ORIGINAL ARTICLE

Effect of Intravenous Dexmedetomidine on Spinal Anaesthesia with Hyperbaric Bupivacine

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Abstract:

Background: Spinal anaesthesia is one of the commonest anaesthetic techniques for infra-umbilical surgeries. Administration of dexmedetomidine for sedation during spinal anaesthesia is found to prolong the duration of block. *Aim and Objectives:* To evaluating the effect of intravenous dexmedetomidine on block characteristic of spinal anaesthesia with hyperbaric bupivacaine. *Material and Methods:* A total of 120 patients scheduled for various elective surgeries under spinal anaesthesia with hyperbaric bupivacaine were included in the study. The patients were divided into two groups of each containing 60 subjects. Group D received 1µg/kg bolus dexmedetomidine over 10 minutes immediately after spinal anaesthesia followed by 0.5µg/kg/hr infusion till the end of surgery and Group S received similar amount of saline. Data collected include onset of sensory and motor blockade, time for two segmental regression of block, duration of analgesia and sedation score were noted. *Results:* Onset of sensory block was faster in group D (2.38±1.48 min) as compared to Group S (3.03±0.22 min). Onset of motor block was significantly faster in Group D (6.97±0.93 min) as compared to Group S (8.01±0.85 min). Time required for two segment regression was prolonged in Group D (122.67±7.15 min) as compared to Group S (65.76±4.71 min). Total duration of analgesia was also prolonged in group D (4.29 ±1.04 hr) compared to Group S (2.24±0.29 hr). *Conclusion:* Intravenous administration of dexmedetomidine prolongs the duration of sensory and motor blockade with arousable sedation.

Keywords: Spinal Anaesthesia, Bupivacaine, Dexmedetomidine

Introduction:

Spinal anaesthesia is the most commonly used technique for infra-umbilical and lower limb surgeries [1]. It has many advantages such as fast onset of action, technically easy to perform, better analgesia, good muscle relaxation and economical [2]. Most commonly used local anaesthetic in spinal anaesthesia is hyperbaric bupivacaine. Using local anaesthetic alone has some limitations like short duration of action and limited post-operative analgesic coverage. To prolong the duration of action of bupivacaine, variety of drugs have been used which include opioids, ketamine, midazolam, clonidine, dexmedetomidine etc [3].

Dexmedetomidine which is selective α-2 adreno-receptor agonist has both sedative and analgesic properties [4]. FDA approved its usage only in intravenous route and its application for sedation has been extensively studied. Recent studies have shown that intravenous dexmedetomidine prolongs the duration of spinal anaesthesia with minimal side effects [5].

This study was conducted to evaluate the effect of intravenous administration of dexmedetomidine on block characteristic of spinal anaesthesia with hyperbaric bupivacaine.

Material and Methods:

This was a prospective, randomised, double-blind study undertaken at a tertiary care setup after taking approval from our Institutional Ethics Committee. The study conducted on 120 patients of either gender, aged 20-70 years, of American Society of Anaesthesiologists (ASA) grade I or II, scheduled for various elective surgeries under spinal anaesthesia.

Exclusion criteria include known hypersensitivity to study drug (dexmedetomidine), severe cardio-vascular, renal, hepatic and thyroid disease patients. All patients were visited on the day prior to the surgery and explained in detail about the procedure and informed written consent was obtained.

They were kept nil per oral as perinstitutional protocol. Patients were randomly allocated to two groups, Group D [Dexmedetomidine (n=60)] and Group S [saline (n=60)] using computer generated random numbers on the day of surgery (Fig.1).

