

Epidural Anesthesia for Delivery Complicated by Benign Intracranial Hypertension

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Benign intracranial hypertension (also known as pseudotumor cerebri) is a rare syndrome characterized by prolonged elevation of intracranial pressure in the absence of intracranial mass lesions, obstruction of the cerebral ventricles, or intracranial infection. Headache and blurring of vision are common symptoms, but diplopia, tinnitus, nausea, and vomiting may also occur. Consciousness, intellect, and mentation are not disturbed. Usually the only sign is papilledema, but abducens palsy may develop. Cerebrospinal fluid (CSF) pressure is in the range of 300–600 mm H₂O, but its composition is normal.¹⁻³

Benign intracranial hypertension includes a heterogeneous group of disorders. The idiopathic form, one of the commonest, occurs in healthy subjects without any known etiologic factor. Other forms have been associated with venous sinus thrombosis following middle-ear infection, obesity, hypervitaminosis A, Addison's disease, hypoparathyroidism, prolonged adrenocorticosteroid therapy, menstrual dysfunction, and use of oral contraceptive agents.^{1,3}

Benign intracranial hypertension occurs in both sexes at all ages, but is most common in nonpregnant women of childbearing age, particularly obese women who have menstrual irregularities.^{4,5}

In general, a disturbance of water and electrolyte balance due to hormonal factors is held responsible for the increase in intracranial pressure, but the pathogenesis is uncertain.² Johnston considers benign intracranial hypertension an example of the reduced CSF absorption syndrome due to an increase in CSF volume secondary to impaired absorption.⁶ The diagnosis can be made only by exclusion following complete neurologic examination and laboratory studies. Treatment includes repeated lumbar punctures and CSF drainage, salt and fluid restriction, weight reduction, diuretics, steroids, subtemporal decompression, and attention to known etiologic factors. Benign intracranial hypertension has a good prognosis; the

syndrome is self-limiting, and spontaneous remissions occur. However, in long-standing cases vision is threatened.

The condition is extremely rare during pregnancy. Only 29 such cases have been reported.^{2-5,7} Its development during pregnancy may be related to a state of functional hyperpituitarism.² It may occur in only one pregnancy or may recur in subsequent pregnancies.⁷ It invariably abates after abortion or delivery.

The paucity of information concerning the management of obstetric anesthesia for patients who have benign intracranial hypertension prompted us to report the following cases.

REPORT OF TWO CASES

Patient 1: A 20-year-old black woman, gravida 2, para 1, height 170 cm, weight 75 kg, 32 weeks pregnant, was admitted complaining of headaches and severe visual disturbances of one month's duration. She had experienced several transient episodes of complete loss of vision. Her previous pregnancy had been normal throughout.

Physical examination revealed bilateral papilledema. Lumbar puncture disclosed clear cerebrospinal fluid at a pressure of 480 mm H₂O. Results of all investigations, including roentgenograms of the skull, brain scan, and CSF analysis, were normal, and a diagnosis of benign intracranial hypertension was made. Treatment consisted of bed rest with an initial course of treatment with steroids and diuretics. During the hospital stay the patient's condition improved, but she continued to complain intermittently of headaches and visual disturbances. Decompressive lumbar punctures performed after 20, 30, and 41 days of hospitalization to relieve her symptoms revealed CSF pressures of 360, 320, and 260 mm H₂O, respectively. Fetal growth, determined by ultrasound, progressed satisfactorily, and serial estriol levels were normal. Papilledema was a constant finding. After 61 days the patient went into labor spontaneously. When contractions were well established and the cervix was 4–5 cm dilated, an epidural catheter was inserted through a 17-gauge Tuohy needle at the L3–4 interspace. Following a test dose of 2 ml, 6 ml of 0.5 per cent bupivacaine provided satisfactory analgesia. A further 8 ml bupivacaine was administered after 70 min. The patient's blood pressure and the fetal heart rate, monitored by Doppler ultrasound (Fetasonde 2100), remained stable throughout. Anesthesia was uneventful, and after a further 20 min, a 3,200-g healthy female infant was delivered spontaneously. The Apgar scores were 9 and 10 at 1 and 5 minutes, respectively.

