

Evaluation of the Efficacy of Intravenous Ketamine on Prevention of Hypotension During Spinal Anaesthesia in Patients with benign Prostatic Hyperplasia

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ABSTRACT

Introduction: Hypotension is a common complication during spinal anaesthesia. The elderly patients due to reduced physiological reserves are more prone to develop adverse effect of hypotension. Hypotension following spinal anaesthesia is caused by paralysis of pre-ganglionic sympathetic fibers resulting in decrease systemic vascular resistance, decrease pre-load, decrease after-load, decrease stroke volume, decrease cardiac output resulting in decrease blood pressure. Current study aimed to evaluate the effects of intravenous ketamine on prevention of hypotension during spinal anaesthesia.

Material and Methods: Ninety (90) patients aged 50 to 70 yrs. (ASA I-II) undergoing surgery for Benign Prostatic Hyperplasia were randomly allocated to two groups – Group K receiving i/v ketamine and Group B without i/v ketamine. After securing i/v line, infusion started with R/L. Following spinal anaesthesia, ketamine was administered in the dose of .5 mg/kg b.w. to patients belonging to Group K.

Results: In both groups, spinal anaesthesia resulted in reduction in Mean Arterial Pressure (MAP). MAP was lower in the group without ketamine than in the ketamine group at all times. There was a significant change in heart rate in the control group compared to ketamine group ($p < 0.05$).

Conclusion: We concluded that ketamine .5 mg/kg b.w. given intravenously just after spinal anaesthesia resulted in greater hemodynamic stability in elderly patients undergoing transurethral resection of prostate compared with control group.

Keywords: Ketamine, Spinal Anaesthesia, Blood Pressure, Hypotension, Transurethral Resection, Benign Prostatic Hyperplasia.

INTRODUCTION

Spinal anaesthesia was pioneered in humans by a German surgeon Dr. August Bier on August 15th 1898 using Quinke method of entering the intrathecal space. It provides simple, effective and safe analgesia in the peri-operative period. It is widely used for transurethral resection of the prostate (TURP) because it allows earlier recognition of symptoms caused by occasionally seen complications of TURP-such as over hydration and hyponatremia.¹

However hypotension is a common complication during spinal anaesthesia and may result in serious adverse outcomes such as cerebral ischemia, thrombosis, reduced renal function, congestive heart failure and myocardial infarction. Elderly patients have reduced physiological reserve and are more prone to adverse effect caused by

paralysis of preganglionic sympathetic fibers. This reduces the systemic vascular resistance and both preload and afterload are diminished, resulting in subsequent reduction in stroke volume and cardiac output causing arterial blood pressure to fall.^{2,3}

Ketamine a phencyclidine derivative is used as an anesthetic agent for poor risk patients due to its relative safety of use and the beneficial effects on cardiovascular functions resulting from its sympathomimetic characteristics.⁴ Ketamine stimulates the cardiovascular system and produces increase in arterial blood pressure, heart rate, cardiac output, cardiac work and myocardial oxygen consumption. The mechanism is centrally mediated increase in sympathetic tone and circulating catecholamines with inhibition of the vagal nerve, inhibition of catecholamine re-uptake at the peripheral ganglia and tissues and norepinephrine release from the sympathetic ganglia.

Hence considering the adverse effects of spinal anaesthesia like hypotension and bradycardia and since establishment of cardiovascular stability is crucial in elderly patient undergoing TURP, ketamine at the dose of 0.5mg/kg body weight have been chosen to find out effectiveness in maintaining hemodynamic stability in patients undergoing spinal anaesthesia for TURP. Current study aimed to evaluate the effects of intravenous ketamine on prevention of hypotension during spinal anaesthesia with the objectives to determine the effects of intravenous ketamine on hemodynamic changes under spinal anaesthesia in patients undergoing surgery for Benign Prostatic Hyperplasia and to note any side effects associated with the said dose of study drug.

MATERIAL AND METHODS

After approval of the institutional ethical committee and

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after obtaining informed written consent from the patient, this prospective randomized clinical trial was conducted on 90 patients aged 50–70 years undergoing TURP for Benign Prostatic Hyperplasia (Grade 2 and 3 prostatic enlargement) under spinal anaesthesia at Assam Medical College & Hospital, Dibrugarh for a period of one year from July 2016–June 2017. All patients belonging to either ASA I or II were included in the study. Patients with co-morbid conditions and those patients unwilling for the trial were excluded from the study.

