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Original Research Article

Effect of Chronic β-Blocker Therapy on Hemodynamic Changes Associated With Spinal Subarachnoid Block (A Comparison between Normotensive V/S Controlled Hypertensive Patients β-Blocker Therapy)

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ABSTRACT

Background: Beta- receptor blocking drugs are now increasingly used in treatment of hypertension. This prospective, randomized study aimed to compare the magnitude and severity of haemodynamic changes after subarachnoid block (with hyperbaric bupivacaine) between normotensive and controlled hypertensive patients on beta blocker therapy.
Materials and Methods: In the study included total 80 patients underwent for infra umbilical surgery in subarachnoid block. The patients were divided in two groups (40 in each group): Patients controlled hypertensive on beta blocker and normotensive without any medication. After fluid preloading, spinal anaesthesia was performed with hyperbaric bupivacaine (0.5%) 3 ml (15 mg). Demographic characteristics and haemodynamic changes, requirement of vasopressor & other side effects were compared. Systolic (SBP), diastolic (DBP) and mean blood pressures (MBP) and heart rate (HR) were also compared before and after spinal anaesthesia.
Results: There was no significant difference between the groups with demographical and characteristics of spinal anaesthesia (onset, height of sensory analgesia, degree of motor blockade). In controlled hypertensive patients group incidence and magnitude of hypotension, bradycardia, requirement of vasopressor, major side effects nausea & vomiting were higher than as compared to hypertensive group ($p<0.05$).
Conclusions: Chronic beta blocker therapy for treatment of hypertension or IHD might be responsible for greater magnitude of fall in SBP, DBP, MAP associated spinal subarachnoid block and also required repeated and higher doses of vasopressors.

Key words: Spinal Anaesthesia, Hypertension, Hypotension, Hyperbaric bupivacaine.

INTRODUCTION

Beta-receptor blocking drugs are now increasingly used in treatment of hypertension, [1,2] cardiac dysfunction, [3,4] ischaemic heart disease presenting with angina pectoris, [5,6] hypertensive obstructive cardiomyopathy. [7]

However, main concern with spinal an aesthesia is its effect on cardiovascular system leading to hypotension, [8] other cardiovascular alterations includes decrease cardiac output, stroke volume, central venous pressure, heart rate and peripheral resistance. [9] The body Plays counter active physiological Mechanism to prevent these alterations of cardiovascular system, one of such mechanism is vasoconstriction of unblocked upper part of body and another one is application of Mary's law by which heart rate is increase to maintain cardiac

output.

Known patient of hypertension controlled with various classes of antihypertensive drugs may compromise the defence mechanism as mentioned above and thus may lead to exaggerated hypotension. Particularly, Beta blockers attenuate the response of vasoconstriction of unblocked part of body and affect the Mary's law. Also these drugs may decrease the effect of vasopressor.

There were few studies [10-12] shown that Spinal Anaesthesia produce unpredictable and more profound arterial hypotension in hypertensive patient's then normotensive counterpart.

There is no recent studies have defined the response of well controlled hypertensive patients on beta blocker drugs, to effects of Spinal anesthetic covering segment up to but no higher than T8 spinal segment.

In view of this we planned this present study to compare the magnitude and severity of haemodynamic changes after subarachnoid analgesia and also to compare the dose of requirement of vasopressor & other side effects between normotensive & controlled hypertensive patients on beta blocker therapy.

MATERIALS AND METHODS

This study was prospective randomized single blind study was conducted at associated groups hospital attached to Dr. S.N. Medical College Jodhpur, after taking approval by the ethical committee and obtaining a well-informed written consent from the patients, total 80 patients who were scheduled for lower abdominal surgery below umbilicus, ages of 40-60 years of either sex (ASA status grade 1&2) were included in study. Patients were divided in two groups. Normotensive patients who has no history of any medication Group A, Patients who has history of hypertension at least 6 month or more and controlled with beta blocker on regular treatment Group B.

