Original Research Paper

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ANTIHYPERTENSIVE THERAPY AND HEMODYNAMIC CHANGES FOLLOWING SPINAL ANAESTHESIA .

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ABSTRACT **Backgrounds and Aims** Hypertensive patients, undergoing surgery with spinal anaesthesia are at more risk of developing unpredictable intraoperative hypotension might be associated with myocardial ischemia and increase risk of stroke. This study designed to find out cardiovascular changes following spinal anaesthesia in normotensive and hypertensive patient, on treatment with different antihypertensive drugs. **Method** Randomised, control, prospective, observational study done during period of August 2020-October 2020, in Group-N-50(normotensive) and Group-H-50(hypertensive patients on antihypertensive treatment), posted for various surgery under spinal anaesthesia with 3ml- 3.5ml (15-17.5mg) 0.5%H bupivacaine. Pre loading done with 10-15ml/kg ringer lactate. Inj. Midazolam 0.03-0.05mg/kg body weight IV given after 10 mins of SA. Perioperative monitoring of PR, RR, SPO_2, SBP, DBP and MBP at every 5 minutes for first 30 minutes and then every 15 minutes till the end of surgery done with standard multipara monitor. Significant hypotension (MBP decreases ≥30% from baseline) treated with inj. Ephedrine 5mg iv and bradycardia (HR decreases below 50 bpm) treated with inj. Atropine 0.6mg iv. **Result** No statistical significance between the Groups in Weight, BMI, duration of surgery, AMI and preoperative Anxiety scores($p>0.05$). Pulse pressure and VOI was significantly high in Group-H as compared to Group-N ($P<0.0001$). Baseline SBP, DBP and MAP significantly high in Group-H as compared to Group-N ($p<0.00001$, 0.00004, <0.00001). Pulse rate changes noted more in Group-H as compared to Group-N at all time intervals. Hypotension noted more in Group-H-22/50(44%) as compared to Group- N-07/50(14%). Need of vasopressure doses more in Group- H-16/50(32%) as compared to Group-N- 06/50(12%). Bradycardia not noted in any of the groups. **Conclusion** We observed maximum hypotension in patients on ABRs+ CCBs, CCBs + ACE inhibitors and CCBs and also required more dose of ephedrine as compared to normotensive patients undergoing spinal anaesthesia.

KEYWORDS : Spinal anaesthesia, antihypertensive drugs, hypotension

INTRODUCTION

In today's modern life style, raised blood pressure [BP] - hypertension [HT] is commonly seen in day to day practice of anaesthesia and surgery. Morbidity and mortality associated with hypertension makes it a global public health concern as it predisposes to cardiovascular events, renal impairment, peripheral vascular disease, Hypertensive retinopathy, cognitive impairment and premature death. Primary hypertension [accounts for 95%] is defined as persistent (average of 2 or more readings) SBP more than 140mm Hg and or DBP more than 90mm Hg in adult in absence of any known precipitant cause. Secondary hypertension [accounts for 5%] is defines as elevation in BP with a known cause as for example pheochromocytoma, renal artery stenosis, etc.[1]

Intraoperative management of HT is important due to cardiac liability [auto regulatory curve may shift to right and brain and kidneys may be compromised by intraoperative hypotension]. Incidence of hypotension following spinal anaesthesia (25%-82%) is more in hypertensive patients (60%) than in normotensive patients (36%).[1]

METHOD AND MATERIALS

This cross-sectional observational study conducted in a tertiary care civil hospital, ahmedabad in April to September-2021. After approval from Institutional Ethics Committee study conducted in total 50 normotensive and 50 hypertensive patients. Inclusion criterias - Age 18-70 years, Patient posted for lower abdominal, lower limb, perineal surgeries, ASA grade I,II,III,IV, Gender- male and female, Patient with essential/primary hypertension and on anti-hypertensive medications. Exclusion criteria - Patient's refusal, Age < 18 years, Pregnant patients, Spine pathology or operated case of spine surgery, Uncooperative patients, patients with other co-existing diseases like Diabetes mellitus, cardiac disease. After all routine investigations, Preoperative pulse rate, SBP, DBP, MAP, pulse pressure, AMI (Ankle arm/systolic pressure index) and VOI (Vascular overload index) were noted in all patients.

