Effect of Ondansetron in Prevention of Hypotension in Elective Lower segment Cesarean Section under Spinal Anesthesia: A Randomized, Double-Blind Study

Sharad Narayan Sharma¹, Gopal Singh Maravi²

¹Senior Resident, Department of Anesthesiology, Netaji Subhash Chandra Bose Medical College, Jabalpur, Madhya Pradesh, India, ²Associate Professor, Department of Anaesthesiology, Netaji Subhash Chandra Bose Medical College, Jabalpur, Madhya Pradesh, India

Abstract

Background: Spinal anesthesia is the preferred modality of anesthesia for lower segment cesarean section, but it is complicated with hypotension and bradycardia, which may be harmful to both parturient and baby. Bezold–Jarisch reflex plays an important role through 5HT3 receptors located in intracardiac vagal nerve endings in causing hypotension and bradycardia. In this study, we evaluated the effect of ondansetron, as a 5HT3 receptor antagonist, on the hemodynamic response following spinal anesthesia in parturients undergoing elective lower segment cesarean section.

Methodology: Sixty parturients who were scheduled for lower segment elective cesarean section were randomly allocated into two groups. Before giving the spinal injection, Group O (n = 30) received intravenous ondansetron 4 mg and Group S (n = 30) received normal saline. Blood pressure, heart rate, and vasopressor requirements were assessed.

Results: Total dose of vasopressor (mephentermine) used in Group "O" was 78 mg (mean±SD = 2.60 ± 4.36) and in Group "S," it was 168 mg (mean ± SD = 5.6 ± 4.43 (P = 0.010). In Group O, the incidence of hypotension was 9 out of 30 patients while in Group S, 21 out of 30 patients developed hypotension at any point of surgery (χ²=9.6 and P = 0.002).

Conclusion: Ondansetron 4 mg, given intravenously 5 min before spinal anesthesia, causes reduction in hypotension and vasopressor use in parturients undergoing elective lower segment cesarean section.

Key words: Cesarean, Hypotension, Ondansetron, Spinal

INTRODUCTION

Spinal anesthesia is a simple technique, wherein a small quantity of local anesthetic is administered into the spinal canal and a part of the body is anesthetized. This technique has been refined overtime and expanded in its practical applications. Spinal anesthesia has progressed greatly since 1885 and is used successfully in a number of different clinical situations.

In cafeteria choice of anesthetic modalities, spinal anesthesia has definitive advantage than its counterparts because a rapid profound analgesia can be produced in large part of the body by relatively simple injection of small amount of local anesthetic agent. Due to its simpler technique and less time-consuming procedure and to avoid the undesired consequences of general anesthesia, it has been adopted universally as the most preferred anesthetic technique for obstetric anesthesia. In concern to complications of spinal anesthesia, hypotension is major one of them, frequency of which may be as high as 60%–100% in emergency lower segment cesarean section (LSCS)¹¹ which is associated with sympathectomy which causes decreased cardiac output and somewhere poses risk to mother and fetus due to compromised uteroplacental flow.¹²⁻¹³ Recent studies show that prophylactic ondansetron in spinal anesthesia decreases the event of hypotension.
Ondansetron is 5HT3 antagonist mostly used as antiemetic. Apart from hypotension, nausea and vomiting related events have incidence of around 50%–80% in patients undergoing LSCS without any prophylactic intervention.\(^{[4]}\)

Parasympathetic shift of autonomic nervous system after spinal anesthesia causes bradycardia from the left ventricular mechanoreceptors due to sudden decrease in the left ventricular volume, i.e., Bezold–Jarisch reflex. Pharmacological studies reveal that serotonin may be an important factor associated with inducing B-J reflex and can be blocked through ondansetron.\(^{[5,6]}\)

Based on these findings, this randomized double-blind study was performed to evaluate the effect of ondansetron in prevention of hypotension in elective LSCS under spinal anesthesia.

**METHODOLOGY**

After getting approval from the Internal Ethics Committee, the present study was carried out in the Department of Anesthesiology and Critical Care, Netaji Subhash Chandra Bose Medical College and Hospital, Jabalpur, Madhya Pradesh, from a time period of March 2017–August 2018.

**Inclusion Criteria**

All patients undergoing elective lower segment cesarean section were included in the study.

- Weighing 50–90 kg
- Height 140–180 cm
- Hemodynamically stable.

