was not significantly different between the two groups ($p = 0.139$ and $p = 0.128$, respectively). The infusions were continued throughout episodes of low blood pressure and slow heart rate and the intensity of these effects did not justify stopping the infusions at any time. Patients in group A showed a noticeably higher sedation score compared to those in group B ($p< 0.001$). The level of sedation was superior in the dexmedetomidine group compared to the placebo group. The occurrence of bradycardia was much greater in the dexmedetomidine group, but it was temporary and could be reversed by atropine.

INTRODUCTION

Spinal anaesthesia with local anaesthetic drugs is widely utilised for procedures on the lower abdomen. The pain reduction provided by spinal anaesthesia is very good when compared to intravenous or epidural anaesthesia. Spinal anaesthesia has several benefits compared to general anaesthesia, which makes it the preferred choice in modern surgical procedures. As it reduces the length of stay, it is cost-effective for both the patient and the hospital. It is appropriate for patients with respiratory conditions and aids in the prevention of intubation-related issues such as laryngospasm. It also aids in keeping the airway open and decreases bleeding[1]. One of the key drawbacks is the restricted length of time that may be obtained using local anaesthetics. In order to address this issue, different additives have been experimented with and proven effective.

The inclusion of adjuvant has additionally enhanced the benefits of regional anaesthesia, such as faster action onset, decreased need for local anaesthetic, reduced risk of local anaesthetic toxicity, prolonged sensory block, improved analgesic effectiveness, improved hemodynamic stability and extended duration of postoperative pain relief. a2-stimulants such as clonidine and dexmedetomidine have been utilised to extend spinal anaesthesia. In addition to sedation and pain relief, they also reduce the activity of the sympathetic nervous system and the body's stress reactions to surgery and anaesthesia[2]. Dexmedetomidine is a more specific a2-A receptor agonist in comparison to clonidine, having stronger sedative and analgesic effects. Several studies have demonstrated the effectiveness of intravenous (IV) dexmedetomidine in extending spinal anaesthesia while also offering satisfactory sedation and postoperative pain relief. This study aims to examine the effectiveness of this combination in our institution and compare the findings with prior studies conducted at other universities. We also evaluate the onset of sensory and motor blockade, Duration of sensory and motor blockade, Hemodynamic effects of intravenous dexmedetomidine on spinal anesthesia, Sedation levels and Adverse effects.

MATERIALS AND METHODS

The current investigation was carried out in a prospective double-blind randomised fashion. 60 individuals who were members of the American Society of Anesthesiologists (ASA) and had a physical status of either I or II, regardless of gender and were between the ages of 20 and 50, were included in the study. The study focused on patients who were scheduled for inguinal hernia repair surgery. Patients who were unable to remain still during needle puncture, had increased intracranial pressure, had a skin or soft tissue infection at the site of needle puncture, had severe hypovolemia, had a coagulopathy, had a pre-existing neurological disease, had a known allergy to the study drug, had heart block or dysrhythmia, or were being treated with a-adrenergic antagonists were not included in the study.

Every patient was assessed during the preoperative appointment one day before the operation. The patients provided signed consent after being informed. They underwent a thorough medical history and a comprehensive physical examination. Standard tests such as haemoglobin (Hb), bleeding time (BT), clotting time (CT), thorough urine examination and any other necessary tests were conducted and documented. The patients were required to abstain from eating for 6 hours before the scheduled surgery. They were given a 0.25 mg dose of alprazolam orally the night before surgery. Upon entering the operating room, patients received a pre-determined amount of lactated Ringer's solution based on their weight and were observed for non-invasive blood pressure (NIBP), pulse oximetry (SpO2), and electrocardiogram (ECG).

All patients were given 2.5 ml of 0.5% hyperbaric bupivacaine through a spinal injection. Patients were randomly assigned using a sealed envelope method to receive one of the following after a subarachnoid block: Group A (with a sample size of 30) Administered an initial dosage of 1 µg kg-1 dexmedetomidine over a period of 10 mins, starting 20 mins after the spinal block. Continued with a maintenance dose of µg kg^{-1} hr-1 dexmedetomidine until the completion of the procedure. A 50 ml syringe was filled with dexmedetomidine (100 µg ml-1) mixed with normal saline to create a concentration of 4 µg ml-1. Group B (n = 30) equal measured amount of normal saline as loading dose over 10 mins+ maintenance until end of surgery.

