More than Just a Headache: Neurocysticercosis Infection in Pregnancy

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Case Study 4
More than Just a Headache: Neurocysticercosis Infection in Pregnancy

Reason for Selecting this Case

Twenty years ago, while working in a refugee camp in Thailand, I observed patients, students, coworkers and friends suffer from tropical and parasitic diseases common to the area. I was not immune and suffered from some of these diseases. Because of these experiences, I became fascinated and horrified by these illnesses.

Five years later, I encountered patients with these type of diseases again while working with refugees through Parkland Hospital, Dallas, Texas. Then, in 2008, I cared for patients with neurocysticercosis and epilepsy while working in Parkland’s neurology clinic. I became interested in this case of neurocysticercosis in a pregnant patient because it was opportunity to review and expand my knowledge both about this disease and about the care of the pregnant patient. This knowledge is indispensable to the practicing clinician because the incidence of neurocysticercosis is rising in the United States due to the increase in immigrants arriving from endemic areas (Serpa, Yancy, & White, 2006).

Types and Number of Encounters

I extracted the case study data from the patient’s hospitalization record. She had given her consent to access her medical record. This patient was admitted to Parkland Hospital in Dallas, Texas, for a total of seventeen days. An Advanced Practice Nurse (APN) in the OB/Gyn (Obstetrics/Gynecology) triage area first evaluated the patient. Her care was subsequently managed by OB/Gyn residents and attending faculty, with input from the Neurology, Infectious Disease, and Neurosurgery services. She was discharged from the hospital by the Neurosurgery surgery services. An Acute Care Nurse Practitioner (ACNP) completed her discharge summary.
The site of this case study was Parkland Hospital, Dallas, Texas.

**Systems Analysis**

Parkland Hospital is a 672 bed, taxpayer funded, county hospital in Dallas, Texas. University of Texas Southwestern (UTSW) medical residents, medical students and APNs, under the supervision of attending faculty, provide health care. In 2010, there were 12,091 births at this hospital. Prenatal care is protocol driven and is based on patient risk factors. Patients classified as low-risk receive pre- and post-natal care by an APN (a Women’s Health Care Nurse Practitioner (WHCNP), Family Nurse Practitioner (FNP), or Certified Nurse Midwife (CNM). A CNM attends low-risk deliveries. In-hospital post-partum care may be provided by a CNM or WHCP. OB/Gyn residents medically manage high-risk patients throughout their pregnancy. UTSW Obstetrics faculty members supervise all providers.

In this case, study, the patient was receiving low-risk prenatal care at one of Parkland’s community-based prenatal clinics. When she became ill, she was initially assessed and treated by an APN in the OB/Gyn triage area located in Parkland Hospital. After she failed to respond to interventions, her care was transferred to an OB/Gyn resident for further management.

Currently, there are no APNS with a Doctor of Nursing Practice (DNP) degree employed in Ob/Gyn services. An important role for a DNP in this population would be to design, implement, and test methods to educate patients and staff about neurocysticercosis infection.

**Day One**

**Initial site.** The initial site of this case study was the OB/Gyn triage area.

**Chief complaint.** The patient’s chief complaint was a headache.

**History of Present Illness.** The patient was a 29-year-old female who presented with a headache that started the previous day. The pain had gradually intensified since the onset of the
headache. She reported that the pain was 10 on a scale of 1 to 10 (with 1 being the lowest score and 10 being the highest). The pain was located on the front, top, and back of the head. She had not taken any medications for the pain.

**Past Medical History.** The patient had no history of migraine headache.

**Medications.** The patient is taking prenatal vitamins.

**Family History.** The patient had a non-contributory family history.

**Allergies.** The patient had no known drug allergies.

**Social profile.** The patient was married and lived with her husband. Her primary language is Spanish. Three months ago, she moved from Mexico to California, then to Texas one month ago.

**Review of systems.**

**General.** The patient had no fever or chills. She had nausea today, but no vomiting.

**HEENT.** She denied cough, congestion, or sore throat.

**Gastrointestinal.** She reported no abdominal or right upper quadrant pain. She reported that she eats every three hours and has early satiety.

**Neurological.** She denied visual changes or gait disturbances.

**Gynecological.** The patient reported no history of abnormal pap smears or sexually transmitted diseases. She had normal, cyclic menses prior to her pregnancy.