Patients were visited on the preoperative day for PAC. Preanaesthetic evaluation was performed in each patient including detailed history taking, thorough physical examination and routine preoperative investigations. The nature and procedure of the study was explained to the patients and written informed consent was obtained from each patient. All patients had routine preoperative fasting for 6 hours before surgery.

Using double blind technique, patients were divided into 2 groups with 45 patients in each group. Group K with Intrathecal Bupivacaine heavy 0.5% with intravenous ketamine and Group B with Intrathecal Bupivacaine heavy 0.5% without intravenous ketamine.

Patient's Preparation

On shifting the patient to O.T. noninvasive blood pressure monitor, pulse oximeter and ECG leads were connected and baseline values were recorded. I/V cannulation done and fluid infusion started with ringer lactate. Patients were given midazolam 0.03 mg/Kg body weight as pre-medication.

Procedure

The order of drug administration was randomized by draw lots and patients were enrolled into study in order of admittance. The solutions were prepared aseptically immediately before intravenous injection by an anaesthetist who was not one of the investigators.

Subarachnoid block was administered in sitting position with a 25G Quinke's spinal needle and three millilitres (15 mg) of bupivacaine was injected over 15 second into the subarachnoid space through L 4-5 intervertebral space. Study group patients received midazolam 0.03 mg/kg before giving spinal anaesthesia as premedication and ketamine 0.5 mg/kg i.v. in 2 cc after giving spinal anaesthesia. No prophylactic vasopressor was used. All patients were operated for TURP using 1.5% Glycine as irrigation fluid. The patients were discharged from the recovery room when the motor block was completely resolved. Other discharge criteria being stable vital signs, minimal nausea or vomiting and no severe

pain or bleeding.

The parameters that were observed are systolic blood pressure (SBP), Diastolic blood pressure (DBP), mean arterial pressure (MAP) and heart rate (HR) immediately before anaesthesia and 1 min after subarachnoid administration of bupivacaine, then at interval of every 3 min for 15 min, at 5 min intervals for the subsequent 30 min and then every half an hour till complete reversal of motor blockade. Patients received oxygen at a rate of 3 L/min through a nasal prong or mask during the procedure. Peripheral oxygen saturation and respiratory rates was measured preoperatively and perioperatively.

Patients were assessed by an investigator blinded to the study for motor and sensory blocks, sedation and side effects-such as nystagmus, dizziness, nausea and vomiting, and psychomimetic effects. Sensory block was assessed by pinprick test. Motor block was assessed by modified Bromage scores. Sedation was assessed every 15 min using a four-point scale (1 awake; 2, drowsy but responsive to verbal stimulus; 3, drowsy but responsive to physical stimulus; 4, unresponsive to verbal and physical stimulus). Offset of sensory block was assumed when bilateral sensation to pinprick test at the S2 dermatome was recovered. Complete motor recovery was assumed when modified Bromage score was zero. Duration of spinal analgesia was determined from the time of administration of spinal bupivacaine to patient's first complaint of pain during the postoperative period.

STATISTICAL ANALYSIS

The data collected was tabulated in Microsoft Excel Worksheet and computer based analysis was performed using the Statistical product and service solution (SPSS) 20.0 software (SPSS, Chichago, Illinois, USA) and Microsoft Excel 2010. Results on continuous measurements are presented as mean \pm standard deviation and are compared using students t test. Discreet data are expressed as number (%) and are analyzed using chi square test. For all analysis, the statistical significance was fixed at 5% (p value < 0.05).

RESULTS

Demographic profile and ASA physical status of the patients in both groups were comparable, and the differences between the groups were statistically not significant (P > 0.05) (Table 1).

The Table 2 shows intraoperative systolic blood pressure variation in the two groups. Systolic blood pressure was found to be significantly lower in Group B as compared to Group K at various intraoperative periods of the procedure.

