

## Preexisting Subclinical Neuropathy as a Risk Factor for Nerve Injury after Continuous Ropivacaine Administration through a Femoral Nerve Catheter

Stephan Blumenthal, M.D.,\* Alain Borgeat, M.D.,† Konrad Maurer, M.D.,‡ Beatrice Beck-Schimmer, M.D.,§ Uta Kliesch, M.D.,|| Michael Marquardt, M.D.,# Judith Urech, M.D.#

NERVE injury is a well-recognized complication of peripheral nerve blocks. Capdevila *et al.*<sup>1</sup> reported an incidence of 0.21% in a large prospective multicenter study including more than 1,400 patients with perineural catheters. Beside factors related to the regional anesthesia technique,<sup>1</sup> other causes such as concomitant patient disease,<sup>2</sup> inadequate positioning or manipulation during surgery,<sup>3</sup> and the neurotoxicity of local anesthetics<sup>4,5</sup> have been implicated.

We report on a patient who developed a femoral neuropathy after continuous femoral block. The only risk factor found in this patient was a postoperatively discovered, preexisting subclinical polyneuropathy.

### Case Report

A 72-yr-old man (American Society of Anesthesiologists physical status II) was scheduled to undergo left total knee arthroplasty. His medical history was unremarkable. All laboratory results were in normal range. The anesthetic procedure included continuous femoral nerve block, single-shot sciatic nerve block, and spinal anesthesia. The patient was orally premedicated with 7.5 mg midazolam. In the preanesthetic room, he was monitored and venous access was established.

For femoral catheter placement, the patient was placed supine. The puncture point was located 2 cm distally of the inguinal ligament and 1.5 cm lateral of the femoral artery. After disinfection and local anesthesia of the skin, a 30° short beveled 21-gauge needle (Stimuplex A; Braun, Melsungen, Germany) connected to a nerve stimulator (Stimuplex HNS 11; Braun Melsungen AG) with initial settings of 1.4-mA current intensity, 0.1-ms impulse duration, and 2-Hz frequency was introduced. The needle was cranially directed with 45° to the skin. After a "dancing patella" response was elicited at a depth of 5 cm with 0.35 mA, a 23-gauge catheter (Polymedic; Te Me Na, Bondi, France) was placed with the cannula-over-needle technique. The catheter was secured by subcutaneous tunneling and fixed to the skin with adhesive tape. After negative aspiration, 30 ml ropivacaine, 0.5%, was administered slowly through the catheter. Nerve localization, catheter place-

ment, and local anesthetic injection were performed without occurrence of pain or paresthesias/dysesthesias at any time.

The sciatic nerve block was performed using a proximal, lateral approach at the thigh. After localization of the puncture point 2 cm distal of the greater trochanter, a 22-gauge short beveled needle (Polymedic) was connected to the nerve stimulator and introduced parallel to the table. Needle placement was considered successful when plantar flexion could be elicited with current intensity of 0.4 mA and 0.1-ms impulse duration. After careful negative aspiration, 20 ml ropivacaine, 0.5%, was slowly injected.

The blocks were tested and considered successful after loss of cold perception in the distribution areas of the femoral, tibial, and peroneal nerves within 15 min after local anesthetic application.

Spinal anesthesia was performed in lateral decubitus position. After skin infiltration with 1% lidocaine, a 27-gauge spinal needle (Whitacre; Becton Dickinson AG, Basel, Switzerland) was introduced at the L4-L5 interspace by the paramedian approach. Correct needle placement was identified by free flow of cerebrospinal fluid, and 2.5 ml hyperbaric bupivacaine, 0.5%, was slowly injected. The spinal needle was removed, and the patient was placed supine. The upper level of the sensory block was determined bilaterally using loss of sensation to cold at the T4 level. The intraoperative course was uneventful. During cementation of the knee prosthesis, a thigh tourniquet was inflated for 23 min with a pressure of 280 mmHg. The surgical procedure was uneventful and lasted 150 min. Postoperative analgesia was provided with 0.2% ropivacaine given continuously through the femoral nerve catheter at a rate of 10 ml/h. One gram acetaminophen was administered every 6 h. Two hundred twenty minutes after intrathecal application of bupivacaine, complete recovery of sensibility in the right leg was documented. Postoperative analgesia in the first 48 h was efficient, and no additional narcotics were necessary. The degree of motor block was not specifically documented, but no adverse or unusual events were noted.