**Obstetrical.** The patient was grvida one with no contractions, vaginal bleeding or leaking fluid. She reported good fetal movement.

**Physical Exam**

**Vital signs.** The patient’s blood pressure was 119/71. She had a pulse of 102, respiratory rate of 18, and temperature of 36.6 degrees Celsius (°C).
Last Menstrual Period. The patient’s last menstrual period was 10/14/2009. The estimated gestational age (EGA) was 35 weeks.

General. The patient was alert, oriented, and in no acute distress.

Abdomen. Her abdomen was gravid. It was soft and nontender. Murphy’s sign was absent and there was no guarding. She had no palpable contractions. The estimated fetal weight was five pounds, and the fetal heart rate was 160 beats per minute.

Gynecological. Her digital cervical exam was significant for one cm of dilation, and her cervix was 25% effaced. The uterus was ballotable. The presentation was cephalic, and the station was -4.

Laboratory. The patient’s urine dipstick was negative for protein and positive for a large amount of ketones. Complete blood count, electrolytes, and liver function tests were ordered. An outpatient sonogram was consistent with a 34-week pregnancy.

Assessment.

1. A 29-year-old G1P0A0, with an EGA of 35 weeks, 1 day by dates and 34 weeks by sonogram.
2. Headache.
3. Dehydration.

Treatment plan. The patient was given one gram of acetaminophen. She was given a 500 ml bolus of D5 lactated ringer’s solution intravenously (IV), then 125 mls per hour. External fetal monitoring was performed for one hour.

Brief discussion of pathophysiology. Physiologic changes during pregnancy that cause headaches include increased circulatory volume, vasodilation caused by high levels of circulating progesterone, and tissue edema resulting from vascular congestion. Other causes
include stress, fatigue, sinus congestion, caffeine withdrawal, low blood sugar, muscle spasm, allergens, hyperventilation, or noxious fumes. Headaches not caused by pathology are very common (Remich, 2004, “Headache,” para 1). Pregnant patients may have sinus infections, tension, migraine, or cluster headaches. More serious causes of headache include cardiovascular disease, (TIA or stroke), pregnancy-induced hypertension, preeclampsia, eclampsia, and HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome (Remich, 2004). The patient’s blood pressure was normal and she had no protein in her urine, so preeclampsia was excluded from the diagnosis. Life-threatening causes of headache include subarachnoid hemorrhage, cerebral venous thrombosis, cerebral infarction, intracranial tumor, and intracranial infection (Lennox & Firth, 2010, “Disorders of the Central Nervous System,” para 2).

**Progression.** Over the next six hours, the patient vomited twice and had an episode of diarrhea. Two doses of Midrin (isometheptene, dichloralphenazone, and acetaminophen), usually given for a diagnosis of tension or migraine headache, were not effective. There was no evidence of fetal distress. The patient’s ketones returned to normal after completion of the IV fluids. The care of the patient was transferred to a second-year obstetrics (OB) resident. When he reviewed her medical history, he learned that the patient usually had had a headache once a month, which resolved spontaneously. The headache she was experiencing now was much stronger, intensified with light and movement, and improved with sleeping. She had no sick contacts, upper respiratory symptoms, urinary complaints, or other neurological symptoms. Her temperature was 37.5°C and her exam was normal. The complete blood count (CBC) was remarkable for an elevated white count of 12.98 (see Table 1). Except for slightly decreased sodium and potassium levels, the metabolic profile was normal (see Table 2).
Table 1

*CBC with Differential*

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>12.98</td>
<td>3.90-10.70</td>
</tr>
<tr>
<td>Hgb</td>
<td>12.3</td>
<td>12.1-16.1</td>
</tr>
<tr>
<td>HCT</td>
<td>35.0</td>
<td>36.8-48.7</td>
</tr>
<tr>
<td>MCV</td>
<td>90.2</td>
<td>74.0-102</td>
</tr>
<tr>
<td>PLT</td>
<td>202</td>
<td>174-404</td>
</tr>
<tr>
<td>Lymphs (absolute)</td>
<td>1.31</td>
<td>2.00 to 4.00</td>
</tr>
<tr>
<td>Monos (absolute)</td>
<td>0.67</td>
<td>0.40 to 0.80</td>
</tr>
<tr>
<td>Eos (absolute)</td>
<td>0.03</td>
<td>0.01 to 0.04</td>
</tr>
<tr>
<td>Baso (absolute)</td>
<td>0.02</td>
<td>0.05 to 1.0</td>
</tr>
</tbody>
</table>

