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Prophylactic Intravenous Ondansetron for Hemodynamic Stability and Shivering Prevention in Elective Cesarean Section under Spinal Anesthesia: Review Article

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ABSTRACT

Background: Due to sympathetic blocking and aortocaval compression by the gravid uterus, spinal anesthesia (SA) during elective Cesarean section (CS) typically causes substantial hypotension and bradycardia, which commonly causes mother pain and fetal impairment. Hemodynamic instability and oxygen demand are further worse by concurrent postoperative shivering. Because traditional preventative measures are still ineffective, research into alternative pharmaceutical therapies to improve maternal hemodynamic stability and comfort is being conducted.

Objective: This review aimed toassess the efficacy of prophylactic IV ondansetron in preventing SA–induced hypotension, bradycardia, and shivering in adult patients undergoing elective CS.

Methods: From 2020 to 2025, a thorough search was carried out in PubMed, Google Scholar, and Science Direct using the following keywords: Ondansetron, SA, Hypotension, Bradycardia, Shivering and Elective CS. The reviewers also assessed the references to pertinent literature. Only the most recent or comprehensive study was considered. Oral presentations, dissertations, conference abstracts, and unpublished papers are a few examples of works that weren't considered important scientific study. Documents published in languages other than English were ignored as a result of lack of translation resources.

Conclusions: Prophylactic IV ondansetron demonstrated high efficacy for reducing the incidence of SA–induced hypotension, bradycardia, and postoperative shivering, significantly lowering vasopressor requirements. Its rapid onset, favorable maternal and fetal safety profile, and accessibility made it a valuable adjunct for enhancing perioperative hemodynamic stability and patient comfort during Cesarean delivery. Further large-scale trials are warranted to standardize dosing and administration protocols.

Keywords: Ondansetron, SA, Hypotension, Bradycardia, Shivering, Elective CS.

INTRODUCTION

SA is frequently utilized in CS procedures. The most significant side effects of SA are bradycardia and hypotension brought on by sympathetic blocking, which occurs between 55 to 100 percent of the time. But, hypotension is more likely when the gravid uterus blocks the venous return [(1)].

Uncomfortable symptoms including shivering, nausea, and vomiting are frequently linked to SA-induced hypotension in the mother. Long-term hypotension in mothers can cause major side effects, including aspiration of stomach contents, apnea, cardiovascular collapse, and loss of consciousness. Furthermore, in situations of prolonged hypotension, uteroplacental blood flow declines, and harmful neonatal outcomes, including fetal acidosis and fetal mortality, may transpire. For the mother's and the newborn's health, SA-induced hypotension during CS must be avoided [(2)].

Additionally, shivering frequently follows SA. Shivering is a rhythmic, unconscious action that uses many muscle groups. Increased carbon dioxide generation, lactic acidosis, and oxygen consumption are all caused by increased muscular activity [(3)].

Researchers examined the complications of the Bezold–Jarisch reflex (BJR) in recent years. The trio of bradycardia, hypotension, and apnea is part of this reaction. Researchers have proposed that the incidence of the BJR following SA is significantly influenced by serotonin and 5-hydroxytryptamine 3 (5-HT3) receptors. There are 5-HT3 receptors in the spine, lung, and heart.

SA-induced decreased venous return activates cardiac chemoreceptors, increasing parasympathetic activity and causing bradycardia and hypotension. According to research, 5-HT3 antagonists may reduce SA-induced hypotension by preventing peripheral vasodilatation, lowering the BJR, and boosting venous return to the heart [(4)]. In this review, we aimed to evaluate the value of prophylactic intravenous (IV) infusion of ondansetron before SA on the hemodynamic parameters and shivering in patients undergoing elective CS.

SPINAL ANESTHESIA IN CS

The most popular kind of anesthesia for lower segment CSs (LSCS) is SA. SA has a high success rate, a defined end goal, and is quicker and simpler to do than epidural method. Particularly when using hyperbaric solutions, it generates blocks quickly, densely, and predictably. The risk of stomach contents aspiration and regurgitation is low. The danger of fetal toxicity is low, and there is very little chance that the medicine will cross the placenta and reach the fetus. The mother is able to enjoy the interaction with her infant when she is awake [(5)].

Pre-operative evaluation: The anesthesia team should evaluate every patient having CS. In order to gather information about co-existing conditions, the patient's anesthesia and obstetric history, and a comprehensive examination that includes a back and airway assessment, a comprehensive pre-anesthesia evaluation is conducted.