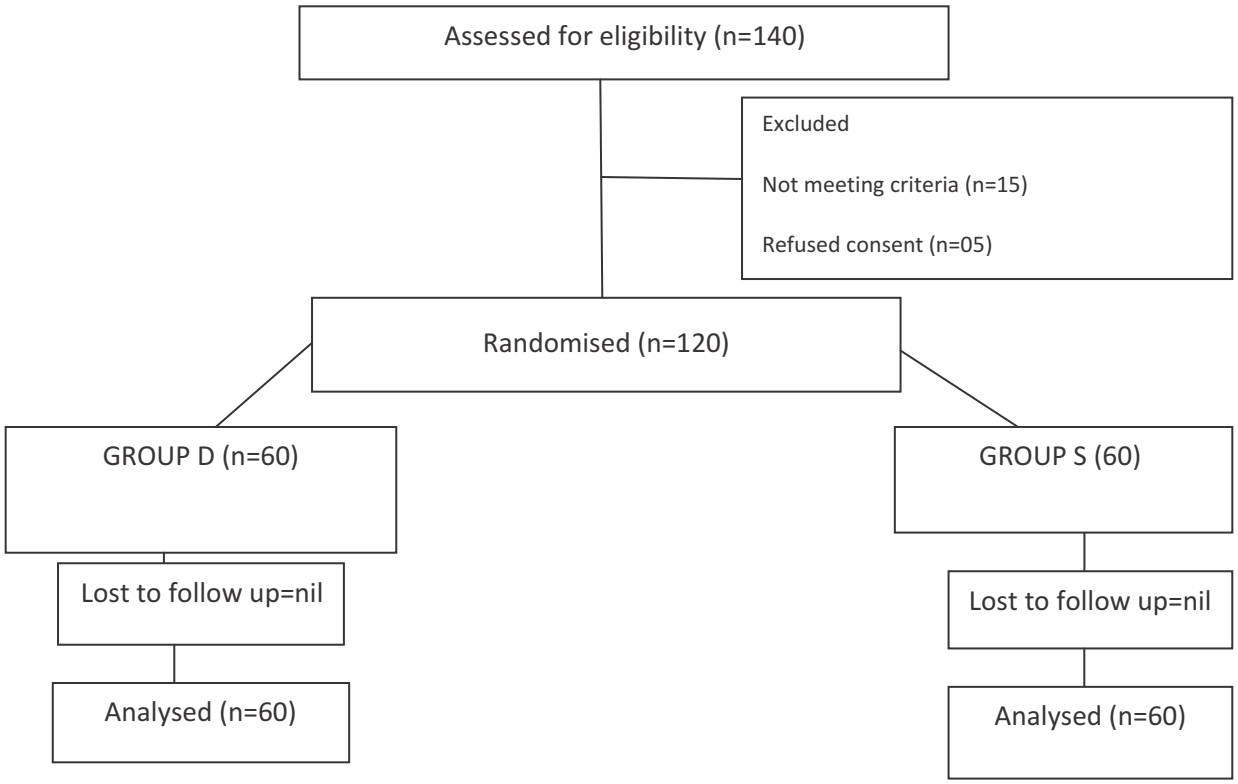

Fig. 1: Consort Flow Chart

All the patients were preloaded with 500ml of lactated Ringer's solution in preoperative room. Inside the operation theatre patients were connected with pulse oximetry, electrocardiogram and non-invasive blood pressure apparatus and basal values were recorded. Spinal anaesthesia administered in sitting position and at L3-L4 interspace. 15 mg of 0.5% hyperbaric bupivacaine was injected intrathecally. Supine position was adopted after administration of spinal anaesthesia. Group D received 1µg/kg slow bolus dexmedetomidine over 10 minutes immediately after spinal anaesthesia followed by 0.5µg/kg/hr infusion till end of surgery and Group S received saline in a similar manner. Parameters were recorded immediately after block and repeated every 3 minutes in first 30 minutes and cycled to 5 min till end of surgery. Intraoperative hypotension defined as SBP <100 mm Hg or fall >20% of baseline values and was treated with Injection Ephedrine 6 mg IV. Intraoperative bradycardia was defined as heart rate less than 50 bpm and was treated with injection Atropine 0.6 mg IV.