Table 2

*Metabolic Profile*

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>134</td>
<td>135-145</td>
</tr>
<tr>
<td>K</td>
<td>3.4</td>
<td>3.6-5.0</td>
</tr>
<tr>
<td>Cl</td>
<td>104</td>
<td>98-109</td>
</tr>
<tr>
<td>CO2</td>
<td>19</td>
<td>22-31</td>
</tr>
<tr>
<td>BUN</td>
<td>5</td>
<td>6-23</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.46</td>
<td>0.51-0.95</td>
</tr>
<tr>
<td>Glucose</td>
<td>114</td>
<td>70-114</td>
</tr>
<tr>
<td>AST</td>
<td>24</td>
<td>10-35</td>
</tr>
<tr>
<td>ALT</td>
<td>20</td>
<td>10-35</td>
</tr>
<tr>
<td>GGT</td>
<td>9</td>
<td>5-36</td>
</tr>
<tr>
<td>Alk Phos</td>
<td>136</td>
<td>35-104</td>
</tr>
<tr>
<td>Total Bilirubin</td>
<td>0.3</td>
<td>0.2-1.3</td>
</tr>
<tr>
<td>Direct Bilirubin</td>
<td>0.1</td>
<td>0.0-0.3</td>
</tr>
<tr>
<td>Amylase</td>
<td>78</td>
<td>28-100</td>
</tr>
<tr>
<td>Lipase</td>
<td>28</td>
<td>7-59</td>
</tr>
<tr>
<td>Albumin</td>
<td>3.2</td>
<td>4-6</td>
</tr>
</tbody>
</table>
Urinalysis showed a moderate leukocystosis, a trace amount of blood, and negative protein. The medical plan was to continue Midrin for a total of five doses, monitor her temperature, and observe for response.

The patient subsequently developed a temperature of 38.0°C and nuchal rigidity. A urine culture was sent to assess for a urinary tract infection and a neurology consultation was requested.

**Neurology consult.** The history and exam completed by the neurology resident was significant for photophobia and nuchal rigidity. There was no papilloedema. The differential diagnosis included bacterial versus viral meningitis, or venous thrombosis. He obtained a lumbar puncture (see results in Table 3). His diagnostic and treatment recommendations included antibiotics (vancomycin and ceftriaxone), dexamethasone, magnetic resonance imaging (MRI) and magnetic resonance venography (MRV).
Table 3

*Lumbar Puncture Results*

<table>
<thead>
<tr>
<th>Test</th>
<th>Results</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening Pressure</td>
<td>Not done</td>
<td>5-20 cm</td>
</tr>
<tr>
<td>Glucose</td>
<td>50</td>
<td>40-70</td>
</tr>
<tr>
<td>Protein</td>
<td>39</td>
<td>15-45</td>
</tr>
<tr>
<td>Cell Number</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Cell Number CSFA</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>RBC</td>
<td>7850</td>
<td>Less than 0</td>
</tr>
<tr>
<td>RBC CSFA</td>
<td>58</td>
<td>0</td>
</tr>
<tr>
<td>Nucleated cells</td>
<td>218</td>
<td>0-5</td>
</tr>
<tr>
<td>Nucleated cells CSFA</td>
<td>293</td>
<td>0-5</td>
</tr>
<tr>
<td>Polys</td>
<td>87</td>
<td>No range</td>
</tr>
<tr>
<td>Polys CSFA</td>
<td>90</td>
<td>No range</td>
</tr>
<tr>
<td>Lymphs</td>
<td>11</td>
<td>No range</td>
</tr>
<tr>
<td>Lymphs CSFA</td>
<td>8</td>
<td>No range</td>
</tr>
<tr>
<td>Monos, macro</td>
<td>2</td>
<td>No range</td>
</tr>
<tr>
<td>Monos, macro CSFA</td>
<td>2</td>
<td>No range</td>
</tr>
<tr>
<td>Appearance CSF</td>
<td>hazy</td>
<td>No range</td>
</tr>
<tr>
<td>Appearance CSFA</td>
<td>clear</td>
<td>No range</td>
</tr>
<tr>
<td>Color</td>
<td>pink</td>
<td>No range</td>
</tr>
<tr>
<td>Color CSFA</td>
<td>Colorless</td>
<td>No range</td>
</tr>
<tr>
<td>Volume</td>
<td>3.0 cc</td>
<td>No range</td>
</tr>
<tr>
<td>HSV PCR</td>
<td>pending</td>
<td>negative</td>
</tr>
<tr>
<td>Gram Stain</td>
<td>negative</td>
<td>negative</td>
</tr>
</tbody>
</table>
After the neurology consult was completed, the OB resident discussed the case with his attending physician. The attending physician observed that the patient’s peripheral white blood count was elevated, but explained that this could be normal for this stage of pregnancy. The lumbar puncture results were not strong evidence for bacterial meningitis. Antibiotics were started while waiting for the results of the imaging studies. Only one dose of dexamethasone was given to the patient due to concerns about its fetal effects.