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The availability of supplies and drugs to properly provide general anesthesia in the event of an unforeseen emergency and challenging airway must always be taken into account, even when SA is planned [6].

The practice of fasting during labor has persisted despite the lack of compelling evidence that it improves outcomes for the mother or the baby. Therefore, it is recommended that women have a 6-hour solid fast before to a simple elective CS, but they may have clear liquids up to two hours prior to anesthesia. All patients are presumed to have a full stomach in emergency situations. Generally speaking, an H2-blocker and a non-particulate antacid, with or without metoclopramide, should be administered prior to any CS [7].

Supine hypotension syndrome is caused by the gravid uterus compressing the inferior vena cava when the woman is in the supine position during term pregnancy. Vasopressors are not a simple way to treat the severe hypotension that follows from this condition. It is not as serious as previously believed that the aortic compression is. Similar amounts of compression are caused by twin and singleton pregnancies. In order to prevent aortocaval compression, precautions should be taken both before and after SA. These include a leftward tilt of the table, a wedge under the right buttock, or even the obstetrician manually moving the uterus to the left [6].

In an effort to lower the risk of spinal-induced hypotension, preloading or co-loading with crystalloids before to or during SA is a common technique. Because it is isoosmolar with plasma, Ringers Lactate (also known as Hartmann's solution or Compound Sodium Lactate) is the most favored crystalloid for both preloading and maintenance. It can also be used as an oxytocin transporter. Since intravenous Dextrose (5%) in water has hypotonic qualities in vivo and may cause water retention when combined with oxytocin, it is not the best option to use as a carrier. Neonatal hypoglycemia, acidosis, and fetal hyperglycemia are other possible risks. However, when there are obvious signs, such a diabetic condition, dextrose can be administered [8].

Administration of SA: One of two positions is used for the intrathecal injection; seated or lateral. When attempting a combined spinal and epidural procedure, or when identifying landmarks is challenging, as in the case of obese individuals, the sitting posture is recommended. A sensory block up to the T4–T6 segmental level is the goal. Dyspnea may result from sensory blockades that extend past the T4 segmental level because intercostal muscle paralysis impairs the ability to feel the expansion of the chest and to voluntarily exhale. Until the fetus is sufficiently extracted, quiet comfort and encouragement to breathe deeply are necessary; no sedative should be given. After the baby is delivered, this feeling goes away since the uterus is empty and constricted, which improves breathing motions. Analgesics like fentanyl or alfentanil can be used to lessen pain related to uterine exteriorization and traction on the peritoneum [6].

The L3-L4 level is the chosen location for intrathecal injection in order to guarantee that the needle is entered well beyond the spinal cord's termination. Because they are less likely to cause post-dural puncture headache (PDPH) than Quincke cutting tip needles, pencil-point needles (Sprotte or Whitacre type) with thin gauge (sizes 25F to 27F) are recommended. Quincke needles, which have the thinnest gauge, can be used in the absence of pencil point needles. Following the observation of free flow of cerebrospinal fluid (CSF), the selected dosage of a local anesthetic medication is administered [9].

Lignocaine (lidocaine) 5% injection has a quicker start and a moderate half-life of 45 to 75 minutes, nevertheless its usage has been restricted due to reports of transient neurological symptoms (TNS) associated with hyperbaric lignocaine. Bupivacaine injections are well-liked because of their quick onset, prolonged duration of action, and effective muscular relaxation. For the parturient, 10–12 mg of intrathecal heavy bupivacaine 0.5% is the ideal dosage. When administered at a dosage of 4–12 mg, injection levobupivacaine, a pure S (-) enantiomer of racemic bupivacaine, exhibits an effectiveness that is comparable to 0.5% heavy bupivacaine [5].

To give postoperative analgesia following LSCS, opioids can be administered to neuraxial local anesthetic. Intrathecal local anesthetics can be supplemented with 0.10–0.25 mg of preservation-free morphine to extend postoperative analgesia for 18–24 hrs. When administered appropriately, 5 mcg dexmedetomidine added to hyperbaric bupivacaine can enhance and prolong SA without having any negative effects on neonates, according to recent research [7].