The continuous ropivacaine infusion was stopped 48 h after the start, and the femoral catheter was removed 6 h later. At that time, a persistent weakness of the left quadriceps was noted. Neurologic examination disclosed hyposensitivity in the medial aspect of the left thigh restricted to the L3 dermatome, and absence of patellar tendon reflex was observed. Ultrasonography in the area of the puncture point of the femoral catheter excluded the presence of a hematoma. Magnetic resonance imaging of the lumbar spine revealed neither a lesion of the nerve roots nor hematoma at the spinal anesthesia puncture site.

On the fourth postoperative day, an electrophysiologic investigation was performed (fig. 1). The needle electromyography showed low-voltage denervation activities, presenting as sharp waves and fibrillation potentials in the medial vastus muscles bilaterally. In the left and right anterior tibial muscles, signs of neurologic degeneration were also found. The neurography revealed normal conduction velocity of the right tibial nerve (43 m/s) and marginal decreased conduction velocity of the left tibial nerve (39 m/s). Latencies of the tibial somatosensory evoked potentials were in normal range on both sides.

On the ninth postoperative day, the patient was discharged with persistent weakness of the left quadriceps muscle, hyposensitivity

\* Consultant, † Professor and Chief of Staff, # Resident, Department of Anesthesiology, Orthopedic University Clinic Zurich. ‡ Consultant, Neural Plasticity Unit, Institute of Child Health, UCL, London, United Kingdom. § Associate Professor, Department of Anesthesiology, University Hospital Zurich, Switzerland. || Consultant, Neurophysiology, Spinal Cord Injury Center, University Clinic Zurich, Balgrist, Switzerland.

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Address correspondence to Dr. Borgeat: Orthopedic University Hospital Balgrist, Forchstrasse 340, CH-8008 Zurich, Switzerland. alain.borgeat@balgrist.ch. Individual article reprints may be purchased through the Journal Web site, www.anesthesiology.org.

