Imaging Intracranial Pressure

An Introduction to Ultrasonography of the Optic Nerve Sheath

In clinical practice it is important to determine whether increased intracranial pressure is present. This allows one to alter hemodynamic management, predict the potential for neurologic deterioration, and guide therapeutic removal of cerebrospinal fluid or avoid such withdrawal. A spinal leak or drainage of cerebrospinal fluid can create an imbalance between supratentorial and infratentorial pressures that could lead to catastrophic herniation of the brainstem. Spinal anesthesia and unintended dural puncture with epidural placement have both been associated with acute herniation of the brainstem in patients with unexpectedly increased intracranial pressure.1,2 In obstetrical anesthesia, we commonly encounter patients who are at risk for increased intracranial pressure but for whom a spinal or epidural anesthetic is medically indicated. In this issue of ANESTHESIOLOGY, Dubost et al. introduce a noninvasive technology, ocular ultrasonography, that can be used to detect and monitor increased intracranial pressure in preeclampsia.3 Optic nerve sheath diameter has been shown in several clinical trials to be highly predictive of intracranial pressure in the settings of trauma, hemorrhage, and hydrocephalus. This technique has the potential to improve the clinical care of preeclamptic patients.

Preeclampsia is a multisystem disease unique to human pregnancy that affects every organ system, including the brain. Although advances have been made in understanding disease pathophysiology, identification of preeclamptic women who will progress to severe disease and/or eclampsia remains a diagnostic challenge. In 10–15% of patients who progress to eclampsia, brain pathology is not mirrored by systemic signs. Hypertension is absent or modest and/or proteinuria not detected. Progression to eclampsia is associated with increased risk of cerebrovascular accident, aspiration, cardiac arrest, and death. In addition, eclampsia is thought to have long-term neurologic consequences, including the increased risk of subcortical white matter lesions and impaired neurocognitive functioning.4,5 Although the mechanism is poorly understood, increased intracranial pressure can be a complication of preeclampsia and likely is associated with progression to eclampsia. The rate of change in intracranial pressure in the setting of preeclampsia is not known, but eclampsia has been associated with increased perfusion pressure (as measured by transcranial Doppler), which resolved promptly after treatment with magnesium.6

Although we commonly worry about increased intracranial pressure, it can be difficult to detect. Symptoms such as hypertension and nausea associated with increased intracranial pressure can be caused by preeclampsia itself and thus are nonspecific. Yet knowledge of the presence of increased intracranial pressure could drastically modify a clinician’s approach by affecting choice of technique, need for additional monitoring, hemodynamic goals, the use of magnesium for seizure prophylaxis, and timing of delivery. The presence of an increased intracranial pressure would make dural puncture less desirable and epidural or general anesthesia preferable.

To determine whether the intracranial pressure was increased, the authors used a 7.5-MHz linear ultrasound probe (available on many ultrasound machines used for vascular access and regional anesthesia) to measure the diameter of the optic nerve sheath 3 mm behind the globe. Five of 26 preeclamptic subjects had an increased optic nerve sheath diameter, with the mean measurement greater than 5.8 mm on the

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day of delivery. This threshold diameter is associated with 95% sensitivity for increased intracranial pressure. No patients without preeclampsia had enlarged optic nerve diameters. This technique may prove to be a noninvasive method for reliably assessing intracranial pressure that is more tractable than a retinal examination for papilledema to the anesthesiologist. This technique has been described as rapidly learned, and one author suggested that only 25 scans are required for a novice sonographer to become skilled in its use. Certainly, most anesthesiologists are more comfortable using an ultrasound machine than an ophthalmoscope.

One important question is whether the increased intracranial pressure identified in this study is of any clinical consequence. None of the 26 preeclamptic patients had neurologic sequelae in this small study. The five patients with increased optic nerve diameter and presumed increased intracranial pressure apparently had no symptoms, and the authors do not state whether or not they received neuraxial anesthesia. Given the uncertainty of the clinical consequences of the degree of increased intracranial pressure identified, the refusal of neuraxial anesthesia might have unintended consequences, including poorly managed pain and the need for tracheal intubation and general anesthesia. Because cerebral edema typically is more diffuse than hemorrhage or tumor, it is not clear that it introduces the same risk of brainstem herniation with removal of cerebrospinal fluid. Those with severe preeclampsia, who have a higher risk of seizures than do those with mild preeclampsia, did not have greater optic nerve sheath diameters in this study. However, the study likely was underpowered to detect this secondary outcome. It is also not known whether this technique would be useful for determining the efficacy of treatment for cerebral edema and seizure prophylaxis in this setting. The immediate time course of change in optic nerve sheath diameter with intracranial pressure is not known, and there would have to be little hysteresis for this test to be used to guide therapy. Larger studies with patients with more severe preeclampsia will be required to answer these questions.

Measurement of optic nerve sheath diameter has been validated as a measure of increased intracranial pressure in a variety of settings. Although not yet validated in an obstetrical setting, it might prove useful in assessing altered consciousness and rapidly determining the severity and potential neurologic threat from other pathologies, such as intracranial hemorrhage or brain tumor. In addition, it is possible that intracranial hypotension might be associated with smaller optic nerve sheath diameter. Indeed, because optic nerve sheath ultrasonography can be used to detect increased intracranial pressure, it might also be able to detect intracranial hypotension in the setting of a dural leak. If so, it might also be useful for predicting improvement with a blood patch after unintended dural puncture or surgical repair of a dural tear associated with larger spine surgeries. If it proves useful for these diverse purposes, essentially as a window into the cranium, it would improve our diagnostic armamentarium and better guide therapy. This article serves as an introduction of this promising technique to anesthesiology, and it is hoped the report will spur future studies to further delineate the uses and limitations of the technique.

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