Table (1): Dosage ranges for different local anesthetic agents and additives

Duration (min)	Dosage range (mg)	Drug
45–75	60–75	Lignocaine (5%) Heavy
60–120	7.5–15.0	Bupivacaine (0.5%)
60-90	10-15	Ropivacaine (0.75%)
60-120	8-12	Levobupivacaine
120–180	7.0–10.0	Tetracaine
30–60	100–150	Procaine
Adjuvant drugs		
—	0.1–0.2	Epinephrine
360–1080	0.1–0.25	Morphine
180–240	0.010–0.025	Fentanyl

Monitoring and post-operative care: Electrocardiograms, non-invasive blood pressure (BP) checks, and pulse oximetry should all be required forms of monitoring. Initially, BP should be tested every two to three minutes since sharp drops are expected, requiring quick action. The larger uterus in the supine posture may create aorto-caval compression, which might worsen the hypotension brought on by sympathetic block. Before giving SA, vasopressors such as ephedrine, phenylephrine, mephentermine, or metaraminol should be prepared in a syringe and kept on hand. Phenylephrine infusions (100 mcg/min) are more successful in avoiding hypotension. Bradycardia or tachycardia should be watched for in all patients [(10)].

As a result of hypotension, tachycardia linked to labor pain may persist for a while. There are two possible causes of intra-operative bradycardia: Increased spinal blocking and vagal stimulation brought on by peritoneal traction. There is a possibility of amniotic fluid embolism or venous air embolism, thus monitoring is particularly crucial when the uterine sinuses remain open until the suturing is finished. When certain obstetricians exteriorize the uterus, there may be a higher chance of an air embolism [(11)].

Continued postoperative care is necessary until SA's effects have fully subsided. It is recommended to maintain monitoring for PDPH for 48 hours. After SA for LSCS, any headache should be evaluated for PDPH. Any rupture in the dura mater is linked to increased CSF loss and a subsequent drop in CSF pressure, which is elevated during pregnancy. A strong headache, stiff neck, nausea, tinnitus, and photophobia are among the patient's symptoms. PDPH is often self-limited and may be treated conservatively with bed rest, frequent analgesics, and intravenous or oral fluids. When supportive treatments are insufficient to manage PDPH, an epidural blood patch is regarded as the gold standard [(10)].

However, additional negative outcomes, such meningitis or neurological abnormalities, can happen during a blood patch surgery, and the epidural blood patch technique itself may result in another unintentional dural puncture. Patients with PDPH or those whose conservative therapy has failed have had their chronic headaches treated with the straightforward, minimally invasive surgery of bilateral greater occipital nerve block. Postdural puncture headache has also been suggested to be treated with transnasal sphenopalatine ganglion block (SPGB) [(5)].

ANESTHETIC COMPLICATIONS DURING CS

To assist prevention of common problems related to neuraxial anesthesia, appropriate patient selection and care should be established. Even though many of the complications are quite rare, it's still important to be aware of them. Although severe problems are thought to be very uncommon, their occurrence is most likely underreported. Backache, post-dural puncture headache, nausea, vomiting, hypotension, hearing loss, total SA, neurological damage, spinal hematoma, arachnoiditis, and shivering are a few of the frequent side effects [(10)].

Shivering after SA: Shivering is characterized by an involuntary shivering of the body brought on by a contraction of the muscles. In an effort to increase metabolic heat generation and sustain the temperature, the body's core temperature rises, triggering this physiological reaction. Shivering, however, is linked to a number of negative outcomes, including increased metabolic heat generation, increased oxygen consumption, and carbon dioxide production, which causes hypoxemia and myocardial ischemia, increased

pain in the wound, delayed healing of the wound and disruption of monitoring [12].

Shivering during surgery is mostly caused by anesthetic administration and operation. The primary causes of surgical patients' susceptibility to shivering, however, are perioperative heat loss, skin exposure in a cool operating room, evaporation from exposed areas, the administration of unwarmed fluids, the systematic release of pyrogens, pain, and inhibition of the thermoregulation system by blocking tonic vasoconstriction. Up to 50% to 65% of people have been observed to experience shivering as a result of receiving SA. Vasodilatation is the mechanism via which SA induces shivering. This transfer of body heat from the core to peripheral tissue increases fast heat loss, leading to hypothermia and shivering [13].