**Discussion of differential.** Bacterial meningitis classically presents as fever, headache, and nuchal rigidity and is often accompanied by a decreased level of consciousness. Other common symptoms include nausea, vomiting, and photophobia. Seizures occur in 20-40% of patients. Bacterial meningitis is a medical emergency, and antibiotic therapy (a third-generation cephalosporin and vancomycin) should be started immediately after blood cultures and lumbar puncture are obtained. Infections are most likely to be caused by Streptococcus pneumonia, Neisseria meningitides, group B streptococci, and Listeria monocytogenes (Roos & Tyler, 2008). Cerebral spinal fluid (CSF) shows a polymorphonuclear leukocytosis, decreased glucose and/or increased protein concentration, and increased opening pressure. Both bacterial infection and the host’s immune response cause obstructive and communicating hydrocephalus, vasculitis, arterial and venous thrombosis. This results in increased intracranial pressure (ICP), cerebral herniation, coma, and death. Mortality ranges from 3% to 20%, depending on the organism. About 25% of survivors have decreased intellectual function, memory impairment, seizures, hearing loss, dizziness, and gait disturbances (Roos & Tyler, 2008).

Patients with viral meningitis present with headache, fever, mild nuchal rigidity, malaise, myalgia, lethargy, anorexia, nausea and vomiting, abdominal pain, and/or diarrhea. The CSF shows a lymphocytic pleocytosis, normal glucose, normal to slightly elevated protein, and a
normal or mildly elevated opening pressure (Roos & Tyler, 2008, “Viral Meningitis”, para.1). Viral meningitis is caused by enteroviruses, herpes simplex virus type 2, and arboviruses. Treatment is primarily symptomatic, and many cases can be treated on an outpatient basis. Most adults recover without long term side effects (Roos & Tyler, 2008, “Viral Meningitis” para. 4).

Cerebral venous thrombosis (CVT) is the formation of a blood clot in the lateral or superior sagittal venous sinuses. Headache is the most common symptom. In the pregnant patient, CVT usually occurs in the postpartum period. The disorder is rare in developed countries but common in undeveloped countries where there are high rates of sepsis and preeclampsia. MRI is the best imaging test (Neurological and Psychiatric Disorders, 2005, “Cerebral Vascular Diseases,” para. 4).

MRI results. The patient’s MRI showed a “cystic mass within the body of the right ventricle, 1.0 cm by 1.4 cm by 9 mm, which may represent intraventricular neurocysticercosis. No frank hydrocephalus. Differential diagnosis includes neoplasm.”

MRV results. The MRV results were negative for venous thrombosis.

Rationale for testing. Computed tomography (CT) and magnetic resonance imaging (MRI) are the most important imaging techniques in the diagnosis of neurological disease. The advantages of using CT are that it is quicker and more readily available. It is particularly useful to detect bleeding and infarcts (in stroke and subarachnoid hemorrhage), intracranial infections, intracranial masses, and to evaluate trauma. MRI is used to evaluate the spine, as well as neurological conditions affecting the white matter (epilepsy, stroke, tumors, and congenital anomalies). MRV is used to evaluate the central nervous system venous system (Molyneux, Renowden, & Bradley, 2010, ”Imaging in Neurological Diseases,” para. 4).
Day Two

The OB attending physician noted that the patient had developed fever and photophobia, and that the MRI was consistent for neurocysticercosis (NCC). Infectious Disease Services (IDS) were consulted. The neurology attending physician recommended that neurosurgery services be consulted and that the patient be placed on seizure precautions.

