Management of pregnant female with meningioma for craniotomy

Sandeep Sahu, Indu Lata1, Devendra Gupta

Departments of Anaesthesiology and Obstetric and Gynaecology (General Hospital), Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, UP, India

ABSTRACT

Intracranial meningioma during pregnancy challenges the skill of obstetricians, neurosurgeons and neuroanesthesiologists in resection of the tumor and to secure delivery of the baby. Advances in fetal and maternal monitoring, neuroanesthesia, and microsurgical techniques allow safe neurosurgical management of these patients. Urgent neurosurgical intervention is reserved for the management of malignancies, active hydrocephalus, and benign brain tumors associated with signs of impending herniation or progressive neurological deficit. Particular attention is given to maintain stable maternal hemodynamics to avoid uterine hypoperfusion and fetal hypoxia intraoperatively. Therefore, the major challenge of neuroanesthesia during pregnancy is to provide an appropriate balance between competing, and even contradictory, clinical goals of neuroanesthesiology and obstetric practice.

Key words: Craniotomy, complications, meningioma, pregnancy

DOI: 10.4103/0976-3147.63101

Introduction

The management of a pregnant patient with neurological disease is particularly challenging because the usual goals of obstetric anesthesia may need modification to simultaneously optimize the neurological milieu interior. However, symptoms of raised intracranial pressure (ICP) may flare during pregnancy and this has been attributed to water retention, engorgement of vessels, and the presence of sex hormone receptors on tumor cells leading to explosive growth of the tumor. The clinical presentation of raised ICP, headache, vomiting, or seizures could be misdiagnosed as hyperemesis gravidarium during early pregnancy or as eclampsia during late pregnancy.[1] An abnormal fundoscopic examination, visual impairment, focal seizures, and lateralizing neurological deficits support the diagnosis of an intracranial mass lesion. It should be, promptly, further investigated with magnetic resonance imaging (MRI) to confirm the diagnosis.[2]

Case Report

A primigravida female of 25 years age, 30 weeks pregnant, presented with headache, vomiting for one month, with two episodes of seizures in the last week. MRI brain showed right frontal partial meningoma. As she was into 30 weeks pregnancy with features of raised ICP, not manageable with drugs, elective craniotomy was planned for decompression or excision of the right frontal partial meningoma. Preanesthetic checkup and proper maternal and fetal well being assessment was normal. Ultra sonography (USG) showed 30 weeks single intrauterine live pregnancy with cephalic presentation. Her routine hematological and biochemical parameters were within normal limit. Treatment with dexamethasone (4 mg BD), Phenytoin (BD), Ranitidine, isoxsuprine was started to decrease intracranial pressure (ICP), prevent seizure and premature termination of pregnancy respectively.

After proper counseling and reassurance, the patient was shifted (on stature in) to the operation theater (OT). All monitoring gazettes and compressive stockings in lower limbs were attached. Intravenous access was secured after injecting local xilocaine 2%. During administration of anesthesia, consideration regarding full stomach was kept in mind, so, tablet metaclopramide; ranitidine was given to decrease gastric volume and acidity at 10 pm and at 6 am. Preoxygenation was done for five minutes with 100% O2. Rapid sequence, smooth induction was done with injection thiopentone 5 mg \ kg, fentanyl 2 mcg \ kg, midazolam 2 mg and lidocaine 75 mg (to prevent intubation response). Intubation was done with 7.5 mm
cuffed ETT after injecting rocuronium 0.9 mg/ kg while maintaining cricoid pressure.

For maintenance, balanced general anesthesia technique was used with air and O₂, isoflurane 0.5-1%, fentanyl, and injection vecuronium. Injection isosuprime infusion drip was started intraoperatively and continued for 24 hours postoperatively. Care was taken to avoid all the factors that could precipitate premature labor, like hypoxia, uterine relaxant (halothane), pain (good depth of anesthesia was maintained with BIS values 40-60, by use of opioid analgesic and isoflurane). Injection mannitol 0.25 mg/kg was started just after scalp incision and before reaching durra.

ETCO₂ was kept between 28-30 mm Hg, avoiding any hyperventilation. Fluid used was intraoperatively iso-osmolar, non-dextrose containing 0.9 normal saline and tetrastarch in 1:2 ratios. Intraoperatively, blood sugar was maintained in euglycemia. Only 600 ml blood loss occurred during surgery, which was acceptable (ABG done baseline and then every two hours intraoperatively to assess pH, blood gases, electrolytes and hematocrit). Total operative time was four hours. Reversal was done with injection neostigmine 2.5 mg with glycopyrrolate 0.4 mg given slowly to avoid acute increase in acetylcholine, which might stimulate uterine contraction. Injection Ondensetron was given and diclofenac suppositories put before the end of surgery.