After written and informed valid consent, 18G/20G intravenous cannula secured in upper limb and pre loading done with 10-15ml/kg intravenous fluid. Patient's baseline vitals pulse, blood pressure [SBP, DBP, MAP], Spo2, ECG, RR recorded using multipara monitors. 3ml-3.5ml (15-17.5mg) 0.5% hyperbaric bupivacaine was given via spinal needle over 10 seconds. Effect of spinal anaesthesia was confirmed by sensory blockage via pin prick sensations and motor block evaluate by Bromage scale after 5 minutes and 10 minutes after giving the spinal anaesthesia. Inj. Midazolam 0.03-0.05mg/kg body weight IV was given 10 mins after SA. Preoperative, intraoperative and postoperative monitoring of pulse rate, RR, spo2, SBP, DBP and MBP at every 5 minutes for first 30 minutes and then every 15 minutes till the end of surgery was done with standard multipara monitor. When MAP decreases ≥30% from baseline value, was accepted as Hypotension and treated with Inj. Ephedrine 5-10mg IV. If HR decreased below 50 bpm, was accepted as Bradycardia and treated with Inj. Atropine 0.6 mg IV and treatment of underlying pathology.

Data analysis of patient characteristics and hemodynamic data (PR, SBP, DBP, and MAP) done by using incidence frequency (%) and mean ± standard deviation. Variables were compared by paired T test within the group. Intergroup comparison of variables between hypertensive and normotensive groups at different time interval were done by unpaired T test. $P<0.05$ considered statistically significant.

RESULT

We had mostly elder age group patients in Group-H as compared to Group-N ($P<0.00001$), but no significance in gender, weight, BMI and mean duration of surgery between the groups. [Table 1] Total dose of Inj.bupivacaine (ml) used in Group-N comparatively higher than Group-H (P-0.009). Pulse pressure was significantly high in Group-H-60.16±13.76 as compared to Group-N- 48±9.63 (Mean±SD) ($P<0.0001$). AMI is predictor of lower limb occlusive disease severity and related to magnitude of systolic tension decrease during spinal anaesthesia. AMI (Ankle/ arm systolic pressure index) value in Group-N-1.18±0.1 and Group-H-1.13±0.04, statistically insignificant (P=0.013). VOI (Vascular Overload Index) is a cardiovascular risk predictor in hypertensive patients and is correlated to the magnitude of systolic tension decrease during spinal anaesthesia. VOI was significantly higher in Group-H-33.38±18.40 as compared to Group-N-10.65±15.37 ($P<0.0001$).

Type of antihypertensive drugs and duration of antihypertensive therapy which patients on are showed in table no 2 & 3.

Hemodynamic Changes-

Baseline SBP, DBP and MAP were statistically significant between the

groups (P<0.00004) but pulse rate were insignificant. Intraoperatively, pulse rate was insignificant between the groups. SBP was significant between the groups at baseline (P<0.00001), 5min (P-0.016), 45min (P-0.011), 60min (P-0.017), 75min (P-0.004), 90min (P-0.031), 120min (P-0.024) and 150min (P-0.003). DBP was significant between the groups only at baseline (P-0.00004). MAP values were significantly higher in Group- H than Group- N at all measurement times but statistical significance noted only at baseline.

Significant hypotension noted high in Group-H- 22/50 (44%) patients as compared to Group- N- 7/50 (14%) patients.[Table 4] In hypertensive group, hypotension was comparatively high in patients on CCBs or on combination drug therapy with CCBs. Bradycardia was not noted in any groups.

DISCUSSION

Spinal anaesthesia is being widely used in orthopaedic, below umbilical abdominal surgeries and urology surgeries nowadays. It is important to know about hemodynamic changes and its mechanism to decrease incidence of hypotension after spinal anaesthesia. Hypertension is most common condition seen in preoperative assessment and it remains one of the most important preventable contributors to disease and death. Risk of significant hypotension increases two fold in hypertensive patients following spinal anaesthesia. [2]

Baseline hemodynamics-

In our study baseline pulse rate (bpm), Group-N-85±10.31 and Group-H-88.76±10.05, statistically insignificant (P-0.06) similar to findings of Poh KS et all [3] observed baseline pulse rate normotensive group-79.2±8.3 and hypertensive group -77.7±11.2, statistically insignificant (P-0.34) and Kavyashree N et all [4] observed baseline pulse rate Group-N (Normotensive)-75.0±5.47, Group-C (CCBs)-72.46±5.81 and Group-B (β- blocker)-67.53±8.7, statistically insignificant (P>0.05).

