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Cesarean Section Following Ruptured Cerebral Aneurysm and Neuroresuscitation

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Perioperative management of the pregnant patient who also has intracranial hypertension can present many problems. There have been numerous reports in the literature with regard to surgical and obstetric management of such patients,[1–3,6] although many of these reports antedated the intracranial pressure-reducing modalities presently in common use. We describe the anesthetic considerations in a pregnant patient presenting for cesarean section during an ongoing acute neuroresuscitation after a ruptured cerebral aneurysm.

REPORT OF A CASE

A 27-year-old right-handed woman, gravida 5, para 3, abortion 1, 36½ weeks pregnant, suddenly suffered a severe headache followed by seizures and loss of consciousness. Her pregnancy had been normal, and she was otherwise in good health.

She had a BP of 110/70 mmHg and heart rate 130 beats/min. She was obtunded, responding to pain with left-sided semipurposeful movements. She exhibited a right seventh cranial nerve deficit, left gaze preference, right arm flexion, and right leg extension. Uterine size was consistent with 36 weeks gestation. Fetal heart rate was 140 beats/min without evidence of distress. Cranial computed tomography showed a left temporal lobar hemorrhage with considerable midline shift.

A nasotracheal intubation was performed. Hyperventilation was instituted, and dexamethasone 10 mg, phenytoin 1 g, and mannitol 12.5 g were given iv. She subsequently was taken to the operating room 3 h after admission for a cesarean section. Systolic BP at this time was 160 mmHg, and she was moving vigorously semipurposefully, requiring restraints. Succinycholine infusion and increments of thiopental totalling 250 mg were given iv during preparation for surgery with BP decreasing to 120/60 mmHg. Radial artery and central antecubital vein catheters were inserted, and she received 100 g of mannitol and 20 mg of furosemide iv. Cesarean section was performed, with the anesthesia consisting of thiopental 850 mg iv, 50% nitrous oxide in oxygen, and fentanyl 150 mcg iv with continued hyperventilation to a Pa_{CO_2} of 27 mmHg. A 2.21-kg baby girl was delivered 5–10 minutes after induction of anesthesia with 1- and 5-min Apgar scores of 6 and 8, respectively. After delivery, the mother was given an iv infusion of oxytocin.

After discontinuing N_2O and succinylcholine, the mother's neurologic examination was essentially unchanged. Subsequently, a cerebral angiogram was performed, which showed a saccular aneurysm of the left middle cerebral artery with hippocampal herniation and a large hematoma in the left temporoparietal region. The patient underwent craniotomy 6 h after admission, with evacuation of the hematoma and aneurysm clipping under thiopental–fentanyl–nitrous oxide anesthesia with paralysis induced by pancuronium. Mean arterial pressure (MAP) was controlled at 70–75 mmHg with nitroprusside and propranolol. Despite these neuroresuscitative efforts, she had profound brain swelling intraoperatively. The surgery was additionally complicated by aneurysmal rupture with an acute blood loss of about 1,300 ml, which was treated rapidly by infusion of blood. Nevertheless, she sustained a further decrease in MAP to 65 mmHg for 5–10 min.

An iv oxytocin infusion was maintained throughout the angiogram and craniotomy. A total of 2,150 mg thiopental was administered iv during the three procedures, which altogether lasted about 10 h.

The baby did well with no electrolyte abnormalities, and was discharged when 3 days old. The mother regained consciousness and is presently caring for the child with assistance, despite a residual right hemiplegia and some personality changes.

DISCUSSION

Most authors agree that the management of subarachnoid hemorrhage in pregnancy can be the same as in the nonpregnant patient.[1–4] If the patient is in stable con-

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dition, angiography usually is well tolerated[1,4] and the lesion should be treated as neurosurgically indicated. The pregnancy can be managed in the usual fashion, performing a cesarean section only for obstetric indications. If neurosurgery is refused or is not possible, the probability of the hemorrhage reoccurring is high.[1,3] If the patient survives, she can deliver vaginally, but straining should be avoided, which can be accomplished with such approaches as a continuous epidural anesthetic. Other clinicians feel that an elective cesarean section should be performed at the time when fetal viability is achieved.[1] If the patient is felt to be unsalvageable and the fetus is viable, then a cesarean section may be indicated,[4] as was judged to be the case with our patient.

Another case was described in which a patient near term had a severe subarachnoid hemorrhage necessitating cesarean section during ongoing neuroresuscitation followed by craniotomy. The anesthetic drugs and neuroresuscitative approaches used, however, were not described.[5] In our case questions arose regarding the effects of intracranial hypertension, neuroresuscitation, and neuroanesthesia on the uteroplacental blood flow and the fetus, and the effects of pregnancy on intracranial pathophysiology.

