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CLINICAL INFORMATION

Postpartum hemorrhage and pregnancy induced hypertension during emergency lower segment cesarean section: dexmedetomidine to our rescue

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Abstract   Dexmedetomidine is a highly selective α-2 agonist which has recently revolutionized our anesthesia and intensive care practice. An obstetric patient presented for emergency cesarean delivery under general anesthesia, with pre-eclampsia and postpartum hemorrhage. In carefully selected cases with refractory hypertension and postpartum hemorrhage, dexmedetomidine can be used for improving overall patient outcome. It was beneficial in controlling both the blood pressure and uterine bleeding during cesarean section in our patient.

Hemorragia pós-parto e hipertensão induzida pela gravidez durante cesariana de emergência em segmento uterino inferior: dexmedetomidina para nosso resgate

Resumo   Dexmedetomidina é um α_2-agonista altamente seletivo que recentemente revolucionou a nossa prática de anestesia e tratamento intensivo. Uma paciente obstétrica foi admitida para cesariana de emergência sob anestesia geral, com pré-eclâmpsia e hemorragia pós-parto. Em casos cuidadosamente selecionados com hipertensão refratária e hemorragia pós-parto, dexmedetomidina pode ser usada para melhorar o resultado geral da paciente. O fármaco foi benéfico no controle tanto da pressão arterial quanto do sangramento uterino durante cesariana em nossa paciente.

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Introduction

Ever since dexmedetomidine has been approved for human use by the FDA in 1999, its indications and ramifications in anesthesia and intensive care has been increasing. Its use in obstetric anesthesia has been delayed due concerns regarding maternal and fetal effects. In view of its several advantageous effects, it has been used in cesarean sections for controlling blood pressure response to intubation and as an alternative agent for labor analgesia as an IVPCA (intravenous patient controlled analgesia). We present a case of PIH for emergency cesarean section under general anesthesia, where dexmedetomidine was used to control her refractory hypertension, uterotonicity, stabilize hemodynamics, prevention of postoperative shivering and supplement postoperative analgesia.

Case report

A 30 year old, 70 kg, primigravida presented to the obstetric emergency in labor, with uncontrolled blood pressure and non-reassuring fetal heart rate at 38 weeks period of gestation. She was a diagnosed case of pregnancy induced hypertension (PIH) on alpha-methyl-dopa therapy. She was posted for emergency cesarean section in view of persistent fetal decelerations. The patient refused any kind of neuraxial block in view of previous back surgery for lumbar slipped disk and recurrent back ache since 2 years. On preoperative evaluation, blood pressure (B.P) was 200×102 mmHg; pulse rate was 120 beats per minute, regular, normal volume; bilateral pedal edema was present with no evidence of congestive cardiac failure. Intravenous (I.V.) Magnesium sulphate therapy was started preoperatively by the obstetricians for seizure prophylaxis. There were no signs of CNS (Central Nervous System) irritation, visual defects, coagulopathy or renal dysfunction. Invasive arterial line was instituted pre-induction for beat-to-beat pressure monitoring. Due to patient refusal, standard general anesthesia with preoxygenation, rapid sequence induction and cricoids pressure was planned after aspiration prophylaxis and appropriate monitoring. To minimize hypertensive response to laryngoscopy and intubation, I.V. preservative free Lignocaine hydrochloride 2% (2 mL) and I.V. Labetolol infusion (5 mg.min^{-1}) were given. Modified Cormack and Lehane grading was 2B.

Despite all the above measures and single attempt gentle laryngoscopic intubation, the B.P was not controlled. Post intubation B.P. was 198×100 mmHg and Pulse rate 118 min, when O_2^+ N_2O^+ Isoflurane inhalational agent were started for maintenance of anesthesia. Induction delivery interval was 4 min. Immediately on cord clamping, loading dose (70 μg.h^{-1} over 10 min) of dexmedetomidine was started, followed by maintenance dose of 35 μg.h^{-1} I.V. infusion (along with syntocinon infusion). B.P and pulse rate started to stabilize and normalize.

Delivery of the baby and placenta was followed by postpartum hemorrhage (PPH). Despite meticulous attention to hemostasis, syntocinon infusion, decreasing inhalational anesthetic agent and prostadin injection, uterine bleeding was not controlled. Dexmedetomidine also has uterotonic effects, which was beneficial in controlling this PPH.

Once B.P was controlled and the uterus contracted after dexmedetomidine infusion, uterine bleeding was also controlled. The apgar scores of the baby were normal at birth, 1 and 5 min. After ensuring adequate hemostasis, followed by uterine and skin closure, preparations for reversal and extubation were made. Dexmedetomidine infusion was continued on maintenance dose. On return of spontaneous respiration and train-of- four responses on neuromuscular monitoring, reversal was given and the patient extubated when fully awake.