Following the spinal block, oxygen was given through a face mask and the maximum level of sensation, muscle block, pulse rate, non-invasive blood pressure, breathing rate, and pulse oximetry were measured. Hypotension, which is characterised by a reduction in systolic blood pressure of over 20% from the initial level or less than 90 mm Hg, was managed by administering increasing doses of ephedrine (3 mg) through an intravenous (IV) route. Additionally, intravenous fluids were rapidly infused as needed. Bradycardia, which is characterized by a heart rate (HR) lower than 50 bpm, was addressed by administering IV atropine at a dosage of 0.6 mg. Sedation was evaluated based on the Modified Wilson Sedation Scale.

Inclusion criteria:

- American society of Anaesthesiologists (ASA) grade I-II
- Age 30-60 years

Exclusion criteria:

- Hb < 8g/dl or
- Patients receiving calcium channel blockers , ACE inhibitors, clonidine and Beta blockers
- Patients on sedative medications, opioids and antidepressants prior to surgery

Preoperative preparation: Following a standard preoperative evaluation, the patient's Initial mean arterial pressure, heart rate and oxygen saturation were documented. IV line initiated. The patients were randomly divided into two groups of 30 each using a closed cover procedure. In the operating room, suitable equipment for airway control and emergency medicines were prepared. The lateral location of the operation table was verified. Patients were moved to the surgery room and placed.

A blood pressure monitor, pulse oximeter and ECG leads were connected to the patient without causing any harm or invasion. Baseline measurements of systolic and diastolic blood pressure, mean arterial pressure, pulse rate and oxygen saturation were taken before the operation. Patients received a 10ml kg dose of ringer lactate 15 mins before the sub-arachnoid block. On the right side, the skin on the back was cleaned with an antiseptic solution and covered with a sterile cloth.

RESULTS

All the patients in both trial groups had ASA status I. Group A consisted of 24 male and 6 female patients. Group B consisted of 25 male and 5 female patients. Statistically speaking, it was shown to be similar (p = 0.271). The age, weight, height and duration of surgery of patients in both groups were also similar (Table1).

The average baseline values of haemodynamic data for both groups were statistically similar. The number of patients who experienced low blood pressure and slow heart rate was not significantly different between the two groups (p= 0.139 and p= 0.128, respectively). The infusions were continued throughout episodes of low blood pressure and/or slow heart rate, and the intensity of these effects did not justify stopping the infusions at any time.Patients in group A showed a noticeably higher sedation score compared to those in group B (p< 0.001). Dexmedetomidine greatly decreased the need for diclofenac injection to relieve pain throughout the 24-hour period after surgery (p< 0.001) (Table 2). No additional issues such as lightheadedness, tiredness, itching, shaking, or headache were noticed in any of the two groups.

DISCUSSIONS

We examined the impact of intravenous dexmedetomidine administered as either a single dose or a combination of a single dose and continuous administration on sub-arachnoid anaesthesia using intra-thecal hyper baric bupivacaine. All patients in both trial groups had an ASA level of I. The age, weight, height, gender distribution and duration of operation of patients in both groups were also similar. Sub-arachnoid anaesthesia is a commonly used method of regional anaesthesia for procedures involving the lower abdomen and lower limbs. Various substances including opioids and alpha-2 agonists, when combined with intra-thecal hyperbaric bupivacaine (0.5%), have been found to extend the duration of sensory and motor blockage[2]. A medication that activates alpha-2 adrenoreceptors, like clonidine, when given through the spinal canal, into a vein, by mouth, is known to extend the duration of spinal anaesthesia[3,4]. Furthermore, the absence of respiratory depression makes dexmedetomidine an appropriate additive in many clinical scenarios. Intravenous dexmedetomidine has also been demonstrated to reduce the amount of anaesthesia needed during general anaesthesia[5]. A little dose of intravenous dexmedetomidine given during spinal anaesthesia prolongs the duration of motor and sensory blockade without creating any adverse effects[6]. Dexmedetomidine is a very specific alpha-2 agonist with a comparatively high ratio of alpha-2 to alpha-1 activity (1620:1) compared to clonidine (220:1)[7]. The half-life of elimination is 2-3 hrs. It affects alpha-2 receptors located in the locus coeruleus in the brain stem, resulting in sedative and analgesic effects. Activation of the post-synaptic receptors in the central nervous system reduces sympathetic activity, leading to a drop in heart rate and blood pressure. Moreover, dexmedetomidine infusion can lead to heightened stimulation of alpha-2 receptors in the spinal cord, which inhibits the transmission of pain signals. The sedative, memory loss and pain-relieving effects of low-dose dexmedetomidine infusions were investigated by Hall *et al*.[8] Both low and moderate

Table 1: Comparison of demographic parameters (Mean S.D.)