Extubation was done smoothly with all the precaution to prevent response after giving lidocaine, 75 mg, and fentanyl 25 mg. The patient was then shifted to neuro-intensive care unit (ICU) for monitoring. O₂ and fentanyl 25 mg. The patient was then shifted to neuro-intensive care unit (ICU) for monitoring. O₂ and fentanyl 25 mg. The patient was then shifted to

Discussion

Management of obstetric patients with brain tumors is complex and requires knowledge of the physiological effects of pregnancy on tumor size, effect and labor on maternal cerebral circulation, auto regulation and cerebral perfusion pressure to get optimal outcome.[3] On review, the literature is generally not helpful with respect to evidence-based neuroanesthetic management for the pregnant patient, and so planning and decision-making need to be based largely on the general principles of neurosurgical and obstetric anesthesia. Primary central nervous system tumors occur in approximately six in 100,000 females,[4] but are not more frequent during pregnancy. Symptoms may present or be exacerbated because of increased tumor growth or edema, increased vascularity or pregnancy-related immunotolerance.[9] Meningioma is the most common primary intracranial neoplasm and some of these tumors grow faster during pregnancy because they contain estrogen and progesterone receptors.[6]

The local effect of brain tumor in pregnancy is loss of function (focal deficits) and seizure. The general effects of tumors are increased intracranial pressure, caused by a) the mass of tumor added to the brain, b) hydrocephalus due to obstruction of CSF circulation and c) cerebral edema, which are life-threatening complications and may cause displacement (herniation) and compression of brain structures with lethal effects. Recent studies show that altered cerebral circulation, as reported during normal pregnancy, can be associated with changes in cerebral vascular reactivity and/or cerebral auto regulation. On transcranial Doppler examination, mean middle cerebral artery blood flow velocity (MCAFV) was lower and increased MCA estimated cerebral perfusion pressure (eCPP) by 52% between 12 and 40 weeks of gestation.[7] Cerebral artery reactivity changes during pregnancy and the postpartum period, predispose the cerebral circulation to forced dilatation of intrinsic myogenic tone of cerebral arteries and arterioles, decreased cerebrovascular resistance, and hyper perfusion, at lower pressures, a response that may lower cerebrovascular resistance and promote hyper perfusion when blood pressure is elevated.[8] Pregnancy also lowers the pressure of auto regulatory breakthrough and enhancing cerebral edema formation, because nitric oxide (NO) production is increased in pregnancy.[9] There was no difference in blood-brain barrier permeability between non-pregnant and late-pregnant in response to acute hypertension, suggesting that pregnancy may predispose the brain to edema through increased hydraulic conductivity.[9] These effects on cerebral circulation get exaggerated in the presence of tumor, making brain more susceptible to ischemia if cerebral perfusion pressure (CPP) decreased and breakthrough if CPP increased above limit producing edema; hence
cerebral perfusion pressure, and not cerebral blood flow, may be the critical determinant of intracranial injury in pregnant female.\(^{[10]}\) We successfully manage this case on the basis of the above principles.

When surgery is indicated, attention should be paid to fetal perfusion. The fetus may be compromised indirectly by maternal hypotension, uterine artery vasoconstriction, maternal hypoxemia, and acid-base changes, indeed any change in maternal physiology that reduces uteroplacental perfusion or compromises fetal gas exchange.\(^{[11]}\) To preserve both cerebral and uteroplacental perfusion, maintaining hemodynamic stability is important, which can be achieved through appropriate fluid administration, avoidance of aorto-caval compression, the prophylactic or early use of vasopressors drugs, and invasive arterial blood pressure (BP) monitoring. To reduce fluctuations in ICP and cerebral blood flow secondary to the intubation-induced hypertensive response or anesthesia-induced hypotension, a smooth rapid sequence induction with pharmacological ablation of the response to laryngoscopy is required.

Patient with SOL of brain is treated with steroid as it reduces peritumoral edema.\(^{[13]}\) The risk of a seizure with the attendant potential for maternal and fetal hypoxia and acidosis far outweighs the risk associated with anticonvulsants use.\(^{[14]}\) So we continued both steroid and anticonvulsant. The likelihood of premature labor and delivery following a neurosurgical procedure is less than with an intra-abdominal procedure. We preferred to use isoflurane to decrease uterine contractility during surgery. Moreover, use of an isoflurane - fentanyl combination provided stable hemodynamic with adequate arterial blood pressure to avoid uterine hypoperfusion and fetal hypoxia. These are also favored in neuroanaesthesia because they reduce cerebral metabolic rate, have the least effect on ICP, and provide a level of cerebral protection and auto regulation in animals.\(^{[15]}\)

We followed the basic neuroanaesthesia principles and the effects of the physiological changes of pregnancy on the cerebral circulation including: Avoidance of further neurological injury secondary to re bleed, cerebral ischemia, raised ICP and or metabolic complications.

Maternal modest (28-30 mm Hg) hyperventilation can facilitate surgical exposure by decreasing cerebral blood volume. Severe hypocarbia may impair fetal oxygen delivery, however, by shifting the maternal oxygen-hemoglobin dissociation curve to the left. Use of hyperventilation should, hence, be judicious and limited in extent and duration with proper fetal monitoring.\(^{[16]}\)

N\(_2\)O due to vasoconstructive property should be avoided, showing weak teratogenic effects in rodents. We use mannitol 0.5 to 1 g / kg to decrease brain bulk and facilitate exposure during craniotomy; there is no evidence to suggest that it has any significant adverse effect on maternal and fetal fluid balance.\(^{[17]}\) Continuous monitoring of both mother and fetus is essential. Fetal heart rate monitoring is believed to be useful for identifying intraoperative conditions leading to impaired uteroplacental blood flow and fetal oxygenation. Neurosurgery during pregnancy mandates a multidisciplinary approach, modification of neuroanaesthetic techniques and obstetric practices to accommodate the safety requirements of the mother and fetus.

References


Source of Support: Nil. Conflict of Interest: None declared.