Patients who had history of hypertension <6 months, on irregular treatment, uncontrolled hypertensive patients with blood pressure >140/90 mm Hg, history of IHD, LVH, LVF, Valvular heart disease and hypertensive neuropathy, contraindication of spinal anaesthesia were excluded from the study. Every patient was assessed properly in pre-anaesthetic clinic one day prior to surgery, patients was instructed to continue his/her beta blockers on the morning of surgery, at least 90 min before surgery. All patients were instructed to NBM 8 hours Premeditated with Tab Ranitidine 150 mg and Tab Alprazolam 0.5 mg orally the night before and also on the morning of surgery.

After arrival of patient in OT, an I.V. Line was secured with 18G or 20 G cannula and standard monitoring including NIBP cuff, ECG leads, and pulse Oximeter probe were monitored. Baseline SBP and heart rate was recorded by taking the mean of 3 consecutive reading taken 1 min apart. Preloading was done with Ringer lactate at a dose of10ml/kg/B.W. Within 30 minutes, The procedure of SAB was explained to the patients, after taking all aseptic precautions and proper draping, lumber interspace either L3-L4or L4-L5 was identified in sitting position & infiltrated with injection Xylocaine HCl 1%. Subarachnoid space was identified by using a 25 G whit-care spinal needle and once free flow of CSF appeared, 15 mg of 0.5% hyperbaric bupivacaine was injected at rate of 1ml/ sec. with direction of bevel of needle was cephalad, after completion of injection, the patients were immediately returned to supine position .

Characteristics of SAB (onset, time, height of sensory analgesia by pinprick method, degree of motor block by using modified bromage scale) were assessed every 30 sec till the desired level of sensory block was achieved. At the same time, regular monitoring &recording of vital parameters (haemodynamic changes SBP, DBP, MBP, and Heart Rate) were carried out at every 1 min interval for initial 20 min and every 5 min thereafter and also assessed

severity & magnitude of hypotension, total amount of fluid, total amount of vasopressor required, side effects like nausea, vomiting, dysthymia etc.

If hypotension occurred (SBP< 90 mmHg or decrease by >20% from the base line SBP), it was treated with rescue i.v. Ephedrine 6 mg bolus dose and in aliquots SOS.I.V. Atropine 0.6 mg given if bradycardia (HR <60 BPM) occurred after sub arachnoid block.

Statistical analysis done by computer software SPSS, Discrete variables are expressed as counts (%) and compared using the Chi-square tests. Continuous variables are expressed as mean±S.D. and compared by means of the unpaired, two-sided t test; Statistical significance was set at P <0.05.

RESULTS

There were 80 patients included into the study, 40 patients in each groups. There was no significant difference between the groups with respect to ASA grade, onset of sensory block, maximum height of sensory block, quality of motor block (bromage scale), duration of spinal an aesthesia, total amount fluid received, nausea and vomiting score.

Comparison of pre-intraoperative hemodynamic the p value for SBP, DBP, MBP were statistically insignificant (>0.05) while of pulse rate (rate/ min was statistically significant (<0.05). Total amount of vasopressor requirement (mg) p value was statistically significant (<0.05). In the controlled hypertensive patients higher incidences of hypotension and nausea & vomiting was as compared to normotensive patients. On statistical evaluation, the p value was found significant (<0.05) for side effects.

Incidences of hypotension were more in study controlled hypertensive group than normotensive group.

Table1: Characteristics of the groups

	Group B(n -40)	Group A (n 40)	P value
Age (yrs)	50.87±6.6456	45.20±6.025	<0.05
Height (cm)	155.45±2.3525	157.87±3.106	<0.0002
Weight (kg)	64.1750±7.39	55.95±7.24	0.0001
ASA grade	25/15	26/14	>0.9999
Hypotension n(%) Observation time(min)	 77.5% 6-14	 37.5% 6-14	
Bradycardia (beats/min)n(%)	5.47%	11.4%	0.0001
Sensory block Onset(min) Height(cm)	 7.0750±1.2276 8.275±1.48	 7.2250±1.3679 8.0±5.244	 0.6072 0.7504
Quality of motor block (bromage scale)	2.45±0.7143	2.60±0.6325	0.3231
Duration of spinal an aesthesia (hrs)	3.266±6.666	3.559±6.676	>0.9999
Total amount of fluid receive (L)	2.225±0.4522	2.1875±0.37	0.6859
Total amount of vasopressor requirement (mg)	6.45±4.7822	2.40±3.5431	<0.0001

