Anesthetic management of cerebral aneurysm clipping during pregnancy: A case report

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A 35-week pregnant female presented emergently for clipping of a cerebral aneurysm under general anesthesia. The patient was neurologically stable with an active fetus. Anesthetic goals for this patient included maintenance of uteroplacental perfusion, fetal well-being, and maternal well-being. Maternal monitoring consisted of invasive arterial blood pressure, central venous pressure, and urine output, in addition to the standard monitors for anesthesia. Fetal monitoring consisted of fetal heart rate by external Doppler and uterine activity by external tocometer. Anesthesia care was directed at ensuring optimal maternal and fetal well-being. The aneurysm was clipped, and the patient emerged from anesthesia without neurological deficits. No uterine activity was noted intraoperatively. Fetal heart rate was maintained between 125 and 160 beats per minute. A healthy baby was delivered 11 days postoperatively by cesarean section under regional anesthesia. The Apgar score was 8/9 at 1 and 5 minutes.

Key words: Cerebral aneurysm, neurosurgical anesthesia, pregnancy.

Introduction
A 33-year-old black female was admitted following a tonic-clonic seizure, loss of consciousness, and transient left hemiparesis associated with crack cocaine use. No visual or neurological deficits were noted, and lumbar puncture was positive for xanthochromia.

The patient was 5 feet 7 inches in height and weighed 72 kg. Her obstetrical history was pertinent for gravida 6, para 4 female, with four previous cesarean sections under regional anesthesia. She had not sought prenatal care for this pregnancy. Ultrasound examination of the abdomen indicated a 35-week intrauterine pregnancy by fetal size and fundal height. Fetal heart rate was 140 beats per minute (BPM) with good baseline variability. No uterine activity was noted.

The patient's past medical history was not significant for diabetes, hypertension, cardiac, pulmonary, or renal disease. She did not receive regular medical attention and was not taking any prescribed medications. Her social history was positive for crack cocaine abuse every 4 to 6 hours daily and heavy alcohol abuse. Her past surgical history was pertinent for four cesarean sections, as noted earlier. She was stabilized on phenytoin and steroid therapy prior to transfer to the Medical College of Pennsylvania Hospital for neurosurgical evaluation of a cerebral aneurysm.

Cerebral angiography demonstrated a 1.0 cm by 1.0 cm internal carotid artery saccular aneurysm with subarachnoid hemorrhage. The patient demonstrated no neurologic deficits. Laboratory values on admission were as follows: room air blood gas pH 7.44, Paco2 34.4 mmHg, Pao2 104.4 mmHg, and O2 saturation 99%. Electrolytes were within normal

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limits. Hemoglobin/hematocrit were 8.8 gm/dL and 27.4%. Prothrombin time/partial thromboplastin time were 12.7/30.0 seconds. Platelet count was 276K. The phenytoin (Dilantin®) level was slightly subtherapeutic.

Case management

The patient presented to the operating room as an ASA physical status IIIIE for microscopic clipping of a cerebral aneurysm under general anesthesia and was positioned in the supine position with left uterine displacement. Peripheral intravenous access was obtained in both upper extremities. An 8.5 Fr cordis was introduced through the right internal jugular vein and was used for central venous pressure monitoring intraoperatively. A 20-gauge right radial arterial line was placed to continuously monitor mean arterial pressure (MAP) and blood gas values intraoperatively. The fetal heart tones and uterine activity were monitored intraoperatively with an external Doppler and tocometer. Baseline fetal heart tones were 140 BPM with baseline variability. The uterus was quiet.

The patient was preoxygenated with 100% oxygen for 5 minutes prior to induction of anesthesia. Curare 3 mg was used for defasciculation. Rapid sequence induction was accomplished using alfentanil 35 µg/kg (2,500 µg), lidocaine 1.5 mg/kg (100 mg), thiopental 4 mg/kg (275 mg), and succinylcholine 1.5 mg/kg (100 mg). The trachea was intubated with a 7.0mm oral endotracheal tube under direct vision of the vocal cords. Induction of anesthesia was smooth with no significant increase in MAP. Preinduction blood pressure was 140/80; postinduction blood pressure was 110/60.