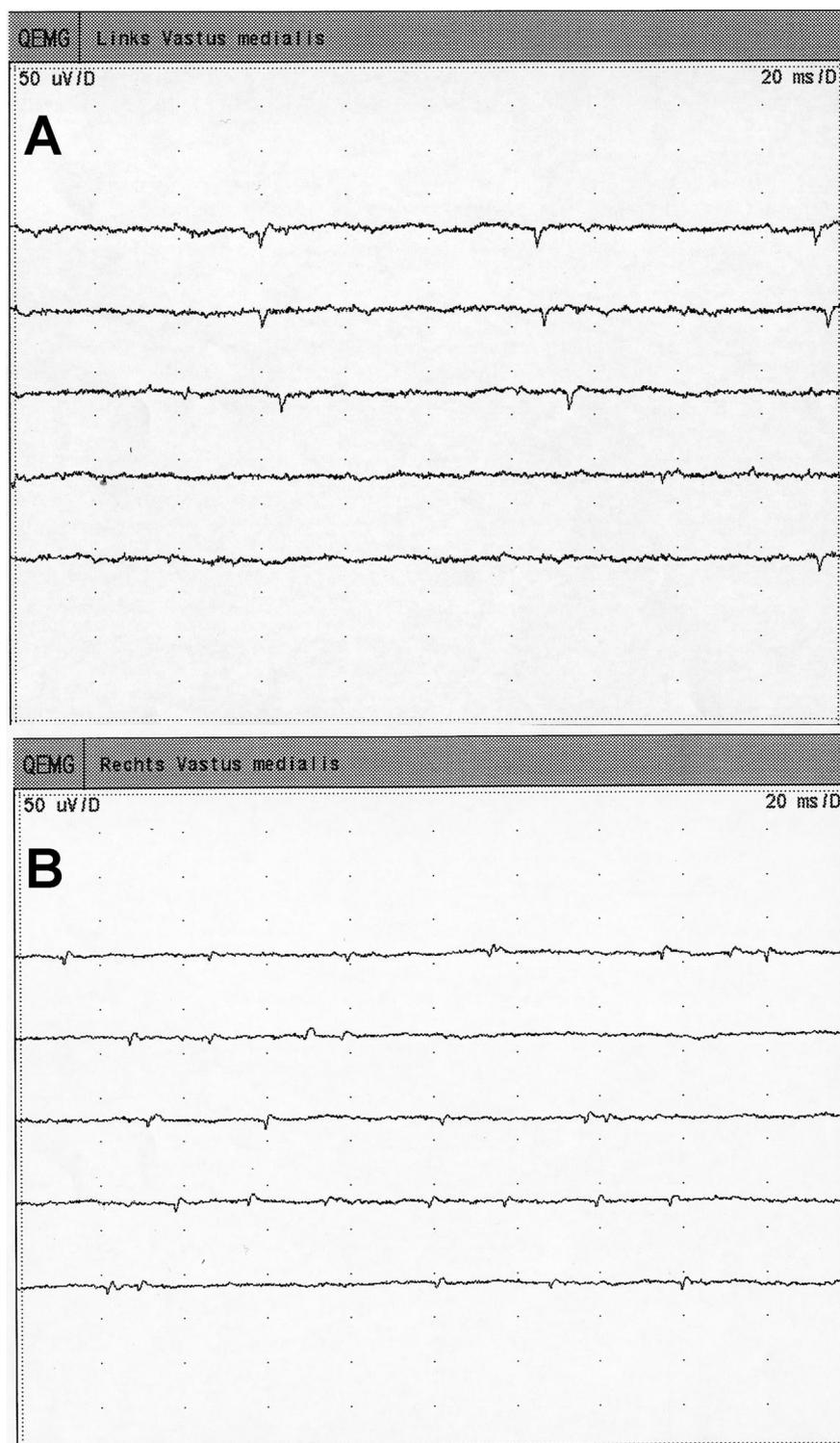


Fig. 1. Needle electromyography of the left (A) and right (B) medial vastus muscle 4 days postoperatively. The y-axis indicates the voltage in 50 microvolts per division ( $\mu\text{V}/\text{D}$ ). The x-axis indicates the time in 20 milliseconds per division ( $\text{ms}/\text{D}$ ). The results show low-voltage denervation activities, presenting as sharp waves and fibrillation potentials, consistent with a preexisting neuropathy.