IDS recommendations included testing the CSF for Bartonella and taenia antibodies, blood cultures, a PPD to exclude tuberculosis, and testing serum for brucella and coxiella antibodies. IDS also advised delaying treatment for NCC until after delivery due to potential central nervous system effects with treatment. They recommended that an ophthalmologist evaluate the patient to exclude ocular cysticercosis. They advise that the patient be observed for headache, alteration in mental status, nausea and vomiting, and focal neurological deficits.

Discussion of neurocysticercosis. Neurocysticercosis, an infection with the pork tapeworm *Taenia solium*, affects 50 million people worldwide. Most infections occur in developing nations where unregulated pig farming takes place. This includes Central and South America, sub-Saharan Africa, India, and Asia. Neurocysticercosis is now a significant emerging infection in the industrialized world due to immigration and travel (Pandian, Venkateswaralu, Thomas, & Sarma, 2007, p. 285).

*Taenia* infection occurs when a human consumes undercooked pork that contains cysticerci in its tissues. The *taenia* larvae attach to the intestinal wall and develop into a tapeworm. Infection may be asymptomatic or there may be minor abdominal pain, distention, nausea, or diarrhea. In this stage, treatment is with niclosamide or praziquantel (Van Voorhis & Weller, 2010).
When a taenia infection is not treated, tapeworms grow in the intestine, intermittently releasing portions of the tapeworm (proglottids) containing eggs into feces. Once excreted, the eggs are eaten by pigs that have access to human waste matter. Alternatively, the eggs contaminate the hands of the carrier. Carriers may then autoinfect themselves or pass the eggs to another person via unwashed hands. When ingested, the eggs hatch into larval onchospheres. These penetrate the intestinal wall and then carried via the bloodstream to the tissues, where they form cysticeri. Cysticeri have been found in the subcutaneous tissue, muscle, heart, and eyes as well as in the central nervous system (Serpa et al., 2006, p. 1052).

When cysticeri invade the central nervous system, the condition is known as neurocysticercosis (NCC). Seizures may occur when the cysticeri lodge in the white matter, and are the most common manifestation of the NNC (Anis et al., 2009). Cysticeri may also be found within the ventricles of the brain. If they block normal CSF circulation, hydrocephalus develops and results in headache, mental status changes, seizures, and neurological deficits. Cysticeri in the subarachnoid space can cause mass effect, seizures, meningitis, or stroke. When located in the pituitary sella, there may be endocrine dysfunction. Obstruction of the cranial nerves can cause ophthalmic deficits. Cysticeri in the spinal cord can cause neurological defects (Serpa et al. 2006).

The cysticeri evolve through four stages. In the first stage, living cysticeri have the ability to modulate the human immune response, thus preventing detection, host inflammatory response, and destruction. As a result, the patient may not have any symptoms. As the cyst ages, it loses the ability to avoid detection by the host’s immune system. The resulting inflammatory response may instigate seizures. In this stage (the colloidal stage), lesions enhance on MRI. In the third and fourth stages, the cyst is replaced by necrotic tissue and then calcifies into small
(one to four mm) lesions (Bales & Schumann, 2000; Serpa et al., 2006; VanVorthis & Weller, 2010). Knowledge of these stages assists the practitioner in choosing the most useful imaging studies and guides treatment decisions. CT with contrast identifies living, non inflamed cysticerci as hypodense areas, while degenerating cysticerci are isodense or hyperdense lesions. MRI can visualize living or degenerating cysts, but does not accurately detect calcified cysts. It is best at visualizing intraventricular cysts (Bales & Schumann, 2000, p. 245.). Antigen testing can assist in the confirmation of infection (Serpa et al., 2006.)

Treatment of NCC is complicated and based on the stage of cyst development, number of lesions, location, and symptoms. Symptoms should be controlled with anti seizure medication, corticosteroids, and, if hydrocephalus develops, shunt placement. Treatment for living or degenerating cysts consists of albendazole 15 mg per kg per day for at least seven days or praziquantel 50 to 100mg/kg/day for 15 days. Left untreated, the cysticerci will eventually die. There is evidence that treating cysts result in faster resolution and decreased seizure recurrence (Serpa et al., 2006). Larval death from anti-parasitic medication instigates a host inflammatory response, usually between the second and fifth day of therapy. The resulting edema and intracranial hypertension leaves the patient at a higher risk for seizures during treatment and rarely, death (Garcia & Gilman, 2010). Patients who have massive infections are not treated with antiparasitic drugs due to the possibility of inducing life-threatening cerebral edema. Treatment of calcified cysts is not indicated. Ventricular cysts should not be treated prior to removal as it makes them friable. Subarachnoid cysts require placement of an intraventricular shunt prior to extended treatment with albendazole and steroids (Serpa et al., 2006.)