Anesthesia was maintained with 0.2-1.0% isoflurane in oxygen and nitrous oxide 1:1. In addition, fentanyl 5 µg/kg (375 µg) was administered. Neuromuscular blockade was achieved with vecuronium after recovery from succinylcholine. A 1,000-µg bolus of alfentanil was given approximately 1-2 minutes prior to placement of the skull clamp pinions. The patient remained hemodynamically stable for pinion placement and skin incision. MAP was closely monitored throughout the procedure and ranged from 70-85 mmHg. Transient increases of MAP greater than 85 mmHg were managed by increasing anesthetic depth and 10-20 mg doses of esmolol.

Fluid therapy consisted of lactated Ringer's solution at 1.5 mL/kg/hr by peripheral intravenous sites. The central venous pressure ranged from 7-11 mmHg. Mannitol 25 gm and furosemide 20 mg were administered 3.5 hours after skin incision. Urine output ranged from 50-350 mL every 30 minutes after diuretic therapy. No uterine activity was noted.

Controlled mechanical ventilation was managed to maintain PaCO2 in the low- to mid-30s. Continuous oxygen saturation by pulse oximeter was 98-99% throughout the procedure.

Fetal heart tones were monitored and recorded continuously for the duration of the procedure. Following induction of anesthesia, the fetal heart rate decreased to approximately 125 BPM. Baseline variability continued until 1.5 hours into the procedure. At that time variability was lost; however, fetal heart rate remained at 125 BPM. Beat-to-beat variability gradually returned toward the end of the case until the fetal heart tones were 140-160 BPM at emergence. No fetal heart tones under 125 BPM were noted. No uterine activity was noted on the tocometer during the procedure.

Following aneurysm clipping, volume expansion was carefully started using 500 mL lactated Ringer's solution and 250 mL Plasmanate®. Blood pressure was allowed to rise to the 140s/80s. One unit of packed red blood cells was given for a hemoglobin of 7.6 gm/dL. Estimated blood loss was 600 mL.

After the head dressing was applied, neuromuscular blockade was reversed with neostigmine 3 mg and glycopyrrolate 0.6 mg. Anesthetic gases were discontinued. Mean arterial blood pressure was controlled on emergence, using bolus doses of esmolol 10 mg (0.15 mg/kg) and labetalol 10 mg (0.15 mg/kg). No uterine activity was noted. The patient gradually responded and was able to move all extremities on command.

The trachea was extubated when active gag reflex and adequate spontaneous respiration returned. MAP ranged from 80-85 mmHg during emergence. The patient was transported to the recovery unit in stable condition with left uterine displacement. Fetal heart rate was 153 BPM with baseline variability; the uterus was soft and quiet.

Discussion

The incidence of cerebral aneurysm and subarachnoid hemorrhage in the general population is estimated to be 1-5%.1 Aneurysm size varies from 1-5 cm in diameter, with the most frequent location at bifurcations of arteries of the circle of Willis.2 Estimates of the incidence of subarachnoid hemorrhage during pregnancy vary from 1 in 2,000 to 1 in 10,000 pregnancies.3 Increased plasma volume, hypertension, and increased cardiac output contribute to rupture during pregnancy.4 Rupture is most common in the late second or third trimester. Management of delivery of the fetus in a cerebral aneurysm patient varies from controlled forceps vagi-
nal delivery to cesarean delivery under regional or general anesthesia. Some clinicians advocate a combined procedure of cesarean delivery followed by aneurysm clipping.

In this case study, aneurysm rupture was associated with crack cocaine use. Inhibition of norepinephrine reuptake at the adrenergic neuron leads to hypertension and tachycardia. In the pregnant patient, these signs can be confused with the onset of pregnancy-induced hypertension. Fetal effects from direct placental transfer are related to a decrease in placental blood flow by as much as 40%. Fetal hypoxia, hypertension, and tachycardia are the result. Crack cocaine use during pregnancy is also associated with an increased incidence of placental abruption and preterm labor.