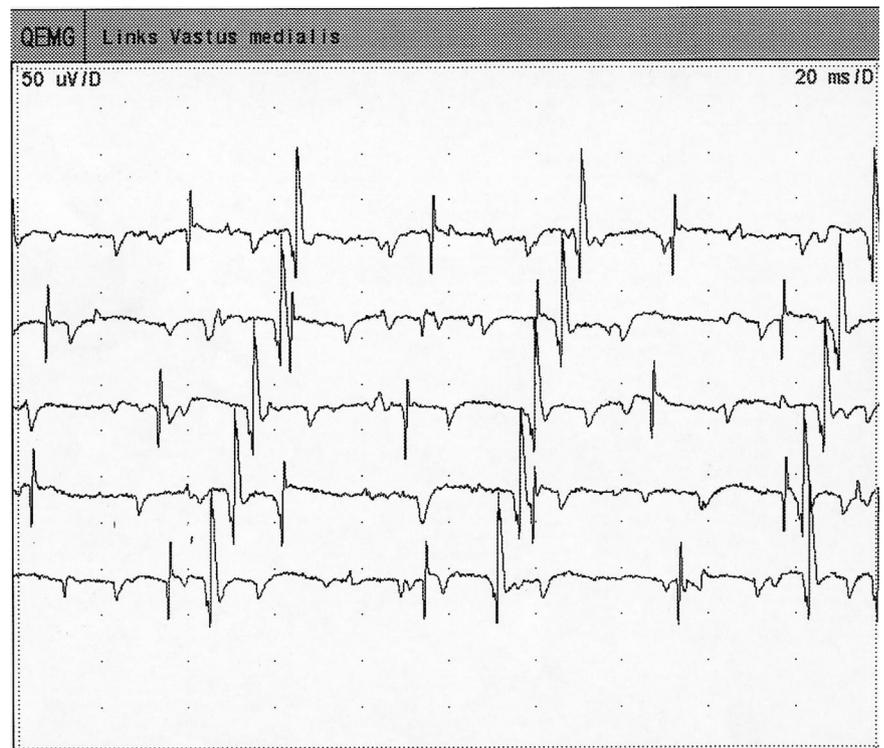
of the medial aspect of the thigh, and abolished patellar tendon reflex.

After 3 months, moderate weakness of the left quadriceps muscle was still present, but the patellar tendon reflex could be triggered on both sides. A new electromyography was performed and revealed high-voltage dense-frequent denervation (fibrillations and sharp waves) within the left quadriceps muscle (fig. 2). Both tibial anterior muscles showed the same low-voltage denervation as before. After 6 months, complete sensorimotor recovery of the left quadriceps muscle was noted.

## Discussion

This case showed the occurrence of prolonged sensorimotor deficit of the femoral nerve after continuous femoral nerve anesthesia/analgesia in a patient with a preoperatively undetected polyneuropathy. None of the usual predisposing factors for the development of a neuropathy was obvious in this patient.

**Fig. 2.** Needle electromyography of the left medial vastus muscle at 3 months postoperatively. The y-axis indicates the voltage in 50 microvolts per division ( $\mu\text{V}/\text{D}$ ). The x-axis indicates the time in 20 milliseconds per division ( $\text{ms}/\text{D}$ ). This examination revealed high-voltage dense-frequent denervation activities (fibrillations and sharp waves), consistent with an acute lesion. The assessment of the right medial vastus muscle at 3 months showed unchanged results compared with the primary electromyography.



Postoperative neurophysiological studies were performed to investigate the left-sided weakness of the quadriceps muscle, the hyposensitivity in the medial aspect of the thigh, and the abolished patellar tendon reflex. The initial investigation showed signs of denervation in the medial vastus muscle, including low-voltage fibrillations potentials and positive sharp waves. These findings are consistent with a preexisting neuropathy and could not be explained by an acute lesion, because it usually takes 3–4 weeks after nerve damage for these signs to be detected.<sup>6</sup> Moreover, the presence of a preexisting neuropathy is supported by similar pathologic needle electromyographic findings on the nonoperated contralateral limb. The second examination, performed 6 weeks after surgery, revealed a partial lesion of the femoral nerve with high-voltage fibrillations and sharp waves, representing signs of acute denervation. No needle electromyographic change was observed on the other side. The second needle electromyography is consistent with worsening of the preexisting neuropathy after continuous application of local anesthetics, which is supported by the absence of new needle electromyographic modifications on the other side.

Little is known about the impact of continuously applied local anesthetics on predamaged nerves. This could possibly include risks for nerve lesions such as mechanical trauma, neuronal ischemia, or local anesthetic neurotoxicity.<sup>4</sup> Neurologic damage after local anesthetic block is explained in most cases by two or more of these factors.<sup>7</sup> A definite mechanism can rarely be stated. In our patient, a direct nerve trauma by the needle or intraneural injection

seems unlikely because no pain or paresthesias/dysesthesias occurred during the block or the catheter placement, and no unusual resistance during local anesthetic injection was encountered.<sup>7</sup> Magnetic resonance imaging of the lumbar spine excluded the presence of a hematoma compressing the femoral nerve root. Nerve trauma due to other external factors such as surgical retractors was unlikely because the surgical site was not in the proximity of the femoral nerve. The thigh tourniquet was used for only 23 min with 280 mmHg and therefore could hardly be responsible for the damage.