In pregnancy there are many hemodynamic alterations that may affect neuropathologic processes. Cerebral blood flow (CBF) and cerebral metabolic rate for oxygen remain unchanged, however.[6] After delivery, oxytocic drugs are used widely to decrease uterine atony and bleeding. Oxytocin is a potent vasoconstrictor in most vascular beds, although its cerebral vascular effects have not been studied. It has been used clinically in patients with vascular malformations without adverse effects.[2]

Hyperventilation was used in our case because of intracranial hypertension. Some investigators have found that marked hyperventilation causes uteroplacental vasoconstriction, resulting in fetal hypoxia and acidosis.[7] However, positive-pressure ventilation-induced decreased cardiac output, and not solely hypocapnea or alkalosis, is probably the primary cause of decreased uterine blood flow and neonatal hypoxia and depression.[7]

Several drugs were given in our case because of intracranial hypertension. These drugs include phenytoin, thiopental, lidocaine, mannitol, and furosemide, all of which cross the placenta. Their potential adverse effects on the fetus are outlined in table 1. It is noteworthy that in usual clinical doses these drugs were all well tolerated.

When our patient arrived in the OR she was somewhat hypertensive. This responded to small increments of thiopental. Had this been ineffective, we may have needed a vasoactive drug such as nitroprusside, nitroglycerin, hydralazine, propranolol, or trimethaphan. In addition, these drugs may have been needed during the craniotomy, despite the postpartum condition of her uterus. These drugs appear to have rather disparate effects on the uteroplacental unit, the adverse effects of which are summarized in table 1. Nitroprusside has been used in a patient with concomitant fetal bradycardia, ultimately producing no obvious adverse sequelae to the neonate.[16] Nitroglycerin has been noted in ewes to cross the placenta minimally, maternal administration resulting, or a fetal/maternal arterial concentration ratio of 0.04 and causing no changes in fetal heart rate or BP, despite a maternal MAP of 70 mmHg.[17] It has been used in hypertensive patients at cesarean section with no BP problems being noted in the neonate when placed in the usual head-down position.[14]

In view of the potential problems with the vasoactive drugs, hypothermia would have been a possibility in our patient had it been a less urgent situation. In gravid dogs, hypothermia has not been found to have deleterious effects on the fetus, although it can decrease UBF and cause uterine hypertonus with increased intrauterine pressure.[25] There have been many case reports in the literature demonstrating the safe use of hypothermia in pregnancy.[1,3,4,26] Fetal mortality has been noted in a few

TABLE 1. Adverse Uteroplacental Drug Effects

Drug	Adverse Effects
Phenytoin	Minimal[8]
Thiopental	Neonatal depression (>8 mg/kg; human). Worsening of preexisting fetal distress due to maternal hemodynamic effects.[9,10]
Lidocaine	Uterine hypertonus and vasoconstruction with fetal distress (toxic doses; sheep). Worsening of preexisting fetal distress.[10]
Mannitol	Oligohydramnios with fetal hyperosmolarity, hypernatremia, dehydration, cyanosis, and bradycardia (12.5 g/kg; rabbit). Fetal hyperosmolarity in humans 1 h after 200 g iv.[11,12]
Furosemide	Possibly dilation of ductus arteriosus. Electrolyte abnormalities.[12,13]
Nitroprusside	Decreased UVR.* Lethal fetal cyanide levels with onset of maternal tachyphylaxis (sheep).[14,15]
Nitroglycerin	Decreased UVR* (Sheep)[14,17]
Hydralazine	Decreased UVR* (Sheep)[18]
Propranolol	Decreased umbilical blood flow (sheep). Premature labor, worsening of preexisting fetal distress. Neonatal acidosis, bradycardia, hypoglycemia, apnea.[19-24]
Trimethapan camsylate	Unstudied

* Uterine vascular resistance.

cases with hypothermia, although there usually have been other contributing circumstances that could be implicated, such as very early gestation, hemorrhage, hypoxia, or acidosis.[26]

In summary, cesarean section during acute neuroresuscitation is an unusual clinical situation. After reviewing the literature, the following conclusions regarding interactions between the pregnancy, the intracranial process, and their management modalities emerge.

1. Oxytocic drugs probably are without deleterious neurologic effects, although they have not been studied extensively in this setting.

2. Controlled hyperventilation is usually an integral component of a neuroresuscitation and may compromise the fetus, especially if the patient is hypovolemic. Maintaining adequate intravascular volume and keeping mean airway pressure as low as possible may lessen adverse fetal effects.

3. If overdosage is avoided, phenytoin, furosemide, thiopental, and lidocaine can be well tolerated when administered with due regard for their possible adverse side effects.