**Exclusion Criteria**

The following criteria were excluded from the study:

- Patients with pre-existing cardiac disease
- Patients with liver and renal dysfunctions
- History of hypertension, pre-eclampsia, and convulsions
- Patient with bleeding disorders.

**Design of Study**

This was a randomized, double-blind, prospective study.

**Mode of Selection of Cases**

Simple randomization technique was used to divide the study subjects into two groups using table of random number. Subjects who satisfy the criteria would be given consecutive numbers and treatment allocation was be done as per the list prepared prior.

**Allocation to Different Groups**

Sixty patients will be equally divided into two groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Drugs used</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group O</td>
<td>4 mg of ondansetron</td>
<td>30</td>
</tr>
<tr>
<td>Group N</td>
<td>10 ml of normal saline</td>
<td>30</td>
</tr>
</tbody>
</table>

**Study Protocol**

- In Group O, patient received 4 mg of ondansetron diluted with normal saline up to 10 ml in 1 min, 5 min before spinal
- In Group S, patient was given 10 ml of NS over 1 min, 5 min before spinal anesthesia
- Under all aseptic precautions, spinal anesthesia was given using 23G Quincke needle and 2.2 ml of bupivacaine 0.5% heavy injected in subarachnoid space.

**METHODS**

After careful pre-anesthetic examination, the patients were included in the study and randomly allocated into the groups by lottery method. Before shifting the patient to the operating table, the table was made horizontal to ground using a fluid-filled leveling device.

After installing all routine monitoring devices such as electrocardiographic leads, non-invasive blood pressure (BP) cuff, pulse oximetry probe, and an intravenous (iv) access were secured using an 18G cannula and the patients were preloaded with 10 ml/kg of Ringer's lactate.

- Group O patient received 4 mg of ondansetron diluted up to 10 ml with NS and injected over 1 min, 5 min before spinal anesthesia
- Group S patient received 10 ml NS over 1 min 5 min before spinal anesthesia
- Patients were positioned on the operating table in the left lateral decubitus position with both lower limbs kept folded to abdomen with back curved and flexed. With all aseptic precautions, the study subjects were painted and draped using sterile solutions who then received a standard lumbar puncture with using 23G Quincke needle at L3-L4 intervertebral space and spinal drug given
- Immediately after injection, the patients were placed in supine position.

**Parameters of Comparison**

Patients were evaluated for the following parameters from the time of induction to 4 h.

1. The time to onset of sensory block and duration of sensory block from intrathecal administration of drug to regression of S2 segment
2. The time to onset of motor blockade (MODIFIED BROMAGE SCALE) and duration of motor block
Sharma and Maravi: Effect of Ondansetron in Prevention of Hypotension in Elective Lower segment Cesarean Section under Spinal Anesthesia: A Randomized, Double-Blind Study

from the onset of motor block to achieve Bromage scale 0.

Assessment Scales
- The sensory block was evaluated by HOLLMEN SCALE SCORE which reached up to score IV bilaterally up to T6 level
- Time of intrathecal injection was considered as 0 min. Intraoperative variables mean arterial pressure (MAP), systolic BP (SBP), diastolic blood pressure (DBP), and heart rate (HR) were monitored every 3rd min up to the delivery of fetus and thereafter every 5 min up to the completion of surgery
- Timing and cumulative doses of atropine and mephentermine were recorded
- The motor block was evaluated by MODIFIED BROMAGE SCALE every 5 min till the score reached 3 and postoperatively every 30 min up to a score of 0
- Result was be noted and analyzed.

Adverse Outcomes and Complications
Although rare, the study had some complications related to the procedure done or related to the drug use. These were following
- Hypotension treated when MAP decreased <20% of baseline, SBP < 90 mmHg, and DBP < 45 mmHg with mephentermine 6 mg iv
- Bradycardia is considered when HR < 60/min, treated by atropine 0.6 mg iv
- Result was noted and analyzed.