It is conceivable that the application of local anesthetics on preinjured nerves may predispose these nerves to further damage. Preexisting pathology has been reported to play a role in the development of postoperative nerve injury.<sup>6,8,9</sup> Alvine and Schurrer<sup>8</sup> performed bilateral nerve conduction studies in patients in whom perioperative ulnar neuropathy occurred. They found abnormal slowing of the nerve conduction on the contralateral side despite the absence of clinical signs or symptoms of neuropathy. They suggested that subclinical neuropathy may become symptomatic after perioperative manipulations, such as traction and stretching.

Local anesthetics are potentially neurotoxic,<sup>4,5</sup> and the neuronal blockade can be seen as a reversible expression of this. The neurotoxicity parallels the anesthetic potency and has been identified to be dose and concentration dependent.<sup>5</sup>

Radwan *et al.*<sup>10</sup> showed that ropivacaine leads to growth cone collapse in chick embryo dorsal root ganglion cells in a concentration below the common clinical

concentrations used for regional anesthesia. The author concluded that local anesthetic application may interfere with the growth and regeneration of neuronal tissue and that the detrimental effects of local anesthetics on growing or regenerating neurons should be considered.<sup>10</sup> According to this statement, although 0.5% ropivacaine is usually not neurotoxic, the underlying neuropathy of our patient may have contributed to make the femoral nerve more susceptible to the potential neurotoxic effect of continuous local anesthetic infusion.

As a clinical implication, the occurrence of unexplained neuropathy after a continuous perineural catheter should warrant for the search of subclinical undetected preexisting neuropathy. The recognition of preexisting subclinical neuropathy is challenging. Few data are available on this subject, and future prospective studies looking at this issue will be needed to evaluate and anticipate the possible implications of its occurrence on the practice of regional anesthesia. Its presence will be helpful for the understanding of the complication and may also have medicolegal implications.

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## Neurocysticercosis Masquerading as Eclampsia

Lydia Grondin, M.D.,\* Robert D'Angelo, M.D.,† John Thomas, M.D.,‡ Peter H. Pan, M.D.†

THE World Health Organization estimates that 2.5 million people worldwide are infected with *Taenia solium* (pork tapeworm, a platyhelminth).<sup>1,2</sup> It is the most prevalent parasitic central nervous system disease and is endemic in Africa, South and Central America, and Southeast Asia, where seroprevalence was estimated to be 4.9-24%.<sup>1,2</sup> Its occurrence is uncommon in developed countries, but there has been a significant increase in cysticercosis prevalence in United States, especially in areas where there is an increasing immigrant population from endemic areas.<sup>3</sup>

Humans are the only definitive host of *T. solium*, and pigs are the common intermediate host. A human may also become an intermediate host by ano-oral contamination or eating food containing *T. solium* eggs. The hatched eggs can penetrate the intestines and then hematogenously spread to somatic muscles, the eyes, and the central nervous system. There, they develop into a cysticercus, a

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fluid-filled cyst with an invaginated scolex, leading to cysticercosis.<sup>3,4</sup> The central nervous system is involved in 60-90% of cases. The brain parenchyma is the most commonly affected. However, the ventricular system, subarachnoid space, and basilar cisterns may be involved and lead to obstructive hydrocephalus, intracranial hypertension, seizures, or meningoencephalitis, all of which have significant anesthetic and analgesic implications for treatment of parturients during labor and delivery.<sup>3,4</sup> Furthermore, diagnosis of neurocysticercosis during pregnancy is often confounded by the fact that many of these associated symptoms, such as headache, nausea/vomiting, visual or other neurologic changes, and seizures, may occur during pregnancy and/or preeclamptic/eclamptic states and they may also be misdiagnosed as post-neuraxial anesthetic complications, such as meningitis or post-dural puncture headaches. A Medline literature search revealed no published case reports and limited information on anesthetic/analgesic management of parturients with symptomatic neurocysticercosis.