	Group A	Group B	p-value
Age (yrs)	35.31±9.99	34.29±10.82	0.302
Weight (kg)	67.00±9.84	69.83±9.24	0.074
Height (mt)	1.81±0.08	2.78±0.09	0.189
Duration of surgery (min)	47.89±18.39	44.65±14.59	0.153

Table 2: Intra-operative parameters

Parameter	Group A	Group B	p-value
Time of VAS = 4 (min)	135.01 ± 29.62	97.00 ± 13.40	<0.001
No. of patients having Hypotension	6	4	0.139
Bradycardia	5	4	0.128
Sedation score	0	30	
1	8	0	
2	20	0	
3	2	0	
4			
Mean ± S.D.	3.78±0.49	1.00±0.00	<0.001
No. of diclofenac injections 1	14	3	
2	14	21	
3	2	6	
Mean ± S.D.	1.69±0.68	3.09±0.52	<0.001

amounts of dexmedetomidine cause notable drowsiness. This sedation can be easily reversed by either verbal or physical stimulus. Once stimulated, the individuals are capable of carrying out a range of tasks that involve speaking, movement and thinking.

Whizar-Lugo *et al*.[9] gave either intravenous dexmedetomidine or intravenous clonidine to extend the duration of spinal anaesthesia caused by 15 mg of 0.5% hyperbaric bupivacaine in patients having open abdominal hysterectomy. Patients in the dexmedetomidine group were given 1 microgram per kg of dexmedetomidine, starting 20 mins after spinal anaesthesia and administered over 20 mins. They then received a continuous infusion of 0.5 micrograms per kg per hour until the end of the surgical procedure. In contrast, patients in the clonidine group received 4 micrograms per kg of clonidine as an infusion over 20 minutes, starting 20 mins after the spinal block. The duration of sensory and motor blockades was significantly extended in both the dexmedetomidine and clonidine groups. Tekin *et al*.[10] investigated the impact of intravenous dexmedetomidine on the length of spinal anaesthesia when using 4 mL of 2% prilocaine. Patients who were given an initial dosage of dexmedetomidine 1 g kg within 10 mins after spinal anaesthesia, followed by a continuous dose of 0.4 g kg h for 50 mins, saw a notable extension of both sensory and motor blockage. A study conducted by Al-Mustafa *et al*.[11] demonstrated that administering dexmedetomidine intravenously after spinal anaesthesia extends the duration of sensory and motor blockage. Spinal anaesthesia is a frequently employed method for inguinal hernia surgery due to its cost-effectiveness and ease of administration. Nevertheless, it is important to pay special attention to anxiety during surgery and pain management after surgery. Dexmedetomidine is a member of the imidazole subclass of a2-receptor agonists, which is comparable to clonidine. It demonstrates a significant level of specificity for the a2-receptor (a2/a1 1600:1) in comparison to clonidine (a2/a1 200:1), which classifies it as a full a2- agonist. The a2-agonists generate their calming and sleep-inducing effects by acting on a2-receptors in the locus ceruleus[12]. Dexmedetomidine has been discovered to provide its pain-relieving effects, both in the spinal cord and in areas above the spinal cord[13]. Dexmedetomidine is utilised as a sedative and analgesic In the intensive care unit for a duration of <24 hrs. Dexmedetomidine, in comparison to clonidine, is a more specific a2-adrenoceptor agonist. This allows for its use in higher doses for sedation and pain relief without the undesired vascular effects caused by activating a1-receptors. Moreover, it is a medication with a shorter duration of action compared to clonidine and has a reversal agent, atipamezole, to counteract its sedative effect. Dexmedetomidine provides a unique form of sedation where patients can be easily awakened while still maintaining their ability to breathe. During surgery, it helps maintain a steady blood flow by reducing the body's stress response. It might provide protection against ischaemia by reducing the neuroendocrine response during the preoperative period. Dexmedetomidine has qualities that make it appropriate for sedation and pain relief throughout the entire preoperative period. It can be used as a premedication, in addition to general and regional anaesthesia, and as a sedative and pain reliever after surgery[14]. Analgesic and sedative effects have been observed when dexmedetomidine is administered intra-thecally, epidurally, or intravenously as an adjuvant[15]. It also creates effective sedative levels in patients, which allows for patient participation and potentially improves operating circumstances without causing major respiratory depression[16]. Atipamezol is a particular and discerning a2- receptor antagonist that quickly and effectively reverses the calming and circulatory effects of intravenous dexmedetomidine[17].

