CAN WE MINIMIZE HYPOTENSION FOLLOWING SPINAL ANESTHESIA FOR CESAREAN SECTION?

Spinal (subarachnoid) anesthesia is considered nowadays as the "Gold standard" technique for cesarean section. The technique has a very rapid onset and provides a dense neural block. Because of the small dose of local anesthetic used, there is minimal risk of local anesthetic toxicity, and minimal transfer of the drug to the fetus[1]. An important "bonus" is the "awake" mother who can witness the wonderful moment of delivery of her newborn.

However, "there is no free dinner". Spinal anesthesia in the pregnant woman is associated with a greater incidence of hypotension than that observed in the non-pregnant. This may be attributed to a higher level of sympathetic block than that achieved by the same dose of the local anesthetic in the non-pregnant. Also, it may be related to the aortocaval compression by the gravid uterus, as well as to the already low decreased systemic vascular resistance associated with pregnancy. The spinal anesthesia-induced hypotension can result in up to 7 fold increase in the peripartum nausea and vomiting. Datta et al showed that avoiding hypotension following induction of spinal anesthesia can result in 6.6 to 7-fold reduction in peripartum emetic symptoms[2]. Supplemental oxygen, despite hypotension, also decreases emetic symptoms.

In order to decrease the incidence of spinal anesthesia-induced hypotension, many techniques have been recommended such as maintaining left uterine displacement, crystalloid or colloid prehydration, and vasopressor administration.

Because the anatomical and physiological changes during pregnancy such as aortocaval compression can result in a higher level of subarachnoid block, a smaller dose of local anesthetic is recommended, supplemented by an intrathecal narcotic such as fentanyl or morphine which can enforce analgesia without increasing the level of sympathetic block. Also, combined spinal-epidural technique (CSE) has been reported as an option for cesarean section. CSE provides a rapid onset of dense surgical anesthesia by a small dose of local spinal anesthetic which can reduce the incidence of high spinal block and hypotension, while the analgesia is supplemented by the epidural local anesthetic.

What about spinal anesthesia in the preeclamptic parturient? Preeclampsia complicates up to 8% of pregnancies. It has recently been proposed that preeclampsia is linked to endothelial dysfunction and excessive activation of coagulation. The abnormality is accompanied by high circulating levels of endothelin, increased coagulation and fibrinolysis with impaired platelet activation and platelet consumption. The biologic balance between prostacyclin and thromboxane is disturbed in favor of increased thromboxane and decreased prostacyclin resulting in generalized vasoconstriction, associated with hypertension and decreased blood volume. Such changes have suggested that neuraxial anesthesia is not recommended in the preeclamptic woman. However, a report of the National High Blood Pressure Education Program Working Group stated that neuraxial techniques can be safely administered to the parturient with preeclampsia, since recent prospective studies demonstrated a lesser degree of hypotension following neuraxial block when compared to healthy parturients undergoing cesarean section[1,3].

The question of how to prevent or treat hypotension during spinal anesthesia for cesarean section has been a central question in obstetric anesthesia. For decades, ephedrine was the drug of choice, based on the classic studies by the Shnider SM group of San Francisco in sheep that suggested deleterious effects of α-adrenergic agents such as Phenylephrine on uteroplacental blood flow[4]. However, multiple reports in the 1990, and early 21st century by Dr. Nagan Kee and his colleagues in Hong Kong have demonstrated that phenylephrine is safe and generally more effective than ephedrine at preventing maternal hypotension and the associated nausea and vomiting[5]. In addition, it has been shown that ephedrine can lead to a higher incidence of fetal acidosis than that following Phenylephrine or other pure α-adrenergic agonists. Recently, this ephedrine-induced fetal acidosis was attributed to the greater placental transfer of ephedrine than phenylephrine across the placenta, as well as to the metabolic stimulation of the fetus by the beta-adrenergic effects of ephedrine[6].

In this issue of MEJA, Professor Mebazaa and his colleagues from Tunisia[7,8] have shown in parturients undergoing cesarean section under spinal anesthesia that using 7.5 mg of isobaric bupivacaine, supplemented by 25 μg fentanyl and 100 μg morphine resulted in a lower incidence of hypotension, nausea and vomiting than that achieved when 10 mg bupivacaine was used, without any impairment of pain relief. In their second report, the authors showed that crystalloid prehydration with a rapid infusion of 20 ml/kg lactated Ringer's solution does not reduce the incidence or severity of hypotension. This may be attributed to the rapid diffusion of the crystalloid solution from the blood into the extracellular space, confirming the findings of Rout et al that crystalloid preload does not decrease the incidence of hypotension after spinal anesthesia for elective cesarean section[9]. Cohydration with a crystalloid solution[5], or prehydration by a colloid solution which stays in the circulation for a long period can be a better alternative[10].

In conclusion, spinal anesthesia-induced hypotension in the parturient undergoing cesarean section may be decreased by left uterine displacement, and by using a lower dose of the local anesthetic, supplemented by an opiate. Also, prehydration with a colloid, or cohydration with crystalloid supplemented by an infusion of an alpha-adrenergic agonist such as phenylephrine can decrease the incidence and severity of hypotension. In addition, supplemental oxygen, despite hypotension can also decrease the peripartum emetic symptoms.

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