The patient's condition improved markedly after delivery of the infant. Visual disturbances and headaches ceased, and only minimal papilledema could be detected.

Patient 2: A 21-year-old white woman, gravida 1, para 0, weight 57 kg, height 154 cm, in good health, was first seen at the antenatal clinic when eight weeks pregnant.

Six months previously she had been hospitalized because of

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severe headaches and papilledema. At that time the CSF pressure had been 340 mm H₂O, but CSF composition had been normal. Extensive neurologic studies, including brain scan, cerebral angiogram, and pneumoencephalogram, had excluded an intracranial space-occupying lesion, and a diagnosis of benign intracranial hypertension had been made. Her condition had improved gradually without treatment over a ten-day period, after which she had been discharged asymptomatic with normal fundi.

Throughout pregnancy the patient was followed at the high-risk antenatal neurology and ophthalmology clinics. Initially no abnormality was detected, but during the twenty-first week of pregnancy, disc margins were blurred, and subsequently, bilateral papilledema occurred. The patient did not complain of headaches or visual disturbance at any stage, the course of pregnancy appeared normal, and therapy was not deemed necessary.

At the patient's final visit to the clinic at 40 weeks gestation, the cervix was found to be 80 per cent effaced and 3 cm dilated. Following her admission to the hospital cerebrospinal fluid pressure was 290 mm H₂O. By the next morning she was in active labor. An epidural catheter was inserted at the L3-4 level, and following a test dose of 2 ml bupivacaine, 0.5 per cent, a further 6 ml produced satisfactory analgesia with a sensory blockade to the T10 level.

Analgesia was maintained with three additional doses of bupivacaine, 6 ml. However, over the next five hours labor did not progress well. Despite good uterine contractions and a trial of oxytocin after full cervical dilatation, a transverse arrest of fetal descent occurred at the +1 to +2 station. Cephalopelvic disproportion was suspected. Sensory blockade was raised to T6-7 by the administration of an additional 10 ml bupivacaine and an uneventful cesarean section performed. A male infant, weighing 2,280 g, was delivered. Apgar scores were 7 and 8 at 1 and 5 min, respectively.

The mother's postoperative course was uneventful except for three days of fever, which was considered to be related to endometritis and was successfully treated with antibiotics. The condition of the fundi improved, and she was discharged ten days after delivery with good vision.

DISCUSSION

In both the cases described the CSF pressures were known to be elevated during pregnancy and further increased during labor. CSF pressure consistently rises in response to myometrial contraction and is associated with increases in central venous pressure, stroke volume, cardiac output and arterial blood pressure.⁸ Such increases in pressure are independent of pain and have been observed during sleep and even in the presence of sensory blockade to the T4 level.⁸ They are, however, exaggerated by pain. Elevations in intracranial pressure during contractions have been shown by Marx *et al.* to be related to skeletal muscle contractions occurring in response to pain.⁹

Analgesic drugs such as meperidine are associated with respiratory depression and increases in PaCO₂ and cerebral blood flow. The use of such analgesics in patients who have abnormally high intracranial pressures is best avoided.

General anesthesia may be associated with increases

in intracranial pressure unless hyperventilation is utilized.¹⁰ Maternal hypocarbia during delivery may produce fetal hypoxia and acidosis, and is therefore undesirable.¹¹ Furthermore, the mechanical effects of hyperventilation may decrease uterine blood flow.¹¹ For these reasons general anesthesia was not chosen. Powell described a case in which general anesthesia was used for cesarean section in a patient who had benign intracranial hypertension; the baby had an Apgar score of 9 at 1 min, but details of anesthetic management were not reported.⁵ Continuous regional anesthesia has been recommended for the management of labor and delivery when increases in intracranial pressure should be avoided.¹² Marx *et al.* have described the successful management of parturients with intracranial tumors employing epidural anesthesia.¹²

When a patient has an intracranial mass lesion, tonsillar herniation following inadvertent dural puncture with an epidural needle is a major hazard, but herniation does not occur in patients who have benign intracranial hypertension. These considerations suggested the use of epidural anesthesia, which proved satisfactory for two such patients.

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