	Group-K (Mean \pm S.D.)	Group-B (Mean \pm S.D.)	p value
Age Group (years)	60.80 \pm 3.90	60.56 \pm 4.98	0.796
ASA (I : II) (Number)	13 : 32	16 : 29	0.499
Height (cm)	152.29 \pm 7.13	152.24 \pm 5.77	0.974
Weight (Kg)	65.93 \pm 6.81	65.07 \pm 7.47	0.567
Duration of Surgery (in minutes)	62.20 \pm 4.57	61.69 \pm 4.24	0.584

Table-1: Demographic Profile

Time	Group	Mean	± S.D.	Average change	Percentage (%) Change	p value
Premedication	Group-K	131.96	3.99			
	Group-B	130.58	3.29			
Immediately after SAB	Group-K	123.64	4.34	8.31	6.298	<0.001
	Group-B	121.00	3.25	9.58	7.335	<0.001
At 3 minutes	Group-K	115.71	3.18	16.24	12.31	<0.001
	Group-B	108.11	3.64	22.47	17.21	<0.001
At 6 minutes	Group-K	112.58	3.19	19.38	14.69	<0.001
	Group-B	103.62	4.17	26.96	20.64	<0.001
At 9 minutes	Group-K	111.04	2.84	20.91	15.85	<0.001
	Group-B	101.31	3.85	29.27	22.41	<0.001
At 12 minutes	Group-K	111.40	3.58	20.56	15.58	<0.001
	Group-B	99.04	3.48	31.53	24.15	<0.001
At 15 minutes	Group-K	112.82	4.12	19.13	14.5	<0.001
	Group-B	96.96	3.69	33.62	25.75	<0.001
At 20 minutes	Group-K	115.78	3.75	16.18	12.26	<0.001
	Group-B	95.96	3.75	34.62	26.51	<0.001
At 25 minutes	Group-K	120.16	3.04	11.80	8.942	<0.001
	Group-B	99.62	4.91	30.96	23.71	<0.001
At 30 minutes	Group-K	122.24	3.39	9.71	7.359	<0.001
	Group-B	108.91	4.76	21.67	16.59	<0.001
At 60 minutes	Group-K	126.22	3.90	5.73	4.345	<0.001
	Group-B	125.98	4.43	4.60	3.523	<0.001
Recovery	Group-K	130.71	3.09	1.24	0.943	0.101
	Group-B	130.20	3.33	0.38	0.289	0.59

Table-2: Variation in systolic blood pressure in Group-K & group-B after subarachnoid block

Time	Group	Mean	± S.D.	Average change	Percentage (%) Change	p value
Premedication	Group-K	80.40	3.31			
	Group-B	81.07	3.48			
Immediately after SAB	Group-K	75.49	3.55	4.91	6.108	<0.001
	Group-B	73.84	3.66	7.22	8.909	<0.001
At 3 minutes	Group-K	72.09	4.23	8.31	10.34	<0.001
	Group-B	71.91	4.18	9.16	11.29	<0.001
At 6 minutes	Group-K	70.16	3.38	10.24	12.74	<0.001
	Group-B	68.33	4.26	12.73	15.71	<0.001
At 9 minutes	Group-K	68.47	2.30	11.93	14.84	<0.001
	Group-B	66.33	3.93	14.73	18.17	<0.001
At 12 minutes	Group-K	67.47	2.99	12.93	16.09	<0.001
	Group-B	65.36	3.61	15.71	19.38	<0.001
At 15 minutes	Group-K	65.71	3.60	14.69	18.27	<0.001
	Group-B	63.64	3.18	17.42	21.49	<0.001
At 20 minutes	Group-K	70.82	4.00	9.58	11.91	<0.001
	Group-B	60.71	1.62	20.36	25.11	<0.001
At 25 minutes	Group-K	72.13	4.00	8.27	10.28	<0.001
	Group-B	62.87	2.89	18.20	22.45	<0.001
At 30 minutes	Group-K	74.69	3.41	5.71	7.103	<0.001
	Group-B	66.84	4.01	14.22	17.54	<0.001
At 60 minutes	Group-K	75.78	3.57	4.62	5.749	<0.001
	Group-B	75.07	2.84	6.00	7.401	<0.001
Recovery	Group-K	78.91	2.87	1.49	1.852	0.025
	Group-B	78.31	3.39	2.76	3.399	<0.001