## Case Report

A 23-yr-old Hispanic woman, gravida 4, para 1012, at 32 weeks' gestation presented to the emergency room with the symptom of waking up in the morning with headache and weakness in the right arm and leg. While in the emergency room, she had two episodes of witnessed tonic-clonic seizures. The seizures were treated initially

\* Obstetrical Anesthesia Fellow, † Associate Professor, ‡ Assistant Professor.

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Address correspondence to Dr. Pan: Section of OB Anesthesia, Department of Anesthesiology, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, North Carolina 27157-1009. ppan@wfubmc.edu. Individual article reprints may be purchased through the Journal Web site, www.anesthesiology.org.

with lorazepam and intravenous magnesium with the presumptive working diagnosis of eclampsia. Although drowsy, she followed commands with no further focal neurologic deficits. She denied any history of seizures, right upper quadrant pain, or visual changes. She was normotensive with no proteinuria. Her liver function and basic metabolic profile test results were normal.

On further evaluation, a noncontrast computed tomographic cranial scan was obtained, which showed evidence of a left parenchymal lesion with significant surrounding cerebral edema consistent with stroke, infection, or tumor. A subsequent magnetic resonance imaging study with and without contrast showed an 8-mm cystic lesion that was thought to be more consistent with either infectious etiology or tumor, such as glioma. Further interrogation of the patient and family revealed a history of living in Mexico on a pig farm, eating undercooked pork and dirt secondary to pica. Together with her clinical history, the diagnosis of neurocysticercosis was made after consultation with neurology, neurosurgery, neuroradiology, and infectious disease. Antihelminthics were not initiated because of possible worsening of symptoms with cyst destruction during pregnancy. Betamethasone was administered for fetal lung maturity, and dexamethasone was administered for controlling cerebral edema. Magnesium was discontinued, and phenytoin was administered, which was subsequently changed to divalproex because of recurrent seizures. Fetal heart tracing was reassuring and the biophysical profile score, a measure of fetal well-being that incorporates a nonstress test and four ultrasound variables (breathing movement, body movement, muscle tone, and amniotic fluid volume), was 8 out of 10. Two days later, the patient again had a witnessed tonic-clonic seizure necessitating an increase in the divalproex dose. The following morning, she also developed new right-sided weakness and a progressive headache. A repeated magnetic resonance imaging study showed no significant interval changes in the cranial scan and no cystic lesions in the spine or spinal cord. Furosemide and mannitol were administered to further decrease cerebral edema. A team discussion about options for delivery included vaginal delivery with passive second stage *versus* a cesarean delivery because of cerebral edema and possible associated increased intracranial pressure. With continual reassuring fetal heart tracing, a cesarean delivery was planned the following morning after overnight fasting and stabilization of the patient.

With cricoid pressure applied, general anesthesia was induced for the cesarean delivery using modified rapid sequence technique with thiopental, succinylcholine, and fentanyl to blunt the sympathetic response to laryngoscopy and intubation. The maintenance anesthetic consisted of oxygen, nitrous oxide, isoflurane, and intravenous morphine. A live infant, weighing 2,081 g, was delivered with Apgar scores of 7 and 9 at 1 and 5 min. The neonate was taken to the neonatal intensive care unit, where he required nasal continuous positive airway pressure support for 6 days and eventually did well. The patient remained stable throughout the operative period and was extubated at the end of the case awake and following commands. The patient was transferred to the intensive care unit. Postoperatively, 400 mg oral albendazole twice a day was initiated in addition to ongoing antiseizure prophylaxis with 500 mg divalproex every 6 h and 4 mg dexamethasone every 12 h. On postoperative day 2, the patient experienced another seizure, but no further neurologic changes or seizures ensued, and the focal deficits present before delivery resolved. On postoperative day 4, she was discharged home in stable condition on divalproex, dexamethasone, and albendazole. She reportedly had full resolution of her neurologic symptoms and intracranial lesion after completion of her 30-day course of treatment.