In our study baseline SBP (mmHg) in Group-N-128.58±13.11 (Mean±SD) and in Group-H-148±16.15, statistically significant (P<0.00001) which was comparable to study reported by Poh KS et all [3] who observed baseline SBP was in normotensive group-122.1±11.7 and hypertensive group -137.7±15.6, statistically significant (P<0.01). In our study baseline DBP (mmHg) in Group-N-80.58±8.22 and in Group-H-87.84±8.82, statistically significant (P-0.00004) which was similar with previous study results of Poh KS et all [3], observed baseline DBP in normotensive group -73.9±6.7 and hypertensive group-81.1±10.3, statistically significant (P<0.01).

Intraoperative changes:-

Mechanmis behind pulse rate changes after spinal anaesthesia can be explained by trigger of compensatory baroreceptor sympathetic response following spinal induced hypotension and causes increase in pulse rate. But after spinal anaesthesia extensive peripheral sympathectomy causes venous pulling in lower extremities, decrease venous return and decrease filling of right atria which decreases response of chronotropic stretch receptors located in right atria and great veins and leading to increase parasympathetic activity. These two opposing responses nullify each other's effects; result in minimal changes in pulse rate after spinal anaesthesia.

In our study, within the group statistically significant fall in **pulse rate** (bpm) noted in Group-N at 5min, 60min and 75mins were (Mean±SD) 85±13.08, 81.77±11.68and 81.58±11.79 respectively (P=0.047, 0.0036, 0.017) and in Group-H at 15min-86.1±12.83, 20min-85.86±12.85, 25min-84.56±13.27, 30min-84.38±13.26, 45min-84.82±12.21, 60min-85.38±11.68 and 75min-84.52±12.56 respectively (P=0.02, 0.03, 0.0047, 0.0037, 0.011, 0.044, 0.02). There was no statistical significant at any time interval between the groups. Kavyashree N et all[4] noted significant fall in pulse rate in Group-B (β-blocker) compare to Group-C (CCBs) and Group-N at 2, 4, 6, 8, 10, 15 minutes (mean difference±SD) were 5.2 ±2.4, 6.5±1.8, 7.7±1.7, 10.2±0.58, 10.4±0.81, 10.5±0.8 (bpm) from baseline (P <0.001, <0.003, and rest <0.000) respectively. Same as our study, Kavyashree N et all[4] also noted significant fall in pulse rate within the group but no statistical significant between the groups.

SBP-

In our study statistically significance in SBP at baseline (P<0.00001), 5min (P-0.016), 45min (P-0.011), 60min (P-0.017), 75min (P-0.004), 90min (P-0.031), 120min (P-0.024) and 150min (P-0.003) between both the groups. Our study results are similar to results of previous studies of Kavyashree N et all[4] noted SBP changes statistically significant between group-N and Group-C at 4, 6 and 10mins, Group-C and Group-B at 4, 6, 8 and 10mins. Meena S et all[5] noted significant fall in SBP- 6-11 min and 13 min in Group-B (β- blocker) and Group-N (Normotensive).

DBP-

we noted statistically significance in DBP only at baseline between both the groups (P-0.00004). Our study results are similar to data of previous studies of Kavyashree N et all[4] noted Group-N and Group-C difference statistically significant at all time except Base (P< 0.02-0.05), Group-N and Group-B was not significant at any time while Group-C and Group-B has highly statistically significant values (P<0.02 to P<0.001) Group-N- (P<0.001). Meena S et all[5] noted Significant fall in DBP- 6-11 min and 13 min in Group-B (β- blocker) and Group-N (Normotensive). Fall in DBP was noted 16.45% in Group B and 13.41% in Group N.

MAP-

In our study, MAP values were significantly higher in Group- H than Group- N at all measurement times but statistical significance noted only at baseline. But Nermin SA et all[6] noted statistical significance at all time interval between the groups.

Our study results shows that following SA blood pressure decrease in both groups, but more fall in blood pressure noted in hypertensive group patients. After SA anaesthesia sympathetic blockade leading to peripheral vasodilatation and venous pulling of blood causes hypotension.[7] Vasoconstriction of vessels supplied by unaffected sympathetic nerves counteracts vasodilatation of vessels supplied by block sympathetic nerves after mean decrease in MAP by 15% patients did not develop reflex tachycardia or increase CO. This may reflect decrease venous return as a result if vasodilatation.