Table-3: Variation in diastolic blood pressure in Group-K & Group-B after subarachnoid block

Time	Group	Mean	± S.D.	Average change	Percentage (%) Change	p value
Premedication	Group-K	97.59	2.83			
	Group-B	97.57	2.74			
Immediately after SAB	Group-K	91.54	2.85	6.04	6.194	<0.001
	Group-B	89.56	2.67	8.01	8.207	<0.001
At 3 minutes	Group-K	86.63	2.88	10.96	11.23	<0.001
	Group-B	83.98	2.98	13.59	13.93	<0.001
At 6 minutes	Group-K	84.30	2.40	13.29	13.62	<0.001
	Group-B	80.10	3.40	17.47	17.91	<0.001
At 9 minutes	Group-K	82.66	1.96	14.93	15.3	<0.001
	Group-B	77.99	3.19	19.58	20.07	<0.001
At 12 minutes	Group-K	82.11	2.74	15.47	15.86	<0.001
	Group-B	76.59	2.78	20.99	21.51	<0.001
At 15 minutes	Group-K	81.41	3.07	16.17	16.57	<0.001
	Group-B	74.75	2.58	22.82	23.39	<0.001
At 20 minutes	Group-K	85.81	3.18	11.78	12.07	<0.001
	Group-B	72.46	1.65	25.11	25.74	<0.001
At 25 minutes	Group-K	88.14	2.96	9.44	9.678	<0.001
	Group-B	75.12	2.69	22.45	23.01	<0.001
At 30 minutes	Group-K	90.54	2.66	7.04	7.219	<0.001
	Group-B	80.87	3.21	16.70	17.12	<0.001
At 60 minutes	Group-K	92.59	2.74	4.99	5.116	<0.001
	Group-B	92.04	2.44	5.53	5.671	<0.001
Recovery	Group-K	96.18	2.35	1.41	1.442	0.012
	Group-B	95.61	2.48	1.96	2.012	<0.001

Table-4: Variation in mean arterial pressure in Group-K & Group-B after subarachnoid block

Time	Group	Mean	± S.D.	Average change	Percentage (%) Change	p value
Premedication	Group-K	73.09	4.39			
	Group-B	72.42	3.95			
Immediately after SAB	Group-K	71.24	3.85	1.84	2.524	0.037
	Group-B	70.87	4.00	1.56	2.148	0.067
At 3 minutes	Group-K	69.18	3.47	3.91	5.351	<0.001
	Group-B	67.60	3.38	4.82	6.658	<0.001
At 6 minutes	Group-K	67.22	3.54	5.87	8.027	<0.001
	Group-B	65.27	3.35	7.16	9.88	<0.001
At 9 minutes	Group-K	66.04	3.38	7.04	9.638	<0.001
	Group-B	63.20	3.65	9.22	12.73	<0.001
At 12 minutes	Group-K	65.16	3.39	7.93	10.85	<0.001
	Group-B	62.38	2.71	10.04	13.87	<0.001
At 15 minutes	Group-K	66.09	2.33	7.00	9.577	<0.001
	Group-B	63.87	2.89	8.56	11.81	<0.001
At 20 minutes	Group-K	67.51	2.52	5.58	7.631	<0.001
	Group-B	65.49	1.97	6.93	9.573	<0.001
At 25 minutes	Group-K	68.18	2.17	4.91	6.719	<0.001
	Group-B	66.56	1.56	5.87	8.101	<0.001
At 30 minutes	Group-K	69.64	2.15	3.44	4.713	<0.001
	Group-B	68.89	2.42	3.53	4.879	<0.001
At 60 minutes	Group-K	71.00	1.85	2.09	2.858	0.004
	Group-B	70.49	1.55	1.93	2.67	0.003
Recovery	Group-K	72.53	2.97	0.56	0.76	0.484
	Group-B	71.84	1.88	0.58	0.798	0.378

Table-5: Variation in heart rate in Group-K & Group-B after subarachnoid block

The maximum fall in SBP after subarachnoid block was 20.91 (15.85%) mm Hg which is to a level of (111.04 ± 2.84 mm Hg) and 34.62 (26.51%) mm Hg which is to a level of (96.96 ± 3.69 mm Hg) in Group K and B respectively from the baseline SBP. This signifies that Ketamine effectively obtunded the fall in SBP. The SBP were found to be more stable in Group K than Group B during intra-operative period.