Neurocysticercosis in pregnancy. A study by Pandian, et al. (2007) addressed maternal and fetal outcomes in Indian women with epilepsy. They compared 30 women with epilepsy due
to NCC with 30 women with epilepsy due to other causes. The participants were age and parity matched. The study found no difference in the outcomes between the groups.

Discussion of differential.

Bartonella is a gram-negative bacteria directly transmitted to humans via fleas, sand flies and mosquitoes. Alternatively, humans may be infected through animals harboring fleas (usually cats, squirrels, mice, rats, and dogs). At least eight species of bartonella cause infection in humans. Of these, two are known to cause meningitis in humans. Bartonella henselae, the organism that causes cat scratch fever, has been found to cause aseptic meningitis as well as encephalitis (Liles, 2010; Pinto et al., 2008). Bartonella wahoensis is a recently identified organism. There are two reported cases of this bacteria causing meningitis (Probert, et al., 2009).

Brucellosis is a bacterial infection caused by several species of the Brucella organism. The disease is transmitted to humans via contact with meat, placentas, or by drinking unpasteurized dairy products from infected cattle, hogs, or goats. In the United States, this disease is rare. A few cases occur in the Midwest, or diagnosed in visitors or immigrants from Mexico, Spain, and South America. Symptoms include fatigability, headache, arthralgias, anorexia, irritability, and intermittent fevers. Chronic infection can cause spondylitis and suppurative arthritis, endocarditis, meningoencephalitis, pneumonitis, hepatitis, and cholecystitis (Brucellosis, 2009).

Coxiella burnetii is caused by a Rickettsia and is contracted by inhaling dust, drinking unpasteurized milk, or handling infected goats, cows, or sheep. Over half of all patients are asymptomatic. The remainder of the patients develop acute Q fever, which manifests as fever, headaches, myalgias, arthralgias, cough, and rash. It may lead to hepatitis, pneumonia,
meningitis, meningoencephalitis, myocarditis, and pericarditis. The infection is particularly dangerous in pregnancy as it can cause spontaneous abortion, intrauterine growth retardation, oligoamnionhydrosis, intrauterine fetal death, and premature delivery. About 1 to 5% of infected persons will develop chronic Q fever. The risk factors for developing chronic Q fever include pregnancy, immunocompromised states, damaged cardiac valves, or abnormal blood vessels. Chronic Q fever may result in endocarditis, osteomyelitis, chronic hepatitis, pseudotumors of the spleen or lung, chronic pericarditis, or infection of a ventriculoperitoneal shunt (Renvoise, & Raoult, 2010).

Tuberculous meningitis is a particularly dangerous form of meningitis as it has a 40% mortality rate. A CT or an MRI may show meningeal enhancement and hydrocephalus. As with other types of meningitis, the CSF opening pressure is usually elevated, and CSF has a high white blood cell count (usually lymphocytosis), elevated protein, and decreased glucose. If tuberculosis meningitis is suspected, antitubercular therapy should be started. Therapy should also include corticosteroids. Tuberculous meningitis is always fatal if not treated (Blumberg & Leonard, 2006, “Extrapulmonary Tuberculosis,” para.8).
Day Three

In the morning, the patient was afebrile and had decreased headache and neck stiffness. After input from Infectious Disease, Neurosurgery, and Neurology services, the obstetrician’s plan was to manage the patient medically until she reached her 37th week of pregnancy. Then, the fetus would be delivered via C-section and the cyst would be removed from the ventricle.

*Rationale for delaying delivery until the 37th week.* Preterm birth is considered any birth before the end of the 37th week of gestation. Infants who are born near term but at less than 37 weeks are at risk for respiratory distress syndrome, sepsis, temperature instability, hypoglycemia, jaundice, and poor feeding. There is an associated increase in hospital costs (Behrman & Butler, 2007).

Later that evening, the patient developed acute mental status changes. She was disoriented, unable to follow commands, and had right upper extremity automatisms. While having an emergency head CT, she developed status epilepticus.