Chronic cocaine use depletes norepinephrine levels, whereas acute cocaine use is associated with increased levels. The chronic abuser presenting for surgery will exhibit a decreased anesthetic requirement. Myocardial effects of cocaine use range from dysrhythmias to ischemia. Verapamil is the treatment of choice for tachyarrhythmias and hypertension in the acutely intoxicated patient. Treatment of these acute symptoms with beta blocker therapy may lead to unopposed alpha-adrenergic stimulation and further increase blood pressure.

Clinically, subarachnoid hemorrhage may present as a sudden onset of severe throbbing headache, hypertension, blurred vision, proteinuria, seizure activity, loss of consciousness, and hemiparesis. In the pregnant patient, the differential diagnosis should include eclampsia. With subarachnoid hemorrhage, lumbar puncture will be positive for grossly bloody cerebrospinal fluid or xanthochromia. Definitive diagnosis and documentation of cerebral aneurysm is obtained by cerebral angiography and a computed axial tomographic scan of the cranium. With appropriate management, neither of these two procedures should be detrimental to the fetus or the mother. Medical management following subarachnoid hemorrhage includes bed rest, control of blood pressure to prevent rebleeding, prevention/treatment of cerebral vasospasm, anticonvulsants, and steroids to decrease cerebral edema.

Anesthetic management of the pregnant neurovascular patient focuses on maintenance of both maternal and fetal well-being. Preoperative medication should allay anxiety without excessive sedation, which might interfere with frequent neurological assessment. A rising PaCO₂ from sedation also causes cerebral vasodilatation. The usual monitors for neurovascular surgery are required, including standard noninvasive anesthesia monitors and an arterial line for close monitoring of blood pressure. A central venous pressure may be helpful in evaluating volume status.

In addition, fetal heart tones are monitored by external Doppler probe. An external tocometer is utilized to monitor uterine activity. Monitoring of both fetal heart tones and uterine activity should continue until at least 24 hours postoperatively. Left uterine displacement will prevent aortocaval compression, with subsequent decreased cardiac output and decreased uteroplacental perfusion.

Induction of anesthesia for a neurovascular procedure focuses on maintenance of a stable transmural pressure gradient across the cerebral aneurysm wall. Rising MAP or decreasing intracranial pressure serves to increase the gradient and presents the risk of aneurysm rupture. Perioperative aneurysm rupture that requires emergency clamping of a major vessel for control carries a 50% mortality rate.

Instrumentation and intubation of the airway are associated with increased blood pressure and a subsequent widening of the transmural pressure gradient. Provision for adequate anesthetic depth prior to intubation will prevent precipitous rises in blood pressure. Decreasing intracranial pressure associated with hyperventilation will also serve to increase the gradient.

The pregnant neurovascular patient complicates the induction by requiring a rapid sequence induction, secondary to the risk of aspiration associated with pregnancy. Adequate depth of anesthesia must be achieved rapidly to attenuate the blood pressure response to intubation. At the same time, hypotension must be avoided to preserve adequate placental perfusion to ensure fetal well-being.

In this case presentation, a combination of short-acting narcotics and barbiturates were used to produce a rapid, deep induction prior to laryngoscopy. MAP did not rise; however, a transient fall in MAP was noted.

Fetal heart tones were constant at the baseline rate throughout induction, indicating a good uteroplacental flow. The short action of alfentanil was advantageous to maintain MAP during skin preparation and draping, a period of little stimulation to the patient. Alfentanil was again administered prior to placement of skull clamp pinions with satisfactory results.

Alternative techniques for management of hypertension associated with laryngoscopy range from judicious doses of thiopental on induction to infusion of sodium nitroprusside. Schnider and Levinson advocate a combination of lidocaine and large doses of thiopental to obtund the hypertensive response. For pregnant patients with preexisting uncontrolled hypertension, they suggest fentanyl.
1-3 μg/kg administered with preoxygenation by mask during arterial line placement with careful monitoring of ventilation to prevent increased PaCO₂. Blood pressure is then decreased by 15-25% with a controlled sodium nitroprusside infusion prior to anesthesia induction.