DISCUSSION

The present study had demonstrated that the patient with chronic β blocker therapy for treatment of hypertension required significantly higher doses of vasopressor (Ephedrine) in comparison to normotensive patients after spinal an aesthesia. The statistically significant fall in SBP, DBP, and MAP was observed at 6-11 min &13 min, 6-14 min, 6-13 min respectively after spinal block. Heart rate tended to be on lower side with statistically significant difference at all times of observation after spinal block. Nausea & vomiting occurred more frequently in study group patients than the control group patients.

The earlier studies in 1950-1960 decade had shown that spinal an aesthesia produce unpredictable and more profound arterial hypotension in hypertensive patients than in their normotensive counterpart. It must be noted that the patients in those series were largely untreated hypertensive patients and that the segmental spread of the spinal an aesthesia was designed to produce "high spinal" conditions (Kety et al (1950) [10],

Pugh and Wyndham (1950) [13], Kleinerman Sancetta and Mackel (1958)[11]. After that there is scarcity in literature on haemodynamic changes associated with spinal an aesthesia in hypertensive patients on chronic treatment with β-blocker therapy.

Singla et al 2006 [14] identified the risk factors for development of early hypotension during spinal an aesthesia & they reported that hypertensive therapy was one of the risk factor for hypotension.

Most important perioperative complications impeding the effective & safe use of spinal anaesthesia are hypotension & bradycardia[15]. Reported incidences of hypotension ranged between 15-33%[16,17] the incidences of hypotension in our series were 77.5% in study group & 37.5% in control group. Incidence of hypotension in patients with chronic β-blocker therapy had not been reported with spinal anesthesia which can be compared with our study

In our study statistically significant fall in SBP, DBP, MAP of 19.37%, 16.45%, 19.79% was observed respectively more in treated hypertensive patients than in normotensive patients in whom fall in SBP, DBP, MAP was 11.81%, 13.41%,and 9.37%, thus our results were in accordance with the finding of Dagino et al[18].

Kennedy, Bonica[9], Ward[19] has found decrease in HR of 3-5%from pre-spinal values in normotensive patients while, Sancetta[20], stevens[21], had found increased in HR 10-20% from pre-spinal values. In our study decrease in HR was more in control group 11.4% than study group 5.47% with significant p value (<0.0001) in all patients.

In our study hypotension was defined whenever the SBP was at or <90 mmHg or decrease was >20% from baseline whenever the hypotension in our study, we gave dose of ephedrine (6 mg) and when required, While comparing both the group requirement of vasopressor was high (52.5%) in controlled hypertensive than normotensive group (32.5%. The mean dose of ephedrine given was higher in study group (6.45±4.78) than normotensive group (2.40±3.54) & statistically significant (p<0.0001).

Carpenter et al (1992)[17] found that nausea & vomiting occurred commonly during spinal an aesthesia (18% and 7% respectively) in normotensive group. In our study group too increased incidences of nausea & vomiting could also be associated with increased incidences of hypotension and it occurred more in controlled hypertensive group (72.5%) than in normotensive group (40%).

The causes & etiogenesis of hypotension with spinal an aesthesia are many and include: - decrease cardiac output, stroke volume. Theories proposed as to the etiology of alterations of cardiovascular function following spinal an aesthesia include:-sympathetic blockade, skeletal muscle atony, haematogenous intoxication, adrenal enervation, vital central paralysis, respiratory insufficiency and hypoxia.

Our Results Support these finding, the β blockade may impair Cardiac Performance and a myocardial Depression by Quinidine like effect, and that such Myocardial have complex pharmacological actions in addition to their competitive inhibitory action at adrenergic beta receptor.

CONCLUSION

In our study we found that chronic β blocker therapy for treatment of hypertension or IHD might be responsible for greater magnitude of fall in SBP, DBP, MAP associated spinal subarachnoid block also required repeated higher doses of vasopressors. Therefore, it is advisable to be extra cautious, whenever patients on β blocker therapy receives spinal subarachnoid block.

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