Data collected, included time of onset for sensory and motor block, regression of the sensory block and total duration of analgesia. Onset of sensory block was considered when the loss of temperature sensation to cold was noted at T10 dermatome. Motor block was assessed by Bromage score [6] and considered complete when the Bromage Score of 3 was achieved (Table 1). Assessment of pain was carried out using Visual Analogue Score (VAS). Total duration of analgesia was defined as time from administration of spinal anaesthesia toVAS $\geqslant$ 3. Injection Diclofenac was used as rescue analgesic. The level of sedation was assessed intraoperatively using Ramsay sedation score (Table 2) [7].

Sample size and dose estimation was based on work of Lugo *et al*. [8]. Assuming the difference of 15 % in duration of analgesia between two groups, with level of significance of 90 %, power of 80%, α error of 0.05 and β error of 0.2, statistical analysis showed 35 patients per group. We included 60 patients to increase the statistical strength. Independent t-test was carried out to find out significance in parametric data and non-parametric data were analysed using chi-square test. p - value< 0.05 was taken as significant.

Table 1: Bromage Score

Score	Bromage Scale
0	The patient is able to move the hip, knee and ankle
1	Patient is unable to move the hip but is able to move the knee and ankle
2	Patient is unable to move the hip and knee but is able to move the ankle
3	The patient is unable to move the hip, knee and ankle

Table 2: Ramsay Sedation Score

Sedation Score	Response
1	Anxious and agitated or restless or both
2	Co-operative, oriented and tranquil
3	Responding to commands only
4	Brisk response to light glabellar tap or loud auditory stimulus
5	Sluggish response to light glabellar tap or loud auditory stimulus
6	No response to stimulus

Results:

The two groups were similar regarding age, gender, weight and ASA grade (Table 3). Onset of sensory block was faster in Group D (2.38 ± 1.48 min) when compared to Group S (3.03 ± 0.22 min) and it was statistically significant (p=0.0010). Onset of motor block was significantly faster in Group D (6.97 ± 0.93 min) when compared to Group S (8.01 ± 0.85) with p value of < 0.0001. Time required for two segment regression was prolonged in Group D (122.67 ± 7.15 min) as compared to Group S (65.76 ± 4.71 min) and it was statistically significant (p<0.0001). Total duration of analgesia was also prolonged in Group D (4.29 ±1.04 hr) when compared to Group S (2.24 ± 0.29 hr) with p of <0.0001 (Table 4). Six patients in Group D and 2 patients in Group S had bradycardia (p=0.2723). Eight patients in Group D and 3 patients in Group S had hypotension (p=0.2057). No patients in either group had nausea and vomiting (Table 5). Mean sedation score in Group D was 2.51 ±0.67 and in Group S was 2.2± 0.14 (p= 0.0016) (Table 6).

Table 3: Demographic Data

Parameter	Group D (n=60)	Group S (n=60)	*P*
Age (years)	41.15 ± 9.92	40.55±6.44	0.695
Gender (M:F)	48:12	45:15	0.662
Weight (kg)	68.36±6.89	69.51±6.09	0.334
ASA (I/II)	35/25	38/22	0.708

Age and Weight are presented as mean ± SD. Test done was unpaired t-test.
n-Age number of patients; SD- standard deviation

Table 4: Characteristics of Spinal Anaesthesia

Parameters	Group D	Group S	*P*
Onset of sensory block (in min)	2.38±1.48	3.03±0.22	0.0010
Onset of motor blockade (in min)	6.97±0.93	8.01±0.85	<0.0001
Time taken for two segment regression (in min)	122.67±7.15	65.76±4.71	<0.0001
Duration of analgesia (in hour)	4.29 ±1.04	2.24±0.29	<0.0001

Analysis was done by student t-test

Table 5: Comparison of Incidence of Side-effects

Side effects	Group D	Group S	*P*
Bradycardia	6	2	0.272
Hypotension	8	3	0.206
Nausea	00	00	-
Vomiting	00	00	-

Analysis was done by chi square test.