Following tracheal intubation, the sodium nitroprusside can be discontinued and additional fentanyl given to deepen the anesthetic. Whittburn and colleagues report use of labetalol 15 mg to reduce systolic blood pressure by 30 mmHg prior to rapid sequence induction.³

Mild to moderate hyperventilation is indicated for intracranial aneurysm surgery. The goal for ventilation of this pregnant patient was to maintain maternal PaCO₂ in the low- to mid-30s. Maternal hyperventilation and reduction of PaCO₂ to levels below normal will cause constriction of blood supply to the uterus and fetal hypoxia.⁴ Alkalosis produces a leftward shift of the oxyhemoglobin curve, unfavorable to the release of oxygen to the placental/fetal circulation.³⁵ An increase of maternal PaO₂ to 600 mmHg increases fetal PaO₂ to about 45-60 mmHg, posing no threat of in utero closure of the ductus arteriosus or retrolental fibroplasia.⁴

Preoperatively, the decision was made in consultation with the neurosurgeons to avoid controlled hypotension. In recognition of the direct relationship between uterine perfusion and MAP, the goal was to maintain MAP above 60 mmHg. Compromised uteroplacental circulation would have been manifest as fetal bradycardia. Significant elevations of MAP were treated with increased depth of anesthesia and labetalol or esmolol in small doses.

Increased uterine tone and fetal bradycardia are complications of beta-adrenergic blocker therapy. James and associates note that, in small doses, these blockers do not seem to pose a threat. For example, beta-1 selective blockers, such as esmolol, should not produce these adverse effects.⁷ Labetalol in doses up to 1 mg/kg, as treatment for pregnancy-induced hypertension, does not compromise placental blood flow.¹⁰ The tocotometer revealed no uterine activity, and fetal heart rate was maintained near 125 BPM.

Had the neurosurgeons requested controlled hypotension intraoperatively, nitroglycerine, which is nontoxic to the fetus,¹¹ would have been the choice for decreasing MAP. Sodium nitroprusside has been used successfully in some instances; however, it readily crosses the placenta. Metabolized to cyanogen, it is potentially toxic to the fetus, since the fetus has less thiosulfate substrate for metabolism.¹² Use of sodium nitroprusside should be limited to small doses and a short duration to avoid this potential complication. Trimethaphan produces venous pooling and decreased cardiac output, further compromising uterine circulation.¹³

If controlled hypotension is used, fetal heart rate should be monitored closely for signs of compromised placental perfusion. Should fetal bradycardia become apparent, MAP should be increased and 100% oxygen administered to the mother in an attempt to improve fetal oxygenation.

Osmotic diuretics cross the placenta easily, raising the fetal as well as the maternal colloidal osmotic pressure. The gradient favors the maternal circulation so that fetal dehydration, hyperosmolality, acidosis, and oligohydramnios can result.¹³ These complications can be avoided if doses of mannitol are limited to 1 gm/kg. Severe maternal dehydration is also associated with precipitation of labor. In this case study, mannitol was limited to 0.3 gm/kg to reduce cerebral edema. Urinary output was brisk, and the central venous pressure remained stable. Uterine and fetal monitors showed no change. Following aneurysm clipping, volume expansion was accomplished using a combination of colloid, crystalloid, and blood products.

During anesthesia, baseline variability of the fetal heart rate was lost, probably reflecting the anesthetized state of the fetus. No fetal heart rates under 120 BPM were observed. Variability gradually returned as the anesthetic depth was decreased toward the conclusion of the procedure. Fetal monitoring continued postoperatively for 24 hours. The fetal heart rate was stable with baseline variability. No uterine activity was noted on the tocotometer during anesthesia and recovery. Twelve hours postoperatively, contractions occurred which required no intervention.

The patient was awake, alert, and oriented without neurological deficits following surgery. She delivered a healthy infant 11 days after the neurosurgery (intrauterine pregnancy 36 weeks) by cesarean section under regional anesthesia following the spontaneous start of labor. The Apgar score was 8/9 at 1 and 5 minutes.

Conclusion

The pregnant neurosurgical patient presents a unique case for anesthetic management. Anesthesia care is aimed at ensuring both maternal and fetal well-being through management of hemodynamics, ventilation, and intravascular volume. The usual neurovascular anesthesia goals of prevention of hypertension and cerebral edema should be modified to include maintenance of adequate perfusion of the placenta and prevention of fetal hypoxia.

REFERENCES

AUTHORS

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