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LETTER TO THE EDITOR

Vasopressors for hypotension in spinal anesthesia for cesarean section

Anna Maria Biava[1] · **Gianni Cipriani**[1] · **Endrit Malja**[1] · **Federico Bilotta**[2]

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To the Editor:

We read with great interest the article "Epinephrine vs. phenylephrine infusion for prophylaxis against maternal hypotension after spinal anesthesia for cesarean delivery: a randomized controlled trial" by Yasmin S. Hassabelnaby and colleagues [1]. This study notably investigates both maternal and fetal/neonatal consequences of arterial hypotension following spinal anesthesia for caesarean delivery and the effects of vasopressors pharmacological treatment. Spinal anesthesia for cesarean section often causes arterial hypotension because of sympathetic vasomotor blockade. Maternal symptoms such as nausea, vomiting, and dyspnoea often accompany severe hypotension and adverse effects on the fetus due to decreased uterine blood flow with umbilical acidosis, fetal bradycardia and decreased neonatal Apgar score. The Authors compared two commonly used vasopressor in obstetric practice, epinephrine and phenylephrine. In the study a total of 196 full-term pregnant women who underwent elective caesarean delivery were randomized to two groups: 98 to epinephrine 0.03 mcg/kg/min (epinephrine group) and 98 to phenylephrine 0.4 mg/kg/min (phenylephrine group) infusion that started after spinal anesthesia and continued until 5 min after delivery. All patients were monitored with 5 lead electrocardiography, pulse oximetry and non-invasive blood pressure and baseline systolic blood pressure was recorded. They received 10 mg metoclopramide followed by a co-load of 15 ml/kg of Ringer's solution. Spinal anesthesia was performed with 10 mg hyperbaric bupivacaine plus 20 mcg fentanyl and after injection patient was positioned in supine position with a left lateral tilt. The primary outcome was a composite outcome of the occurrence of hypotension, hypertension, bradycardia and/or tachycardia. The secondary outcomes were the incidence of hypotension and number of hypotensive episodes; incidence of bradycardia, hypertension and tachycardia, total dose of phenylephrine, epinephrine and ephedrine; incidence of intraoperative nausea and vomiting. Neonatal outcomes were umbilical artery blood gas and Apgar Score. Reported results suggest that epinephrine and phenylephrine produced comparable maternal hemodynamic and neonatal outcomes, however, in epinephrine group was observed a higher incidence of tachycardia and likely a lower incidence of hypotension than phenylephrine group.

In obstetric anesthesia best prophylactic vasopressor drug should associate with the less possible incidence of hypotension restoring systemic vascular resistance, which is predominantly due to α agonist activity, and should also minimize other cardiac adverse events, such as tachycardia, bradycardia and hypertension [2]. Maternal and neonatal adverse effects were related to the severity and duration of hypotension with duration may be more important than severity [2]. Epinephrine has a high affinity for α1-, β1-, and β2-adrenergic receptor with α1 effects more significant at high doses while β effects are predominant in a low dose with a better-preserved heart rate [3], while phenylephrine is a direct α1 agonist with no β effect but it may induce at higher doses bradycardia with consequent reduction in maternal cardiac output (CO) [4]. Maternal CO is closely related to uteroplacental blood flow [5].

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✉ Anna Maria Biava
annamariabiava@gmail.com

Gianni Cipriani
giannicipriani56@libero.it

Endrit Malja
eniabi@yahoo.it

Federico Bilotta
federico.bilotta@uniroma1.it

[1] Department of Anesthesiology, Fatebenefratelli San Pietro Hospital, Rome, Italy

[2] Department of Anesthesiology and Critical Care, Sapienza University of Rome, Rome, Italy

The study is interesting but there are some methodological issues that should be better detailed:

Regarding non-pharmacological measures to prevent and treat hypotension due to sympathetic vasomotor block caused by spinal anesthesia, in this study the patients were positioned supine with a left lateral tilt immediately after the injection of local anesthetic into the subarachnoid space. However, the Authors did not specify the angle of the table tilt. To reduce inferior vena cava compression and, obtain a cardiac output (CO) average, recommended left uterine displacement with a left lateral tilt at $\geq 15°$ while if patients were tilted to $< 15°$ CO decreases by more than 20% due to severe inferior vena cava compression [6]. Furthermore, when manual displacement of the uterus was compared with a left lateral tilt of 15° it was found that the incidence of hypotensive events was lower [7]. We wonder if the authors considered that manual displacement of the uterus could be more effective for reducing hypotension than left lateral tilt.

Other measure to prevent hypotension by spinal anesthesia is intravascular fluid loading and crystalloid co-loading may be more effective than pre-loading to decrease hypotension and vasopressor requirements [2]. In this study, all patients received 15 mL/kg crystalloid co-loading. We ask if the authors consider that the patient's preoperative volemic status could be different. Did they consider volume status assessment with a non-invasive approach such as ultrasonography such as transthoracic echocardiography (TTE)?

In the present study, hemodynamic changes were measured as heart rate and non-invasive systolic blood pressure. Changes in CO are more closely related to uteroplacental blood flow than systolic blood pressure [5]. The measurements of systolic blood pressure (SBP) can be used as a surrogate for maternal CO in predicting uterine blood flow. However, with the reliable non-invasive techniques of assessing maternal CO, it can now be measured reliably and accurately [4, 5]. We wonder if the authors have also considered using non-invasive CO measurement techniques to make their results more reliable on the comparison of the efficacy and safety of the two vasopressor agents for the management of hypotension after spinal anesthesia in cesarean section.

Declarations

Conflict of interest The authors have no conflicts of interest.

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