Hollmen Scale Score for Sensory Block

<table>
<thead>
<tr>
<th>Score</th>
<th>Observation</th>
</tr>
</thead>
<tbody>
<tr>
<td>i.</td>
<td>Normal sensation of pin prick</td>
</tr>
<tr>
<td>ii.</td>
<td>Weaker sensation of pin prick</td>
</tr>
<tr>
<td>iii.</td>
<td>Pin prick recognized as touch with a blunt object</td>
</tr>
<tr>
<td>iv.</td>
<td>No perception of pin prick</td>
</tr>
</tbody>
</table>

Modified Bromage Scale

<table>
<thead>
<tr>
<th>Score</th>
<th>Observation</th>
<th>Degree of block</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No motor block</td>
<td>Nil 0%</td>
</tr>
<tr>
<td>1</td>
<td>Can flex knee, move foot but cannot raise leg</td>
<td>Partial 33%</td>
</tr>
<tr>
<td>2</td>
<td>Can move foot only</td>
<td>Almost complete 66%</td>
</tr>
<tr>
<td>3</td>
<td>Unable to move foot or leg</td>
<td>Complete 100%</td>
</tr>
</tbody>
</table>

OBSERVATION AND RESULTS

No vasopressor was used in 21 patients in Group O and in 9 patients in Group N. In Group O, 6 mg of vasopressor used once in five patients while it was used in 14 patients in Group S. In Group O, 6 mg vasopressor used twice in four patients while in seven patients of Group S.

Total dose of vasopressor (mephentermine) used in Group “O” was 78 mg (mean ± SD = 2.60 ± 4.36) and in Group “S,” it was 168 mg (mean ± SD= 5.6 ± 4.43 (P = 0.010).

In this study, no significant episode of bradycardia was observed in either group. After spinal anesthesia, hypotensive episodes were observed in the form of fall in SBP, DBP, and MAP. Six minutes after spinal anesthesia, significant difference was observed in SBP between two groups with P = 0.0096, whereas at 0 min after giving spinal anesthesia, significant difference was observed in DBP in between two groups with P = 0.0034 [Tables 1-7 and Graphs 1-5].

MAP in both groups when compared had significant difference at 0, 3rd, and 6th min after giving spinal anesthesia with P = 0.0034, 0.026, and 0.0377, respectively.

In Group O, the incidence of hypotension was 9 out of 30 patients while in Group S, 21 out of 30 patients developed hypotension at any point of surgery (χ²=9.6 and P = 0.002).

No significant difference was seen in time to delivery of fetus and total duration of surgery in between two groups.

DISCUSSION

Attempts to find measures for the prevention of hypotension were vividly called by Alison Macarthur “the quest for the holy grail,” in obstetric anesthesia.[7] Effect of iv crystalloids, colloids, vasoconstrictors, and various physical methods as limb elevation, bandaging, etc., is studied in the past in various studies.[8]

Unopposed parasympathetic dominance after spinal anesthesia leads to fall in systemic vascular resistance and causes peripheral diversion of circulation. This causes
hypotension. Low volume received in the left ventricle stimulates mechanoreceptors in heart wall triggers the Bezold–Jarisch reflex and this results in reflex bradycardia, vasodilation, and hypotension.\(^{[9-11]}\)

Chemoreceptor activation also occurs due to low blood volume mediated through serotonin release from activated thrombocytes.\(^{[12,13]}\) Serotonin receptors are G protein-coupled receptors and only 5HT3 is a ligand-gated ion channel. Activation of this causes increased vagal tone and causes bradycardia and hypotension.\(^{[14]}\)

In the present study, demographic and preoperative variables were comparable in both groups and had no significant difference [Tables 8 and 9 and Graph 6].

Likewise to our study, other different studies who assessed the effect of ondansetron in attenuation of post-spinal anesthesia hypotension similar doses of hyperbaric bupivacaine for intrathecal injection were used. Sahoo et al.\(^{[15]}\) and Palmese et al.\(^{[16]}\) used 10 mg of 0.5% bupivacaine heavy for spinal anesthesia, whereas Marashi et al.\(^{[17]}\) used 15 mg dose in non-obstetric patients. Trabelsi et al.\(^{[18]}\) used 10 mg bupivacaine along with 2.5 mcg sufentanyl and Potdar et al.\(^{[19]}\) used 12 mg hyperbaric bupivacaine with 60 mcg buprenorphine. Intrathecal opioids were used to enhance the quality of block, but we did not use any adjuvant to hyperbaric bupivacaine.

Throughout intraoperative period, non-invasive monitoring was done. For the purpose of analysis, pulse rate (PR), SBP, DBP, MAP, and SpO\(_2\) were recorded every 3rd min up to delivery of fetus and thereafter at every 5th min up to the completion of surgery [Tables 1 and 2 and Graphs 1 and 2].