In our research, the duration until VAS = 4 was 134.05±28.68 minutes in group D and 97.00±13.40 minutes in group P, which had a very strong statistical significance ($p<0.001$). The amount of diclofenac injections needed for pain treatment within the 24-hour postoperative period was considerably lower in group D compared to group P (1.60±0.61 vs 2.08± 0.49) ($p< 0.001$). Harsoor et al also observed that the length of pain relief, defined as the time from the

start of SAB until the first report of pain (VAS = 3), was significantly longer in group D compared to group C (222.8±123[4]. mins vs 138.36±21.62 mins, p< 0.001), even though a lower initial dose of 0.5 µg kg-1 was used. The analgesic effect is mainly attributed to the suppression of the locus ceruleus in s the brain stem, as they stated. Furthermore, the infusion of dexmedetomidine may lead to heightened activation of a2-receptors in the spinal cord, which in turn inhibits the transmission of pain signals. This impact appears to be facilitated by both pre-synaptic and post-synaptic a2-receptors. In their study, Whizar-Lugo *et al*.[18] observed that patients in the placebo group received their first dose of analgesics at 150 mins when their pain level reached 4/10 on the visual analogue scale (VAS). In comparison, patients who received dexmedetomidine had their first analgesic dose at 220 mins, while patients who received clonidine had their first dose at 240 mins after the surgery ended. The difference in timing was statistically significant, with dexmedetomidine patients receiving analgesics later than placebo patients (220 ± 30 mins vs 150 ± 20 mins, p< 0.05) and clonidine patients receiving analgesics even later than placebo patients (240 ± 20 mins vs 150 ± 20 mins, p< 0.05). There were no statistically significant differences between dexmedetomidine and clonidine (p> 0.05). They stated that when a2-adrenergic agonists are injected systemically or neuraxially, they provide pain relief via affecting the spinal level, specifically laminae VII and VIII of the ventral horns. The most widely recognised process involves the release of acetylcholine and nitric oxide. The locus ceruleus and the dorsal raphe nucleus are other significant central neuronal regions where these medicines have an effect, causing drowsiness and analgesia. We discovered that all the patients in group D did not have a sedation score of 1. A sedation score of 2 was observed in 6 patients, while a sedation score of 3 was observed in 18 patients in group D. Additionally, a patient in group D had a sedation score of 4. All patients in group P had a sedation score of 1. The average sedation score in group D was larger than in group P (2.76±0.48 vs 1.00±0.00) and was determined to be very significant (p< 0.001). Harsoor et al observed that the average intra-operative RSS in Grrddxx+oup D was 2.34±1.1, while in Group C, it was 2.0±0.0 (p= 0.034). Nevertheless, RSS was similar in both groups over the postoperative period. Dexmedetomidine induces drowsiness through its central action, which appears to vary according on the dosage. The majority of the patients who received dexmedetomidine were sedated, but could be easily awakened. All the patients had RSS <=3 during the observation period, which demonstrates the benefit of using a lower dosage. The observations we made align with the outcomes they obtained. Al-Mustafa *et al*.[18] also observed that the Ramsay sedation score (RSS) was 2 in all patients in group C and varied from 2-5 in group D, with a maximum score of 5 in three patients, 4 in nineteen patients and 3 in one patient. Additionally, the highest average sedation score (3.96 ± 0.55) was attained 30 mins after initiating the dexmedetomidine infusion. They described that dexmedetomidine induces drowsiness and anxiety reduction by attaching to a2-receptors in the locus ceruleus, which reduces the release of norepinephrine and blocks sympathetic activity, resulting in a decrease in heart rate and blood pressure. No complications such as dizziness, tiredness, itching, shaking or headache were seen in any of the two groups.

CONCLUSION

The current study concludes that The start of sensory and motor blockades were sooner in the dexmedetomidine group. The length of both sensory and motor blockades were extended in the dexmedetomidine group compared to the placebo group. Both groups exhibited similar hemodynamics. The level of sedation was superior in the dexmedetomidine group compared to the placebo group. The occurrence of bradycardia was much greater in the dexmedetomidine group, but it was temporary and could be reversed by atropine. There was a lower occurrence of post-operative shivering in the group that received dexmedetomidine compared to the group that received a placebo.

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