Patient maintained her vitals well and her postoperative B.P was 132×80 mmHg and pulse rate 88 beats/min. Pain management was done with multi-modal analgesia (intravenous paracetamol infusion 1 g, intramuscular diclofenac injection 75 mg and incision site local anesthetic infiltration) along with dexmedetomidine infusion (30 mcg.h^{-1}).

Patient was shifted to a high dependency care unit for observation and monitoring. After 2 h, dexmedetomidine infusion was slowly tapered-off and stopped after 1 h. In the meanwhile, her B.P remained within normal limits. After stopping dexmedetomidine infusion, B.P. was controlled with low dose intravenous labetolol (5 mg slow I.V. boluses over 1 min) as and when required (labetolol was required only twice in 24 h) and later with oral amlodepine (10 mg twice daily from next day). The newborn was put to breast feeding within 4 h of birth and taken care-off in the nursery. Patient was shifted to ward with normal vitals the next day (after removing arterial line) and later discharged, with oral antihypertensive therapy and advised for regular follow-up with the obstetrician.

Discussion

Dexmedetomidine hydrochloride is a highly selective α-2 receptor[1] agonist (α-1 to α-2 ratio is 1:1600) with various applications in anesthesia and intensive care. It is the S-enantiomer of medetomidine,[2] with empirical formula C13H16N2.

It is given in a loading dose[3] of 1 μg.kg^{-1}.h^{-1} over 10 min, followed by maintenance intravenous infusion of 0.2–0.7 μg.kg^{-1}.h^{-1}. It produces[4] sedation, sympatholysis, cardiovascular stability, anxiolysis, analgesia, neuroprotection and anesthesia-sparing effects. Its purported advantages[5] include minimal respiratory depression, renoprotection, quick offset and cardioprotection. The main side effects include dry mouth, nausea, hypotension and bradycardia. Its reluctant use in obstetrics is due to concerns regarding possible maternal and fetal effects.[6] Dexmedetomidine is lipophilic and its placental extraction is high. Hence its fetal transfer through the placenta is minimal. In animal experiments, dexmedetomidine has not been found to cause adverse fetal effects.[7] It has been used in obstetrics for control of B.P during cesarean section and for labor analgesia. Isolated case reports have asserted the safety of dexmedetomidine in obstetric anesthesia.[8] Our case report highlights the usefulness of dexmedetomidine in controlling refractory hypertension in patients with PIH. It also emphasizes the unmatched role of dexmedetomidine in cardiovascular stability during cesarean section under general anesthesia. One unique aspect brought to light in this case is the uterotonic[9] effect of dexmedetomidine.

The uterine bleeding got controlled only after starting dexmedetomidine infusion. PPH is a devastating complication during delivery. We did not prefer using nitroglycerine for B.P control due to its uterine relaxant effects.

Inhalational anesthetics could also confound PPH, hence Isoflurane concentration was decreased on starting dexmedetomidine infusion. Once the B.P was controlled with dexmedetomidine, the bleeders also stopped oozing. Timely control of PPH is this case could be due to the combined effects of B.P control and uterotonicity. This uterotonic effect of dexmedetomidine needs to be investigated and researched further. Blood transfusion and its attendant complications could be avoided in patients with PPH. Analgesia was also supplemented by dexmedetomidine infusion both intra- and postoperatively. Her hypertension was also under control in the postoperative period. Dexmedetomidine was used only after cord clamping, so the question of fetal effects does not arise in this case, as highlighted by the normal apgar scores. Invasive arterial and neuromuscular monitoring was used along with other standard monitoring devices in this patient. She also was observed postoperatively in an intensive care unit for 24 h and then transferred out.

Conclusions

Though it is too ambitious to routinely recommend the use of dexmedetomidine in obstetric anesthesia, its unparalled multipurpose use in sedation, cardiovascular stability, analgesia and blood pressure control cannot be ignored. We need to be cautious as there is a possibility of maternal hypotension and fetal bradycardia.

Since its placental extraction is high, its fetal effects are usually negligible. Its placental retention ratio is high (Maternal to Fetal Index/Ratio 0.77) and it also directly potentiates the amplitude and rate of uterine contractions. Its uterotonic effect and efficacy in PPH, needs to be studied further in large randomized trials before being put to tangible clinical use. In carefully selected cases with refractory hypertension and post-partum hemorrhage, dexmedetomidine can be used for improving overall patient outcome. It was beneficial in controlling both the blood pressure and uterine bleeding during cesarean section, as well as for analgesia in our patient. Such cases of patient refusal or some relative contraindication to regional anesthesia for emergency operative delivery may pose a challenge for the anaesthesiologists, especially when complicated by PIH or PPH.

Conflicts of interest

The author declares no conflicts of interest.

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