Table 5: Comparison of Incidence of Side-effects

Score	Group D	Group S	P
Mean Sedation score	2.51 ±0.67	2.2± 0.14	<0.001

Analysis was done by student t-test

Discussion:

Administration of α2 adrenoreceptor agonists like clonidine and dexmedetomidine intravenously were known to prolong the duration of spinal anaesthesia [9]. Analgesic effect of α 2 adrenoreceptor agonist is mainly due to inhibition of locus ceruleus and inhibition of nociceptive impulse transmission at spinal cord level [4]. Clonidine, the first developed and widely used α2 adrenoreceptor agonist known to prolong the duration of spinal anaesthesia when given in oral, intravenous, intrathecal route. Dexmedetomidine is a newer α2 adreno receptor agonist, also prolong the duration of analgesia similar to clonidine but it differs from clonidine in more selectivity towards

α2 receptors [10]. Clonidine selectivity towards α1:α2 is 1:200 when compared to dexmedetomidine 1:1620 [11]. This greater selectivity makes dexmedetomidine more sedative and analgesic compared to clonidine with minimal side effects. Our study conducted to evaluate the effectiveness of intravenous dexmedetomidine on spinal anaesthesia with 0.5 % bupivacaine and results indicated that dexmedetomidine when given as bolus dose followed by intravenous infusion throughout the surgery hastened the onset of sensory block and motor block, prolonged the duration of spinal anaesthesia.

Lugo *et al.* [8] conducted the study to assess the effect of dexmedetomidine on spinal anaesthesia with hyperbaric bupivacaine, they used 1μg/ kg bolus followed by 0.5μg/kg/hr infusion prolonged the duration of sensory and motor block. In another study, it was conducted by Al-Mustafa *et al*. [9], there was also prolongation of both sensory and motor blockade with similar dose of dexmedetomidine. Results of both studies were similar to our study. In contrary to above, studies used bolus dose of dexmedetomidine didn't show prolongation of duration of motor blockade [12]. Literature analysis showed that this difference may be due to concentration dependent effect of α receptor agonists [13].

Our study showed dexmedetomidine hastened the onset of sensory and motor block. This is comparable to study conducted by Harsoor *et al*. in terms of onset of sensory blockade [14]. Though our study showed delay in onset in motor block as compared to Harsoor *et al*. [14] but it was similar in terms of faster onset in study group. Analysis of our study showed time for two segment regression was delayed in dexmedetomidine group and it was similar to study conducted by Gupta *et al.* [15] and Lugo *et al.* [8]. Dexmedetomidine induces sedative effects via centrally mediated post synaptic α2 adrenoreceptors [16]. Mean sedation score in Group D was 2.51 ± 0.67 and in Group S was 2.2 ± 0.14 ($p < 0.001$) and these results are comparable to study conducted by Kaya *et al*. [12] and Pathak *et al*. [17].

Hemodynamic effects of dexmedetomidine like changes in heart rate and blood pressure were also studied. Dexmedetomidine produces transient rise in blood pressure with reflex bradycardia, followed by hypotension [18]. These effects are seen after bolus dose with higher dose range. Initial response is due to its direct effects on β adrenoreceptors of smooth muscle in blood vessels and delayed response of hypotension is attributed to centrally mediated sympatholysis. Measures available for overcoming the initial response are slow infusion of drug, omitting the bolus dose etc. We followed slow infusion of study drug. We found out that number of patients who had bradycardia and hypotension were more in dexmedetomidine group but the significance was less and were comparable to other studies [18-19]. Incidence of nausea, vomiting were also recorded. No patients in either group had nausea and vomiting. These findings were comparable to other studies [19].

Conclusion:

Intravenous supplementation of dexmedetomidine in dose of 1μg/kg bolus followed by 0.5μg/kg/hr infusion fasten the onset of sensory block, prolong the duration of analgesia and increases the time for two segment regression with lesser incidence of bradycardia and hypotension.

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