In our study, the overall incidence of hypotension in Group O was 30% in comparison to 70% in Group N and there was a significant difference between two groups \((P = 0.002)\). No incidence of bradycardia was reported in either group.

Similar to our study, Sahoo et al.\(^{[15]}\) reported less frequent incidence of bradycardia than hypotension, i.e., bradycardia in 2.1–4.9% of patients and hypotension in 36.8–52% of patients.

In concordance to the present study, Owczuk et al.\(^{[20]}\) showed that iv ondansetron attenuates the arterial BP drop due to spinal anesthesia using 8 mg ondansetron in patients of 20–70 years of age group. Drop in SBP below 90 mmHg in sequential 5 min observations in ondansetron group was 2.8% against the normal saline group, in which SBP <90 mmHg was observed in 20% of patients, which was statistically highly significant.

In our study, hypotension was defined as fall in SBP, DBP, and MAP >20% from its baseline value. Meanwhile, Owczuk et al.\(^{[20]}\) did not define the hypotension criteria in their study, whereas Sahoo et al.\(^{[15]}\) defined hypotension as SBP <90 mmHg and DBP <60 mmHg.

Similar to our study, parameters defined in the study of Abbas et al.\(^{[21]}\) fall in SBP >20% from baseline value was considered as hypotension. Arivumani et al.\(^{[22]}\) and Mohamed et al.\(^{[23]}\) defined hypotension as fall in MAP >20% of its baseline value.

Wang et al.\(^{[24]}\) used four different doses 2 mg, 4 mg, 6 mg, and 8 mg of ondansetron and concluded that 4 mg of prophylactic i/v ondansetron is an optimal dose to prevent post-spinal hypotension and bradycardia.

Sahoo et al.\(^{[15]}\) showed the effect of ondansetron in prevention of hypotension on 40 patients of obstetrical entity using 4 mg iv ondansetron before spinal anesthesia. In their study, Group N (normal saline) had significantly lower MAP between 14\(^{th}\) and 35\(^{th}\) min. Significant differences in MAP in both groups were observed at 5\(^{th}\) min (Group O 88 ± 11.7 vs. Group S 82.2 ± 10.5 mmHg) and at 6\(^{th}\) min (Group O 87.5 ± 11.3 vs. Group S 80.4 ± 10.8 mmHg).

Similarly, in our study, significant difference was observed of MAP in both groups in initial 6 min after spinal anesthesia. At 0 min, MAP in Group O was 76.16 ± 5.65 mmHg and in Group N was 72.03 ± 4.75 mmHg \((P = 0.0033)\). At 3\(^{rd}\) min, MAP in Group O was 74.46 ± 5.51 mmHg and in Group N was 71.53 ± 4.34 mmHg \((P = 0.02)\). At 6\(^{th}\) min, MAP in Group O was 73.43 ± 4.80 mmHg and in Group N was 70.86 ± 4.53 mmHg \((P = 0.037)\) [Table 6 and Graph 7].

Our study was in concordance to the study by Marashi et al.\(^{[17]}\) who used iv 6 mg ondansetron and 12 mg ondansetron against the placebo (normal saline) before spinal anesthesia for the prevention of hypotension where both the doses found to be equipotent for the prevention of hypotension after spinal anesthesia. There was no statistically significant difference observed in MAP and HR in both groups using iv ondansetron 6 mg and 12 mg \((P = 0.06)\). In the group using normal saline as placebo, 12 patients (17.14%) had MAP <80 mm in comparison to zero number of patients in the ondansetron groups. There was a significant difference with \(P = 0.04\). None experienced significant hypotension or bradycardia that required treatment.

Likewise, the study of Hasanein an El-Sayed\(^{[25]}\) was in concordance to our study who demonstrated that hypertensive
bradycardia events reduced in patients from 20.4% using normal saline to 6.1% using 4 mg ondansetron and 6% in group using 8 mg ondansetron. No difference was observed in groups using 4 mg and 8 mg ondansetron. They concluded that 4 mg ondansetron is optimal dose for the prevention of hypotension after spinal anesthesia. Therefore, in our study, iv 4 mg ondansetron before spinal anesthesia was used.

In our study, significant difference in SBP between two groups was observed at 6th min, SBP in Group O mean ± SD was 103.80 ± 8.07 mmHg and in Group N 98.10 ± 8.40 mmHg, P = 0.009 [Table 3].