Table-3 shows that the intraoperative diastolic blood pressure of Group B patients were at significantly lower level as compared to Group K at various intraoperative periods of the procedure except for baseline level. The maximum fall in DBP after subarachnoid block was found to be 14.69 (18.27%) mm Hg which is to a level of (65.71 ± 3.60) mm Hg and 20.36 (25.11%) mm Hg which is to a level of (60.71 ± 1.62 mm Hg) for Group K and B respectively from the baseline DBP. This shows that hemodynamic effects maintained better in Group K during subarachnoid block.

Table-4 shows that mean arterial pressure changes of Group B patients were at significantly at lower level as compared to Group K at various intraoperative periods of the procedure. The maximum MAP difference after subarachnoid block was 16.17 (16.57%) mm Hg which is to a level of (81.41 ± 3.07 mm Hg) in case of Group K and 25.11 (25.74%) mm Hg which is to a level of (72.46 ± 1.65 mm Hg) in case of Group B from the baseline value. This shows that hemodynamic effects was maintained better in Group K during subarachnoid block.

Table-5 shows intraoperative heart rate changes of patients at various intervals of the procedure. The maximum fall in HR after subarachnoid block in Group K was 7.93/min (10.85%) whereas in Group B it was 10.04/min (13.87%) from the baseline heart rate which was significant. Thus heart rate was found to be more stable in Group K compared to Group B which showed wide range of variation during the whole intra-operative period.

Nystagmus was observed in 21 (46.67%) patients and Dizziness was found in 10 (22.22%) patients of Group K only. Nausea and vomiting and PDPH was seen in both the groups and were statistically comparable. Psychomimetic effects was not seen in any of the groups.

DISCUSSION

Spinal anaesthesia blocks efferent sympathetic fibers and reduces systemic vascular resistance by decreasing the vascular tone causing peripheral venous pooling of blood which may reduce cardiac output.⁵ Such changes frequently result in systemic hypotension which is the most common complication of spinal anaesthesia with an incidence of 20% in the elderly age group.⁶ Prevention of hypotension during spinal anaesthesia is a contentious subject without a perfect method to prevent it. Mechanical method, volume loading and vasopressor have been tried from time to time with variable results.

Vasopressors including ephedrine, methoxamine and adrenaline are highly effective in preventing hypotension but may result in cardiac arrhythmias and myocardial ischaemia.⁷ Many studies have been conducted on

prevention and treatment of spinal anaesthesia induced hypotension⁸ but there is no established strategy to prevent or treat these sequences.

In 1962 Dr. Calvin Stevens at the Parke Davis Laboratories in Ann Arbor, Michigan, synthesized ketamine, a phencyclidine derivative which is an age old anaesthetic agent most commonly used via intravenous access.⁹

In this study 0.5mg/kg ketamine has been used, as at this dose haemodynamic depression was less which may be advantageous in patients with unstable haemodynamics.¹⁰ Moreover it does not causes respiratory depression at small doses (<1mg/kg).¹¹

Hence this study was undertaken to evaluate the efficacy of i.v ketamine in maintaining hemodynamic stability during spinal anaesthesia in 90 patients undergoing TURP for BPH at Assam Medical College & Hospital. The patients were divided is two groups viz Group K receiving inj. Ketamine 0.5 mg/kg iv and Group B not receiving inj ketamine.

In this study, 90 male patients were selected and divided randomly into two group, Group K and Group B, each group with 45 patients. Most of the patients (Group K 53.33% and Group B 51.11%) in both the groups were aged between (60-69 years). The mean age of patients in Group K was 60.80 ± 3.90 years and in Group B was 60.56 ± 4.98 years with a p value more than 0.05 and hence both groups were comparable.

The mean weight of patients in Group K was 65.93 ± 6.81 kg and in Group B was 65.07 ± 7.47 kg. with p value of more than 0.05 which is not significant, hence both the groups were comparable.

The mean height of patient in Group K was 152.29 ± 7.13 cms and in Group B was 152.24 ± 5.77 cms with p value of more than 0.05 which is not significant and hence both the groups were comparable.

