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REVIEW ARTICLE

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Anesthetic management of labor and delivery in patients with elevated intracranial pressure

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ABSTRACT
The anesthetic management of labor and delivery in patients with elevated intracranial pressure is complex. This review discusses the etiologies of diffuse and focal pathologies which lead to elevated intracranial pressure in pregnancy. The role of neuraxial and general anesthesia in the management of labor and delivery is also examined. Finally, a comprehensive review of strategies to minimize increases in intracranial pressure during general anesthesia for cesarean delivery is presented.

Keywords: Intracranial pressure; Anesthesia; Analgesia; Pregnancy

Introduction

The anesthetic management of labor and delivery parturients with elevated intracranial pressure (ICP) is complex and controversial. Studies of obstetric patients with intracranial hypertension are rare, thus many principles that guide management are extrapolated from neurosurgical literature. Of necessity, clinical decisions are often made based on known principles of neurologic and obstetric physiology and case reports, rather than randomized clinical studies. Hemodynamic management of these patients is critical. Cerebral perfusion pressure (CPP) is defined as mean arterial pressure (MAP) minus ICP. To maintain adequate CPP and brain tissue oxygenation, acute increases in ICP or decreases in MAP must be avoided. While the primary anesthetic goal is prevention of further elevation of ICP, anesthetic management of labor and delivery can vary widely based on the etiology of increased ICP. This review discusses physiologic changes in pregnancy that may impact ICP as well as the etiology of focal and diffuse lesions that increase ICP (Fig. 1). The role of neuraxial and general anesthesia for labor and delivery in the presence of neurologic diseases that may alter ICP is also reviewed.

Dynamics of intracranial pressure

The physiologic principles described by the Monro-Kellie doctrine are critical to understanding elevations in ICP that occur with intracranial lesions. The Monro-Kellie doctrine summarizes the dynamics of ICP in the setting of pathologic intracranial processes and explains how ICP increases occur in a relatively predictable fashion, based on the volume of intracranial contents. Normal structures within the cranium can be divided into the brain, blood, and cerebrospinal fluid (CSF). Because the intracranial compartment and vertebral canal form a relatively fixed volume structure, any increase in the volume of one component or addition of a new pathologic structure requires a compensatory decrease in the volume of other components. Of the three normal structures, the brain is the least compressible, so the blood and CSF compartments offer the greatest degree of compliance. This intracranial compliance allows for ICP to increase marginally with small increases in the volume of intracranial components. Additional compliance is afforded as the brain shifts and eventually herniates across dural compartments. Once these compensatory mechanisms fail, ICP rises exponentially and may eventually compromise CPP, leading to further neuronal injury.

Physiological changes during pregnancy and relationship to intracranial pressure

The physiologic changes of pregnancy have the potential to alter ICP. Plasma osmolality and albumin concentration decrease, while blood volume and cardiac output increase (35% and 40–50%, respectively). These changes, in conjunction with sodium and free water retention, make the pregnant patient susceptible to cerebral edema.[1] The increase in cardiac output, combined with

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estrogen-mediated vasodilatation, results in a progressive increase in cerebral blood flow (CBF) which reaches a peak of 20% above non-pregnant values in the third trimester.[2] Other changes which might have an impact are the fluctuation in brain volume, which decreases intrapartum and increases postpartum, and the increase in size of the pituitary gland.[3,4] Despite the increased susceptibility to cerebral edema, CSF pressure is unaltered in normal pregnancy (normal ICP 7–15 cmH_2O; upper normal limit 20–25 cmH_2O).[5] However, CSF pressure can increase during the first and second stages of labor to 39 cmH_2O and 71 cmH_2O, respectively.[5] Epidural blood vessels become engorged during the third trimester of pregnancy, resulting in a reduction in both CSF volume and dural sac surface area.[6] This may explain the increased sensory level achieved by neuraxial anesthesia in pregnancy.[6]