Whereas statistically significant difference in DBP between two groups was observed at 0 minute, DBP in Group O mean ± SD was 61.83 ± 6.60 mmHg and in Group N 57.23 ± 4.86 mmHg, P = 0.003 [Table 4].

Similarly, in a study of Potdar et al, SBP (mmHg) at 5th min in 4 mg ondansetron group was 114.71 ± 19.08 and in patients where 8 mg ondansetron group was 109.43 ± 21.44. SBP in placebo group at 5th min was 107.82 ± 15.78. The difference between placebo and 4 mg group was statistically significant, P = 0.005. At 10th min, SBP (mmHg) in the placebo group, 4 mg ondansetron group, and 8 mg ondansetron group were 103.64 ± 24.12, 108.45 ± 14.02, and 101.67 ± 37.96, respectively. The difference between placebo and 4 mg group was statistically significant not for 8 mg group (P = 0.03) [Table 10 and Graphs 5 and 6].

In their study, there was a significant difference in DBP (mean ± SD) (mmHg) between the placebo and ondansetron group when compared at 5th min. In group using 4 mg ondansetron, DBP was 66.82 ± 13.22 and in the placebo group 60.92 ± 11.17 (P =0.03) [Table 5 and Graphs 5 and 8].

Our study was in concordance to the study by Potdar et al where there was a significant difference in MAP at 5th and 10th min which was observed between the control group and ondansetron group. MAP (in mmHg) in the ondansetron group at 5th and 10th min was 83.78 ± 17.47 and 80.90 ± 11.09, respectively, whereas it was 79.08 ± 15.31 and 75.51 ± 17.93 in the control group (P = 0.02) [Table 7 and Graph 9].

Yet, another variation was observed in different models of studies on ondansetron for the prevention of post-spinal hypotension in their oxytocin infusion protocol after delivery of fetus.

Ortiz-Gómez et al used low doses of oxytocin in the form of IV bolus than continuous infusion at 2.5 U/h, whereas Trabelsi et al used bolus of 5 U oxytocin then 2.5 U/hr.

Wang gave 10 U of oxytocin in 250 ml NS slow infusion. Mohamed et al used 5 U of oxytocin bolus just after delivery of fetus followed by 40 U infusion. Here, in the present study, we used 20 U of oxytocin in 500 ml of normal saline infusion at 10 ml/min. This factor needs to be mentioned because it has higher propensity to alter the maternal hemodynamics, but oxytocin infusion post-delivery was equally employed in either group of patients. Bolus doses of oxytocin cause profound hypotension, which are avoided in this study.

In our study, the total average consumption of vasopressor in each patient in Group O was 2.60 ± 4.36 mg, whereas it was 5.6 ± 4.43 mg in Group N patients (P = 0.0107) [Table 11 and Graph 10].

Similarly, Hajjan et al showed decreased consumption of vasopressor in the ondansetron group when compared to placebo. The total amount of ephedrine consumption in the ondansetron group was 5.8 mg and 10.7 mg in the placebo group with significant difference in between two groups (P = 0.009) [Graph 11].

Observations from the study of Trabelsi et al also support our study where average ephedrine consumption was 5.10 ± 7.78 mg in the ondansetron group in comparison to 12.90 ± 9.24 mg in group using normal saline (P < 0.001). There was no incidence of bradycardia reported in either group which requires treatment using i/v atropine.

In our study, neither in Group O nor in Group N, no case of significant bradycardia is reported, but its occurrence is infrequent.

The phenomenon of the incidence of bradycardia due to spinal anesthesia found to be independent from hypotension.

In our study, there was no significant difference in time to delivery of fetus and duration of surgery between two groups (mean ± SD). In Groups O and N, time to delivery