Regarding ASA grading, Group K comprised of 28.89% and 71.11% ASA I and II respectively and Group B comprised of 35.56% and 64.44% ASA I and II respectively. The p value was more than 0.05 and hence both the groups were comparable with respect to ASA physical status.

Regarding the change in SBP, Ozakan F et al¹² (2011) in the patients scheduled for TURP under SA found fall in SBP which was upto level of 111.10 ± 1.532 mmHg in ketamine group and upto level of 99 ± 1.697 mm of Hg in Group B which is similar to our study where we observed SBP decreased to a level of 111.04 ± 2.84 mmHg in Group K and 96.96 ± 3.69 mmHg in Group B. Jain et al¹³ (2014) observed SBP fall from baseline to be 10.92 ± 0.73 mmHg in ketamine group and 16.44 ± 1.08 mmHg in ketamine free group which is almost similar to our study in which fall in SBP in Group K is 20.91 (15.85%) less from baseline and in Group B 34.62 (26.51%) less from baseline value, although the change in our study is more than the above mentioned study. The baseline systolic blood pressure in our study was 131.96 ± 3.99 mm hg and 130.58 ± 3.29 mm hg in Group K and B respectively.

Regarding DBP, Vaidya et al¹⁴ (2012) observed in their studies that the DBP in both the groups were below baseline value

till 100 minute and was found to be statistically significant from baseline value. This change in DBP correlated with our study. In our study DBP decreased to a level of 65.71 ± 3.60 mmHg in Group K which is 14.69 (18.27%) less from the baseline DBP and 60.71 ± 1.62 mmHg in Group B which is 20.36 (25.11%) less from the baseline DBP. The mean diastolic blood pressure at baseline level in our study was 80.40 ± 3.31 mmHg in Group K and 81.07 ± 3.48 mmHg in Group B.

Vaidya et al¹⁴ (2012) observed that MAP in both the groups decreased from the baseline and fluctuation in Group B was $\pm 10\%$ and in Group K was $\pm 8\%$ from the baseline value which is almost similar to our study but fluctuation in our group was 16.57% in Group K and 25.74% in Group B from the baseline. This difference between the percentage change in our study and the study mentioned above may be due to different route of drug administration or may be due to age difference in our patients.

Ozkan F et al¹² (2011) in TURP surgery using Ketamine in one group observed MAP lower in the placebo group at 3, 9, 15, 25 and 30 min time, as compared to ketamine group. This change in MAP is found to be similar in our study where there is lower value of MAP in Group B at 3, 6, 9, 12, 15, 20, 25 and 30 min in comparison to Group K.

After the block and throughout the intraoperative period the MAP decreased to a level of 81.41 ± 3.00 mmHg in Group K which was found to be 16.17 (16.51%) less from the baseline MAP and 72.46 ± 1.65 mmHg in Group B which is 25.11 less from the baseline. Mean arterial pressure at baseline measured 97.59 ± 2.83 mmHg in Group K and 97.57 ± 2.74 mmHg in Group B and the difference is statistically significant ($p < 0.05$).

Ozkan F et al¹² (2011) observed lower heart rate in placebo group at 2, 5, 6, 7, 8, 9 and 10 min as compared to ketamine group which was statistically significant. This is similar to our study in which decrease in heart rate in Group K was lower than in Group B at 3, 6, 9, 12, 15, 20 and 25 min and the change is statistically significant.

Jain A et al¹³ (2014) in their study observed decrease in heart rate from baseline was 9.0 ± 0.62 /min in ketamine group and 20.92 ± 1.04 /min in Group B which tallies with our study where we observed decrease in heart rate upto 10.85% (7.93/min) from the baseline in Group K and decrease in heart rate upto 13.87% (10.04/min) in Group B. Though fall in heart rate was observed in our study also, but the fall is comparatively less than in their study.

The baseline heart rate in Group K was 73.09 ± 4.39 /min and it was 72.42 ± 3.95 /min in Group B.

CONCLUSION

On the basis of the findings of our present clinical study, we can come to a conclusion that Inj. ketamine 0.5mg/kg i.v. administered after subarachnoid block was able to maintain haemodynamic stability without any major side effects of the drug, in otherwise healthy patients undergoing TURP for BPH.

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