In order to make meaningful comparisons of therapies, it is essential to grade the degree of shivering. The Bedside Shivering Assessment Score (BSAS) typically uses a scale such as grade zero for no shivering, grade one for mild fasciculation of the face or neck or peripheral vasoconstriction but no visible shivering, grade two for visible muscular activity in only one muscle group, grade three for muscular activity in multiple muscle groups but not generalized, and grade four for gross muscular activity involving the entire body [14]. Shivering can be prevented and treated using both non-pharmacological and pharmacological approaches. Non-pharmacological interventions include providing warm fluids and clothing, using radiant heat and forced air warmers, and raising the operating theater's ambient temperature. Furthermore, it can be treated pharmacologically with pethidine, tramadol, clonidine, dexmedetomidine, biogenic amines (serotonin 5-HT3 receptor antagonist), low dosage ketamine, dexamethasone, and magnesium sulfate. However, due to the safety, expense, and unavailability of those interventions in settings with limited resources, we are forced to use alternative methods, such as pre-warming patients for 15 mins using a cotton blanket, warming gowns, giving warm fluids, and using low doses of ketamine, tramadol, dexamethasone, and magnesium sulfate as prevention and management based on the evidence that is currently available. Consequently, the use of these substitute treatments minimizes the undesirable side effects of the drugs in question, helps hospitals and patients save needless expenses, and opens the door for more study in this area [12].

Hypotension after SA: Because SA causes sympathetic vasomotor inhibition, systemic vascular resistance is reduced, mostly due to arterial dilatation but also to some vasodilation. Baroreceptors mediate compensation in healthy persons, leading to vasoconstriction in the unblocked segments and an increase in heart rate and stroke volume. The cardiovascular system's capacity to adjust is hampered in a term pregnant woman since baseline heart rate, stroke volume, and cardiac output are already elevated to fulfill the fetus's metabolic needs. The inferior vena cava is compressed by the gravid uterus when the patient is supine, which lowers cardiac output and venous return [10].

Fetal oxygenation and nutrition supply may be impacted by hypotension, which can drastically lower blood flow to the placenta. Fetal acidity, decreased Apgar scores (which evaluate a baby's health at delivery), and perhaps long-term neurological problems in the infant are all consequences of this. Additionally, maternal nausea and vomiting may be exacerbated by decreased blood supply to the brainstem, specifically the vomiting area. This problem can be reduced with effective blood pressure management [6].

Using a vasopressor medication effectively is the cornerstone of treatment. Adjunctive preventive methods include intravenous fluids, a minimum pelvic tilt of 15, and left lateral displacement of the uterus using a wedge or table tilting. It has been discovered that preloading with intravenous crystalloid fluids before to SA is mainly unsuccessful in lowering hypotension. It works better to preload with intravenous colloids. Thus, it is advised to administer a free-flowing infusion of 1 L crystalloid as soon as possible following the spinal injection to initiate SA, utilizing a pressure bag if necessary [15].

PHARMACOLOGY OF ONDANSETRON

Indications: Serotonin is blocked centrally in the chemoreceptor trigger zone, peripherally, and on gastrointestinal (GI) vagal nerve terminals by selective serotonin receptor (5-HT3) antagonists. Strong antiemetic effects are the outcome of this blockage. Four 5-HT3 receptor antagonists are currently available on the market: Ondansetron, granisetron, dolasetron, and palonosetron. These drugs have FDA approval for preventing nausea and vomiting in both adults and children associated with radiation therapy, chemotherapy, and the effects of postoperative anesthesia. Additionally, dolasetron can be used to alleviate postoperative nausea and vomiting in both adults and children [16].

Numerous formulations provide patients and professionals a wide range of choices for efficient dosage delivery. Constipation, exhaustion, and headaches are the most frequent side effects. In rare cases, 5-HT3 receptor antagonists may result in QTc prolongation if they are used with other drugs that prolong QTc or in people who have a history of congenital long-QT syndrome. Serotonin syndrome instances in vulnerable patient groups have also been reported. Additionally, there is the issue of concealing

intestinal obstruction symptoms in older or postoperative patients. There is no known lethal dosage for 5-HT3 receptor antagonists, and overdose is uncommon. In these situations, the primary treatment is supportive. All things considered, some serotonin receptor antagonists constitute a potent class of antiemetic drugs with a broad therapeutic index and a low incidence of adverse effects [17].

Adult FDA-approved indications:

- Preventing nausea and vomiting brought on by chemotherapy
- Preventing nausea and vomiting brought on by radiation therapy
- Preventing nausea and vomiting following surgery

Adult Non-FDA-approved indications:

- Management of nausea and vomiting following surgery
- Severe or persistent nausea and vomiting throughout pregnancy [17].

Mechanism of action: Selective serotonin receptor (5-HT3) antagonists have potent antiemetic effects by blocking serotonin centrally in the chemoreceptor trigger zone in the region postrema of the fourth ventricle and peripherally on vagal nerve terminals in the GI system [16].