<table>
<thead>
<tr>
<th>Time up to delivery of fetus (min)</th>
<th>Group O Mean ± SD</th>
<th>Group N Mean ± SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>79.26 ± 8.29</td>
<td>77.56 ± 6.90</td>
<td>0.39</td>
</tr>
<tr>
<td>3</td>
<td>78.06 ± 7.66</td>
<td>76.96 ± 6.31</td>
<td>0.54</td>
</tr>
<tr>
<td>6</td>
<td>77.60 ± 8.13</td>
<td>76.43 ± 5.57</td>
<td>0.51</td>
</tr>
<tr>
<td>9</td>
<td>76.83 ± 7.34</td>
<td>75.63 ± 5.48</td>
<td>0.81</td>
</tr>
<tr>
<td>12</td>
<td>76.63 ± 6.68</td>
<td>74.44 ± 4.15</td>
<td>0.13</td>
</tr>
<tr>
<td>15</td>
<td>75.17 ± 6.77</td>
<td>73.54 ± 4.89</td>
<td>0.31</td>
</tr>
<tr>
<td>21</td>
<td>73.83 ± 6.41</td>
<td>72.92 ± 4.83</td>
<td>0.65</td>
</tr>
<tr>
<td>24</td>
<td>73.62 ± 7.61</td>
<td>72.87 ± 4.05</td>
<td>0.81</td>
</tr>
<tr>
<td>27</td>
<td>76.0 ± 7.07</td>
<td>72.66 ± 3.51</td>
<td>0.62</td>
</tr>
</tbody>
</table>

Table 1: Pulse rate (per min) up to delivery of fetus every 3rd min in both groups.
of fetus was 18.96 ± 3.05 min and 18.16 ± 3.24 min, respectively ($P = 0.33$). The duration of surgery in Group O was 57.20 ± 4.24 min, while in Group N, it was 55.66 ± 7.18 min, ($P = 0.31$) [Table 12 and Graph 12].

Blauw et al. demonstrated the vasodilatory effect of serotonin by injecting serotonin into radial artery of healthy volunteers and this effect was vanished off by administrating 5HT3 antagonist. [28] Serotonin receptors are present peripherally as well in central nervous system (CNS) and serotonergic mechanism of CNS supposed to be a factor involved in cardiovascular collapse after spinal anesthesia.[29] On the contrary, ondansetron shows poor permeability across blood–brain barrier.

Although there is no evidence in favor of direct effect of 5HT3 receptor antagonism on cardiac output, ondansetron is believed to abolish the Bezold–Jarisch reflex and hypotension after spinal anesthesia through blocking the
Table 8: Demographic variables in both groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group O Mean ±SD</th>
<th>Group N Mean ±SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>26.13 ± 3.80</td>
<td>24.73 ± 2.59</td>
<td>0.10</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>59.83 ± 3.40</td>
<td>59.36 ± 4.75</td>
<td>0.66</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>151.16 ± 3.34</td>
<td>151.83 ± 2.98</td>
<td>0.41</td>
</tr>
</tbody>
</table>

Table 9: Pre-operative vitals in both groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group O Mean ±SD</th>
<th>Group N Mean ±SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse rate (per min)</td>
<td>77.5 ± 9.11</td>
<td>76.23 ± 7.71</td>
<td>0.56</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>114.36 ± 8.07</td>
<td>114.03 ± 5.24</td>
<td>0.85</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>67.13 ± 5.20</td>
<td>66.36 ± 3.39</td>
<td>0.50</td>
</tr>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>82.93 ± 3.90</td>
<td>82.23 ± 3.26</td>
<td>0.45</td>
</tr>
<tr>
<td>SpO2</td>
<td>98.76 ± 0.85</td>
<td>98.66 ± 0.75</td>
<td>0.63</td>
</tr>
<tr>
<td>RR (per min)</td>
<td>15.73 ± 1.80</td>
<td>15.46 ± 1.88</td>
<td>0.58</td>
</tr>
</tbody>
</table>

Table 10: Systolic blood pressure (mmHg) after delivery of fetus up to the completion of surgery every 5th min

<table>
<thead>
<tr>
<th>Time after delivery</th>
<th>Group O Mean ±SD</th>
<th>Group N Mean ±SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>of fetus (min)</td>
<td>Mean</td>
<td>Mean</td>
<td>Mean</td>
</tr>
<tr>
<td>0</td>
<td>105.60 ± 5.36</td>
<td>106.83 ± 3.63</td>
<td>0.30</td>
</tr>
<tr>
<td>5</td>
<td>107.13 ± 4.41</td>
<td>108.00 ± 3.42</td>
<td>0.39</td>
</tr>
<tr>
<td>10</td>
<td>108.23 ± 4.31</td>
<td>108.60 ± 3.15</td>
<td>0.70</td>
</tr>
<tr>
<td>15</td>
<td>109.66 ± 3.92</td>
<td>108.90 ± 2.69</td>
<td>0.38</td>
</tr>
<tr>
<td>20</td>
<td>110.23 ± 4.26</td>
<td>108.90 ± 2.23</td>
<td>0.13</td>
</tr>
<tr>
<td>25</td>
<td>109.50 ± 3.63</td>
<td>109.20 ± 2.69</td>
<td>0.71</td>
</tr>
<tr>
<td>30</td>
<td>109.53 ± 3.43</td>
<td>109.56 ± 2.80</td>
<td>0.96</td>
</tr>
<tr>
<td>35</td>
<td>110.16 ± 4.82</td>
<td>109.69 ± 2.51</td>
<td>0.64</td>
</tr>
<tr>
<td>40</td>
<td>110.19 ± 4.05</td>
<td>109.66 ± 2.76</td>
<td>0.63</td>
</tr>
<tr>
<td>45</td>
<td>111.81 ± 3.02</td>
<td>110.45 ± 3.50</td>
<td>0.34</td>
</tr>
</tbody>
</table>