Hypotension-

After spinal anaesthesia peripheral vasodilatation occurs due to blockage of vasoconstrictor nerve fibers. Decrease vasomotor tone at preganglionic level and affect both arterioles and veins this explains mechanism behind spinal induced hypotension.[8] In our study, significant hypotension after spinal anaesthesia occurred in both groups from 5min to 120 min, but more significant fall in SBP from baseline occurred in Group-H patients. In hypertensive patients, increased sympathetic activity and norepinephrine levels cause loss of elasticity of arterial walls and structural changes in vessel wall leading to increased peripheral resistance.[6] Venous capacitance decreased in hypertensive patients. Structural changes in arteriolar walls (medial hyperplasia and hypertrophy) plays important role in hemodynamic response following spinal anaesthesia. [3,4,6,9] So, after spinal anaesthesia, sudden sympathectomy causes decrease peripheral resistance and redistribution of blood and leads to greater fall in blood pressure in hypertensive patients as compared to normotensive patients. Hypertensive patients on calcium channel blockers developed more hypotension due to already existing vasodilatation effect of CCBs. This explains statistical significance between both the groups at 5min, 45min to 90 min, 120min and 150min.

Kavyashree N et all[4] observed hypotension more in Group-C (CCBs)-n=18/30[60%] as compared to Group-N (Normotensive)-n=12/30[40%] and Group-B (β- blocker)-10/30[33.3%]. **Meena S et all[5]** observed overall higher Incidence of hypotension in Group-B (β-blocker) (77.5%) than Group-A (Normotensive) (37.5%) In our study, we noted that in Group-H, patients on CCBs and with combination drug therapy with CCBs needed more dose of ephedrine as compared to patients on other antihypertensive drugs and normotensive patients. Our study results are similar to previous study data of Kavyashree N et all[4] and Kaimar P et all[10].

In previous study, **Nermin SA et all[6]** observed that total iv fluid given was approximately 500-550ml and **Meena S et all[5]** noted that total iv fluids given was 2000-2300ml. In our study, total fluid given was Group-N -1595±519.44 (Mean±SD) and Group-H -1742.5±656.35 (Mean±SD), statistically insignificant (P-0.21).

Neuraxial blocks are known to produce a vagotonic baroreceptor response, which can be restored by the cardiac filling pressures.[8] In our study, **bradycardia** was not noted in any groups. According toprevious study results of Kavyashree N et all[4] and Kaimar P et all[10],

bradycardia noted in patients on β- blocker drugs.

Our study results supports finding of previous studies that patients on treatment with antihypertensive drugs have higher incidence of hypotension following SA as compared to normotensive patients. We noted episodes of hypotension mostly within 20-25min after administration of SA.

CONCLUSION

From our study results, we concluded that elder age, recently diagnosed hypertension, treatment with CCBs and orthopaedic surgery are associated with higher incidence of hypotension following SA, which require vigilant monitoring and preventive measures for timely therapeutic intervention and to prevent SA induced hypotension.

Tables 1:- Demographic Data

Variable		Normotensive	Hypertensive	P value
Age (Mean ± SD)		40±16 year	60±11 year	>0.00001(S)
Sex	Male	48(96%)	36(72%)	
	Female	2(4%)	14(38%)	
Height (Mean ± SD)		163.2±6.57	166.32±8.13	0.037(S)
Weight (Mean ± SD)		62.89±8.04	64.48±11.86	0.43(NS)
BMI (Mean ± SD)		22.46±3.34	23.27±3.85	0.26 (NS)
Duration of Surgery (Mean±SD)		96.4±34.61	97.6±28.14	0.84 (NS)
Preoperative Anxiety score		11.62±1.99	11.96±1.39	0.32 (NS)

Table 2:- Duration of Hypertension

DURATION OF HYPERTENSION	NO. OF PATIENTS
<7 Days	15/50 (30%)
7 Days- 1 Month	4/50 (8%)
1 Month- 1 Year	9/50 (18%)
>1year- 5 Years	7/50 (14%)
>5years- 10years	13/50 (26%)
>10years	2/50 (4%)