Adverse events:

1) **1% to 10% incidence:** The following symptoms may be present: Drowsiness, sedation, dizziness, agitation, anxiety, paresthesia, cold, pruritus, skin rash, diarrhea, gynecologic illness, urinary retention, elevated serum aminotransferases (more than two times), injection site response, hypoxia, and fever [18].
2) **Less than 1%:** Abdominal pain, accommodation disturbance, anaphylactoid reaction, anaphylaxis, angina pectoris, angioedema, atrial fibrillation, bradycardia, bronchospasm, bullous skin disease, cardiac arrhythmia, cardiorespiratory arrest, chest pain, chills, depression of ST-segment on ECG, dyspnea, dystonic reaction, ECG changes, extrapyramidal reaction, flushing, hepatic failure, hiccups, hypersensitivity reaction, hypokalemia, hypotension, ischemic heart disease, laryngeal edema, laryngospasm, liver enzyme disorder, mucosal tissue reaction, myocardial infarction, neuroleptic malignant syndrome, oculogyric crisis, palpitations, positive lymphocyte transformation test, prolonged QTc interval on ECG (dose-dependent), second-degree atrioventricular block, serotonin syndrome, shock, Stevens-Johnson syndrome, stridor, supraventricular tachycardia, syncope, tachycardia, tonic-clonic seizures, torsades de pointes, toxic epidermal necrolysis, transient blindness, transient blurred vision, urticaria, vascular occlusive events, ventricular premature contractions, ventricular tachycardia, weakness and xerostomia [16].

Use in Pregnancy: FDA pregnancy category B includes 5-HT3 receptor antagonists. Early pregnancy is not associated with a high incidence of congenital abnormalities, according to human research that are currently available. Cleft palate and septal abnormalities are somewhat more likely. There is no elevated risk in the early stages of pregnancy, according to animal research. The presence of 5-HT3 receptor antagonists in breast milk is unknown [19].

Contraindications: The following are the main relative contraindications: (1) Intolerance to 5-HT3 receptor antagonists or any of the formulation's ingredients because of possible cross-reactivity. (2) Concurrent usage with apomorphine because of the possibility of hypotension and a lowered state of consciousness [20].

Toxicity: There is now no known lethal dosage, and overdose is uncommon. 5-HT3 receptor antagonists seldom cause moderate side effects and have a wide therapeutic index. The main focus of treatment is support. Keep an eye out for the uncommon conditions of serotonin syndrome, ventricular arrhythmias, disguised intestinal obstruction or ileus, and ECG abnormalities. There is minimal financial incentive or clinical necessity for antidote development because the lethal dosage is unknown and there are very few reported cases of overdose resulting in major morbidity or fatality. Furthermore, amiodarone, amisulpride, apomorphine, and bosentan have a few noteworthy pharmacological interactions with 5-HT3 antagonists [20].

Ondansetron uses in SA: One of the cutting-edge treatment strategies being used to combat the well-known hemodynamic side effects of SA, bradycardia and hypotension, is the use of 5HT3 receptor antagonists. Thirty-three percent of individuals with hypotension and thirteen percent with bradycardia have the worsening hemodynamic aftermath of SA. However, because of the gravid uterus's additional constriction of the vasculature, these numbers soar to 75% in patients having CS [21].

Massive morbidity results from hemodynamic instability, which can cause moderate symptoms like headache, nausea, and vomiting, but if left untreated, can cause severe symptoms including altered sensorium, cardiac arrest, and even death. In a similar vein, deteriorating hemodynamic state triggers fetal discomfort. The use of rescue drugs, such as vasopressors (phenylephrine and ephedrine) and atropine, which all have negative effects on mothers

and/or newborns, is one of the current treatment approaches used to address this problem [10].

While the local anesthetic agent's intrathecal injection stops sympathetic outflow in the affected areas, causing parasympathetic overdrive and a subsequent drop in blood pressure and heart rate, this decrease in hemodynamic parameters activates 5HT3 receptors in the left ventricle's inferoposterior wall. As a result, the vasomotor center is inhibited, which lowers blood pressure and heart rate even further (the Bezold Jarisch reaction). Therefore, preventative blockage of these receptors will reduce the risk of bradycardia and hypotension, which will eventually reduce the need for the previously described rescue drugs [6].

CONCLUSION

Prophylactic IV ondansetron demonstrated high efficacy for reducing the incidence of spinal anesthesia–induced hypotension, bradycardia, and postoperative shivering, significantly lowering vasopressor requirements. Its rapid onset, favorable maternal and fetal safety profile, and accessibility made it a valuable adjunct for enhancing perioperative hemodynamic stability and patient comfort during Cesarean delivery. Further large-scale trials are warranted to standardize dosing and administration protocols.

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