

QUANADEX AS A COMPONENT OF ANESTHETIC MANAGEMENT FOR CESAREAN SECTION IN WOMEN WITH PREECLAMPSIA

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Background

Preeclampsia is a severe complication of pregnancy that accounts for approximately 70,000 maternal and 500,000 perinatal deaths worldwide each year. Severe forms of the disease frequently necessitate delivery by cesarean section, which remains the definitive treatment. The main anesthetic techniques include general multicomponent anesthesia with mechanical ventilation and neuraxial anesthesia. However, general anesthesia is associated with the risks of difficult intubation, a hyperdynamic hemodynamic response, and drug-induced neonatal depression, whereas neuraxial anesthesia may be accompanied by hemodynamic disturbances and insufficient postoperative analgesia.

In 2011, the European Medicines Agency authorized the use of dexmedetomidine (Quanadex), a highly selective α_2 -adrenergic receptor agonist, as a sedative agent. Dexmedetomidine has sedative, anxiolytic, and analgesic properties and can be used as an adjunct during general and regional anesthesia and for sedation in intensive care settings. Despite the available evidence, a comprehensive assessment of its effects on both the mother and newborn in preeclampsia remains insufficient.

Objective

To evaluate the effects of intravenous Quanadex administration during cesarean section under spinal anesthesia in women with preeclampsia on maternal vital physiological systems and early neonatal adaptation.

Materials and Methods

The inclusion criteria were preeclampsia, gestational age of 37–39 weeks, and ASA physical status II. Exclusion criteria included cardiovascular disease, morbid obesity, and refusal of spinal anesthesia.

All women were divided into two groups. In Group I (n=105), premedication consisted of diphenhydramine (0.2 mg/kg) and diazepam (Sibazon) 2.5 mg. Subarachnoid puncture was performed at the L3–L4 interspace using 25–26 G pencil-point needles. A standard dose of 0.5% hyperbaric Longocain Heavy solution was administered intrathecally over 2 minutes.

In Group II (n=108), Quanadex was administered intravenously at a rate of 0.6–0.8 $\mu\text{g/kg/h}$ starting at the time of premedication. Surgery was initiated 4–6 minutes after the development of all clinical signs of an adequate surgical level of spinal anesthesia.

No specific replacement of intraoperative blood loss was performed. The total volume of intraoperative infusion therapy was 10–12 mL/kg and consisted of crystalloids (Rheosorbilact and a balanced crystalloid solution). If bradycardia below 55 beats/min occurred, atropine 0.3 mg was administered intravenously.

Vital physiological functions were assessed using standard clinical parameters. Central hemodynamics were evaluated by echocardiography. Acid–base status, electrolytes, lactate, glucose, and hematocrit were measured using a UCARE6000 blood gas analyzer (Wondfo/Rain Sen Da). N-terminal pro-B-type natriuretic peptide (NT-proBNP), cystatin C, interleukin-6 (IL-6), and S100 β protein were determined using a Finecare FIA Meter III Plus, Model FS-205 (Wondfo) immunofluorescence analyzer. Sedation was assessed using the Richmond Agitation–Sedation Scale (RASS). Neonatal status was assessed using Apgar scores at 1 and 5 minutes after birth.

Results and Discussion. At baseline, the hemodynamic status of the patients was characterized by hypertensive–hypodynamic dissociation. Acid–base assessment revealed subcompensated metabolic acidosis accompanied by mild hypoxemia.

Comparative assessment of the effects of dexmedetomidine and diazepam on the level of consciousness using the RASS demonstrated that dexmedetomidine-induced sedation was characterized by easier arousability, allowing more effective interaction and communication between patients and medical personnel.

After delivery of the fetus, the level of sedation also remained moderate in Group I, whereas in Group II it was mild (RASS -2 ± 0.1), which had a favorable effect on the mother's psycho-emotional state and facilitated early interaction with the newborn.

Changes in hemodynamic parameters, NT-proBNP levels, and acid–base status demonstrated greater hemodynamic stability in Group II, accompanied by attenuation of the stress response to surgical and anesthetic interventions. At the most traumatic stage of surgery, IL-6 levels in Group I were 30.6% higher than those in Group II, and at the end of surgery they remained 28% higher.

Newborns in Group I, whose mothers received diazepam as part of premedication during spinal anesthesia, had lower Apgar scores at both 1 and 5 minutes after birth. They also demonstrated subcompensated metabolic and respiratory acidosis, increased blood lactate levels (3.8 ± 0.09 mmol/L), moderate hypoxemia, and higher S100 β concentrations. These changes were associated with greater respiratory depression and impaired early neonatal adaptation in Group I.

Conclusion

Patients receiving dexmedetomidine (Group II) were more readily arousable than those receiving diazepam and demonstrated better hemodynamic stability and more effective antinociceptive protection against surgical and anesthetic stress, without pronounced arterial hypotension and with only moderate bradycardia. The use of dexmedetomidine was not associated with significant adverse neonatal outcomes.