Table 3:- Anti Hypertensive Drugs and Hypotension

Sr. No	Type of anti hypertensive drug	No of patient	Hypotension	Ephedrine			Not required Ephedrine dose
				1 Dose	2 Dose	>2 Dose	
1	T.Amlodipine (Calcium Channel Blocker)	27/50 (54%)	10/27 (37%)	6/10 (60%)	1/10 (10%)		3/10 (30%)
2	T.Telmisartan (ARBs)	2/50 (4%)	2/2 (100%)		1/1 (50%)		1/1 (50%)
3	T.Enalapril (ACE Inhibitors)	1/50 (2%)	0 (0%)				
4	T.Amlodioine+ T.Enalapril (Calcium Channel Blocker+ ACE Inhibitors)	6/50 (12%)	3/6 (50%)	2/3 (66.6%)			1/3 (33.3%)
5	T.Amlodipine+ T.Atenolol/Metoprolol (Ca. Channel Blocker+ β-Blockers)	5/50 (10%)	1/5 (20%)	1/1(100 %)			
6	T.Amlodioine + T. Losartan/Telmisartan (Ca. Channel Blocker+ ARBs)	6/50 (12%)	5/6 (83.3%)	1/6 (16.6%)	1/ 6 (16.6%)	2/6 (33.3 %)	1/6 (16.6%)
7	T.Losartan + T.Chlorthiazide (ARBs+ Thiazides)	1/50 (2%)	0 (0%)				
8	T.Amlodipine+ T.Chlorthaizide/ Hydrochlorthiazide (Ca. Channel Blocker+ Thiazides)	2/50 (4%)	1/2 (50%)	1/2 (50%)			
	Total	50	22(44%)	11/22 (50%)	3/22 (13.6%)	2/22 (9%)	6/22 (27.2%)

Table 4 :- Incidence of Hypotension and requirement of ephedrine

Variable	Normotensive		Hypertensive	
Hypotension developed (no. of patients)	7/50 (14%)		22/50 (44%)	
Hypotension required Vasopressure (no of dose)	6/50 (12%)	05/50-1dose (10%)	16/22 (72.72%)	11/50- dose (22%)
		01/50-2dose (2%)		3/50- 2dose (6%)
		0/7- 3 dose (0%)		2/50- 3dose (4%)
Hypotension not required vasopressure	01/50 (2%)		6/50 (12%)	
Bradycardia	0/50		0/50	
Atropine required	0/50		0/50	

REFERENCES:-

1. Misra S. Systemic Hypertension and Non cardiac Surgery. Indian Journal of Anaesthesia; 2017; 61: 697-704.
2. Hartmann B, Junger A, Kalsen J, Benson M, Josh A, Banzhaf A, Et al. The incidence and risk factors for hypotension for spinal anaesthesia after spinal anesthesia induction: An analysis with automated data collection. AnaesthAnalg. 2002; 94:1521-9.
3. Poh KS, Lim TA, Airini I. Perioperative blood pressure changes in Normotensive and Hypertensive Patients. Med J Malaysia; 2017; vol.62:97-103.
4. Kavyashree N, Patel M, Girish C. Ravishankar R. Comparison Of Hemodynamic Profile after spinal Anaesthesia in patietns on regular treatment with calcium channel blocker and β-blockers. J. Evolution Med. Dent. Sci; 2016; 5(34): 1923-1928.
5. Meena S, Meena J, Joshi V. 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). International Journal of Research and Review; 2016; 3(10): 57-61.
6. Nermin A, Sinan U, Mehmet T, Ibrahim A, Ayse V.Spinal anesthesia with hyperbaric bupivacaine: A comparison of hypertensive and normotensive patients e-ISSN 1643-3750 © Med SciMonit, 2013; 19: 1109-1113 DOI: 10.12659/MSM.889412.
7. Park S. Prediction of hypotension in spinal anaesthesia. Korean J Anesthesiol; 2013; 65(4):291-292.
8. Casati A, Fanelli G, Aldegheri G, Colaghi E, Casaletti E, Cedrati V, et al. Frequency of hypotension during Conventional or Asymmetric hyperbaric spinal Block. Regional Anaesthesia and Pain Management; 1999; 23(3): 214-19.
9. Dobson P, Caldicott L, Gerrish S, Cole J, Channer K. Changes in Haemodynemic variables during Transurethral resection of the Prostate: Comparison of General and Spinal Anaesthesia. British Journal of Anaesthesia; 1994; 72: 267-271.
10. Kaimar P, Sanji N, Upadya M, Mohammed K. A Comparison of hypotension and bradycardia following spinal anaesthesia in patietns on calcium channel blockers and B-blockers. Indian J Pharmacol; 2012; 44(2): 193-196.