4. Hypotensive agents generally can have adverse effects on the fetus, but in some case reports have been used successfully. They have not been studied in pregnant humans.

5. Mannitol has been found to have deleterious effects in rather high doses, maternal–fetal effects remaining unstudied at usual clinical doses. It was clinically well tolerated in the case we report and, if indicated, we feel should be given in as low a dose as possible, recognizing its possible adverse fetal effects.

6. Hypothermia has been shown repeatedly to be a safe modality to use during pregnancy, although it has not been studied in a controlled fashion in this setting. Because of the urgent nature of the case we presented, it would have been impractical to implement and likely would have caused hypothermia-related problems in the neonate. However, for elective cerebrovascular surgery during pregnancy it is probably a good modality to utilize in optimizing operating conditions without adversely affecting mother or fetus.

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Sudden Cardiac Arrest during Percutaneous Ultrasonic Nephrostolithotomy

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Ultrasonic percutaneous nephrostolithotomy is a relatively new procedure for the removal of renal and upper ureteral stones. Two groups have reported on the use of the procedure in a total of 32 patients.[1,2] There have been few major complications of this technique reported. The procedure uses ultrasonic energy delivered by a metal probe through a percutaneous track into the renal pelvis to fracture stones. The fragments then are flushed out with large quantities of irrigating fluid. We wish to report a cardiac arrest as a major complication of this new procedure.

REPORT OF A CASE

The patient was a healthy 41-year-old, 60-kg woman with a staghorn calculus in her right renal pelvis. She had previously had a percutaneous nephrostomy tube inserted under local anesthesia without difficulty. Laboratory values included a serum sodium of 138 mEq · l^{-1}, serum potassium of 4.5 mEq · l^{-1}, hematocrit of 39%, and total bilirubin of 0.1 mg/dl. Meperidine, 50 mg, promethazine, 25 mg, and atropine, 0.4 mg, were given im 1 h prior to surgery. Anesthesia was induced with 300 mg thiopental iv in divided doses and maintained with inspired concentrations of 60% N_2O and 0.7 to 1.0% halothane using controlled ventilation. Nondepolarizing neuromuscular blocking drugs were not used.

The case proceeded uneventfully for approximately 90 min. By this time, sterile water irrigation fluid had been in use for about 45 min and a large portion of the stone had been fractured and removed. The returning irrigating fluid had been clear. The vital signs had been stable with a systolic blood pressure of 90 mmHg, and a regular heart rate of 80 beats/min with a sinus rhythm. Blood then began returning with the irrigation fluid, and immediately a 30-s period of bradycardia unresponsive to atropine 0.8 mg iv occurred. This was followed by asystole. Closed chest cardiac massage was initiated immediately and ventilation was controlled with a $F_{I_{O_2}}$ of 1.0. Resuscitation was successful with the addition of epinephrine, 1.0 mg iv, sodium bicarbonate, 50 mEq iv, and calcium chloride, 500 mg iv, followed by electrical cardioversion. A systolic blood pressure of 120 mmHg was reestablished with a sinus tachycardia at a rate of 130 beats/min approximately 5 min after the arrest. Small amounts of frothy blood-tinged secretions were suctioned from the endotracheal tube after the arrest. A blood sample drawn during resuscitation showed a serum sodium of 121 mEq · l^{-1}, serum potassium of 4.5 mEq · l^{-1}, and a spun hematocrit of 28% (capillary tube). The serum was not examined for gross color changes. Surgery was terminated. The patient awoke in 1 h without signs of any cardiovascular or neurologic sequelae. Furosemide, 10 mg iv, was administered and the pulmonary edema resolved over the next few hours. Pertinent laboratory data 5 h postoperatively included a serum sodium of 126 mEq · l^{-1}, serum potassium of 4.4 mEq · l^{-1}, and hematocrit 26%. The following day serum sodium was 131 mEq · l^{-1}, serum potassium was 4.0 mEq · l^{-1}, total serum bilirubin 3.4 mg/dl, serum hemoglobin 600 mg/dl, and the hematocrit 32%. The patient was discharged on the third postoperative day with no evidence of impairment of renal, cardiac, or neurologic function.

DISCUSSION

There are several possible causes for the cardiac arrest. One is simple intravascular volume overload with acute heart failure. This would be unlikely to cause a sudden cardiac arrest without other premonitory signs of volume overload and so we suspect this did not occur. Another possibility would be acute hyponatremia from free water intake. Under general anesthesia this could present as a sudden cardiac arrest without earlier signs of hypervolemia. A third possibility is the sudden entrance of a bolus of hypotonic fluid with acute hemolysis and hyperkalemic cardiac arrest. This was not excluded by the normal serum potassium values 5–10 min following the arrest, since the

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