Table 11: Dose of vasopressor used in both groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group O Mean ±SD</th>
<th>Group N Mean ±SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasopressor (in mg)</td>
<td>2.60 ± 4.36</td>
<td>5.6 ± 4.43</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Table 12: Time to delivery of fetus and total duration of surgery in both groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group O Mean ±SD</th>
<th>Group N Mean ±SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to del. of fetus (min)</td>
<td>18.96 ± 3.05</td>
<td>18.16 ± 3.24</td>
<td>0.33</td>
</tr>
<tr>
<td>Total duration of surgery (min)</td>
<td>57.2 ± 4.24</td>
<td>55.66 ± 7.18</td>
<td>0.31</td>
</tr>
</tbody>
</table>

5HT3 receptor. White et al. observed the efficacy of iv 5HT3 blocker granisetron in suppressing hypotension and bradycardia in rabbit model.\[15\]

In our study, low incidence of hypotension was observed in Group O than Group N in initial intraoperative period after spinal anesthesia. After the delivery of fetus, no significant difference was observed for hypotension in between two groups. There is a limitation of our study that
we did not conduct a study on dose-dependent response of ondansetron in prevention of hypotension in that we used only single dose of ondansetron. Due to the limitation of resources, fetal outcome was also not measured using lactate level of cord blood. This is further aspect of the study to be reevaluated that least altered maternal hemodynamics in initial postpartum period have better impact on fetal well-being.
In conclusion, our study revealed the similarity in study model from various past studies and all these studies support the concept of our study Ortiz, Sahoo, Owczuk, Potdar etc., gave prophylactically 4 mg iv ondansetron before spinal anesthesia which works on cardiac level by abolishing Bezold–Jarisch reflex and peripherally which causes less fall in SBP, DBP, and MAP and is effective in preventing post-spinal anesthesia-induced hypotension. Rather, this effect was not consistently observed throughout intraoperative period, but lasts only for initial few minutes.

**SUMMARY AND CONCLUSION**

Out of 60 patients included in our study, 30 patients were randomly allocated into Group O (ondansetron) and another 30 patients were allocated into Group N (normal saline). Group O received 4 mg ondansetron, diluted up to 10 ml with normal saline and Group N received 10 ml of normal saline over 1 min, 5 min before spinal anesthesia.

There was no significant difference in PR in intraoperative period between two groups. There was a significant difference in SBP at 6th min after giving spinal anesthesia. There was a significant difference in DBP at 0 min, assuming supine position just after giving spinal anesthesia. Statistically significant difference was there in MAP in both groups at 0, 3rd, and 6th min after giving spinal anesthesia. No significant difference was observed in PR, SBP, DBP, and MAP in groups after delivery of fetus up to the completion of surgery.

Incidence of hypotension was significantly higher in Group N. Vasopressor (mephentermine) consumption (mean±SD) in each patient in Group O was significantly lesser in comparison to Group N. No incidence of bradycardia was reported in either group. Non-pharmacological measures were taken to prevent shivering, no other side effects were seen in the study.

In our study, considering all these observations, it is concluded that 4 mg iv ondansetron given before spinal anesthesia prevents fall in BP, but its effect exists for initial minutes.

**REFERENCES**


Sharma and Maravi: Effect of Ondansetron in Prevention of Hypotension in Elective Lower segment Cesarean Section under Spinal Anesthesia: A Randomized, Double-Blind Study


Source of Support: Nil, Conflicts of Interest: None declared.