Focal etiologies of increased intracranial pressure

Intracranial masses

Tumors

Although intracranial neoplasms are very rare during pregnancy, space-occupying lesions can cause pathologic elevations in ICP. Tumors increase ICP by occupying that part of the intracranial compartment that would normally accommodate non-pathologic structures. Tumor progression eventually overcomes intracranial compliance mechanisms, leading to pathologic increases in ICP. The incidence of intracranial neoplasms in pregnancy is not known; however, the incidence in women of child-bearing age is estimated at 3.4–13.2 per 100 000.[7] The frequency of intracranial neoplasms is not affected by pregnancy, with the possible exception of choriocarcinoma.[7] Although the incidence may not increase during pregnancy, physiologic changes can lead to significant tumor growth and symptomatology.

Intraparenchymal hemorrhage/cerebral infarction

Intracranial hemorrhage (ICH) is uncommon during pregnancy. It can, however, be a devastating complication, accounting for 7.1% of total pregnancy related mortality with an in-hospital mortality rate of 20.3%.[8] A USA nationwide database of pregnancy-related admissions found that subarachnoid hemorrhage (SAH) occurred in 5.8 per 100 000 deliveries over a 13-year period and accounted for 4.1% of maternity-related deaths.[9] Like intracranial tumors, SAH increases ICP by occupying that part of the intracranial compartment that would normally accommodate non-pathologic structures. The risk of rupture of an aneurysm or arterial-venous malformation (AVM) may be increased during pregnancy secondary to increased cardiac output, increased blood volume, and hormonally-mediated vascular connective tissue changes.[10] The risk of ICH during pregnancy is highest in the postpartum period. Known risk factors include, advanced maternal age, African-American race, Hispanic ethnicity, intracranial venous thrombosis, drug and alcohol abuse, pre-existing hypertension (especially if superimposed with preeclampsia), coagulopathy, and tobacco use.[8,9]

Subdurallepidural hematoma

Most reported subdural hematomas in obstetrics occur postpartum with trauma.[11] However, antepartum atraumatic subdural hematoma has been reported in patients with preeclampsia.[11] A case of subdural hematoma as the initial presenting symptom of metastatic choriocarcinoma has been reported.[12] Choriocarcinoma should be ruled out in any woman of reproductive age presenting with intracranial hemorrhage of unknown origin.[12] Radiographic imaging often fails to demonstrate choriocarcinoma lesions. Diagnosis is made through CSF β-human chorionic gonadotropin levels and histologic examination.

Brain abscess

Though rarely associated with pregnancy, brain abscesses are a potentially life threatening condition. The abscess occupies intracranial space normally filled by non-pathologic tissue, and ICP rises when intracranial compliance limits are exceeded. Risk factors include pre-existing infection such as sinusitis, otitis media, mastoiditis, dental and scalp infections, foreign body and immunosuppression. A small review of pregnancies complicated by brain abscess demonstrated that up to 30% of patients had no risk factors.[13] Presenting symptoms are often non-specific indicators of increased ICP such as headache, mental status changes, and seizures. Diagnosis is made via radiologic imaging combined with surgical aspiration and culture. Treatment varies with the size, etiology and severity of the lesion, but typically includes antibiotic therapy and, potentially, surgical intervention. Term vaginal delivery appears safe in the absence of other maternal or fetal indications for cesarean delivery.[13]

Non-communicating hydrocephalus

Hydrocephalus is a condition characterized by excess CSF in the cerebral ventricles leading to elevated ICP. Non-communicating hydrocephalus (NCH) is caused by physical obstruction of CSF from the choroid plexus to the subarachnoid space. Non-communicating hydrocephalus (NCH) increases ICP via expansion of the CSF compartments upstream from the CSF flow obstruction. As these compartments accumulate CSF and expand in size, the additional volume of CSF in the fixed space increases ICP. Obstruction can be caused by external compression or intraventricular masses, and can occur at multiple anatomic areas. Obstruction at the foramen of Monro can be unilateral or bilateral and leads to