## Discussion

Multiple neurologic disorders may complicate pregnancy, leading to seizure activity or altered mental sta-

tus.<sup>5</sup> Preeclampsia and eclampsia occur in 6–8% and 0.04–0.1% of pregnancies in developed countries, respectively. However, signs of severe preeclampsia (increased blood pressure, proteinuria, and edema) may be mild or absent in up to 30% of eclamptic patients.<sup>6,7</sup> Until the diagnosis is confirmed, all new-onset seizures occurring during pregnancy should be considered eclampsia.<sup>5,6</sup> However, new onset seizures during pregnancy without increased blood pressure or proteinuria merit a full neurologic investigation to rule out cerebral, parenchymal, or metabolic diseases.

In this patient, computer tomographic and magnetic resonance imaging studies combined with clinical history led to the diagnosis of neurocysticercosis. Initial treatment is control of seizures using antiepileptic drugs. Antiinflammatory agents, although controversial, may also be used because symptoms often are caused by an inflammatory reaction to a ruptured cyst.<sup>2,4,8</sup> Although safe use of antihelminthics during pregnancy has been reported, these are often withheld until the postpartum period if symptoms are well controlled.<sup>9</sup> An increase in cerebral edema from an inflammatory reaction to cyst destruction by antihelminthics can exacerbate the neurologic symptoms in 50–80% of patients, usually occurring between days 2 and 5 after initiation.<sup>9</sup> Our patient showed exacerbation on day 2 of treatment but without further symptoms afterward. Medical or surgical treatment, such as cerebrospinal fluid diversion or cyst removal, is required if seizures are uncontrolled, if intracranial hypertension or encephalitis is present, or if the neurocysticercosis is an extraparenchymal form.<sup>8</sup>

The location and size of the cystic lesion may preclude the use of neuraxial anesthesia because cerebral edema and mass effect may be exaggerated. The decrease in colloid oncotic pressure and increase in blood volume and cardiac output during pregnancy can further increase cerebral edema and intracranial pressure.<sup>10</sup> This patient continued to have seizures, altered mental status, and new neurologic deficits despite treatment with antiepileptics and dexamethasone. Therefore, both furosemide and mannitol were administered to reduce her cerebral edema.

Seizures, altered mental status, cerebral edema, and intracranial hypertension make the administration of neuraxial anesthesia controversial in this patient. Vaginal delivery would have required a passive second stage, which is also controversial because painful uterine contractions can increase intracranial pressure.<sup>11</sup> In other circumstances, where neurologic symptoms are well controlled or absent, vaginal delivery with epidural analgesia and passive second stage forceps-assisted delivery seems appropriate.<sup>12</sup>

With spinal anesthesia being relatively contraindicated and after careful airway evaluation, general anesthesia was the appropriate cesarean delivery anesthetic. The goal of anesthetic induction without further increase of

intracranial pressure was achieved using thiopental, opioid, and muscle relaxant together with gentle, quick laryngoscopy and intubation. Were antihypertensives needed, labetalol or esmolol would have been our first choice because they do not cause cerebral vasodilation. The use of succinylcholine is controversial, but the longer onset time of nondepolarizing muscle relaxant for induction may expose the patient to aspiration, hypoventilation, and hypercapnia. Although hyperventilation reduces intracranial pressure, normocapnia was maintained because hypocapnia and alkalosis result in the deleterious umbilical and uterine vasoconstriction with a left shift of the oxyhemoglobin dissociation curve causing fetal acidosis.<sup>13</sup>

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