*Discussion of status epilepticus.* Status epilepticus (SE) is defined as a prolonged seizure (lasting more than five to 15 minutes), continuous seizures, or multiple seizure episodes without intervening periods of consciousness (Cooney & Stone, 2008, p. 277). The overall mortality of SE is about 20% but varies based on age, etiology, and the duration of the event. Status epilepticus can have severe long-term manifestations. About 20% to 40% of patients develop epilepsy after a single episode of SE. Several studies have shown that that SE can lead to the development of chronic encephalopathy (Manno, 2003, p. 510).

The patient’s seizures did not respond to Keppra, (levitcitertram), Versed, (midazolam) Ativan, (lorazepam), or Cerebyx (fosphenytoin). She became hypotensive and was intubated. The fetal heart tones (FHTs) ranged from 140’s to 170’s, which was within normal limits. The
patient seized for two and a half hours. Her obstetrics team considered preterm delivery so that the patient could receive management (i.e. surgical removal of the ventricular cyst). The CT scan showed “Interval development of hydrocephalus with prominence of bilateral temporal horns and third ventricle. No definite evidence of herniation. Stigmata of remote neurocysticercosis.” These findings and the patient’s clinical status prompted the emergency placement of an external ventricular drain (EVD) to relieve the buildup of CSF.

A repeat CT scan after the EVD was placed showed worsening hydrocephalus and transependymal edema. The patient was transferred to the neurosurgical intensive care unit (ICU) for further monitoring. She remained intubated and was monitored for seizure activity via continuous EEG (electroencephalogram), and for increased intracranial pressure (ICP) via the EVD. She was placed on a propofol drip and intravenous levetiracetam to suppress seizures. At that point, her condition had stabilized. The FHTs were normal and the maternal pH was stable. An ICU attending physician commented that much of the neurological activity that was thought to represent seizure activity during the patient’s episode of SE was actually posturing caused by hydrocephalus. An ophthalmologist examined the patient and did not find any evidence of ocular cysticercosis.

**Rationale for exam.** The presence of ocular neurocysticercosis precludes the use of antihelminthics. Their use can cause an inflammatory reaction around the cyst and result in serious damage to the eye. A cyst requires surgical removal prior to the use of antihelminthics (Van Voorthis & Weller, 2010).

A repeat CT scan showed “Significant interval improvement in hydrocephalus, ventriculostomy catheter in place. Left frontal lobe consistent with prior neurocysticercosis.”
Days Four through Eight

The patient was weaned off propofol without recurrence of seizures, and then extubated without problems. EEG and ICP monitoring continued. Her mentation gradually cleared. The patient was observed to be emotionally overwhelmed and a referral to the chaplain was made.

Comment: There are no other mentions of the patient’s mental and emotional status in this case report. Besides profound physical and emotional trauma and language barrier, she must have experienced extreme concern for the well-being of her child. An important role of a DNP/APN would be to assist in assessing and obtaining emotional support systems for this patient.

The patient remained seizure-free on levetiracetam, and able to eat without problems. The results of serologies were negative (see Table 4). There were no concerns regarding fetal distress. There was no change in the plan to perform a C-section the following week when the patient completed her 37th week of pregnancy and to remove the ventricular cyst while the patient was still in the operating room.
Table 4

*Serology Results*

<table>
<thead>
<tr>
<th>Organism</th>
<th>Result</th>
<th>Ref. Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bartonella antibody</td>
<td>negative</td>
<td>negative</td>
</tr>
<tr>
<td>Brucella antibody</td>
<td>&lt;1:20</td>
<td>No range found</td>
</tr>
<tr>
<td>Cysticercosis antibody</td>
<td>0.33 O.D.</td>
<td>0.00-0.34 O.D.</td>
</tr>
<tr>
<td>Cysticercosis interpretation</td>
<td>negative</td>
<td>No range found</td>
</tr>
<tr>
<td>Q Fever Phase I</td>
<td>&lt;1:16</td>
<td>&lt;1:16us</td>
</tr>
<tr>
<td>Q Fever Phase II</td>
<td>&lt;1:16</td>
<td>&lt;1:16</td>
</tr>
<tr>
<td>Herpes simplex Virus (CSF)</td>
<td>negative</td>
<td>negative</td>
</tr>
<tr>
<td>Blood cultures (two)</td>
<td>negative</td>
<td>negative</td>
</tr>
<tr>
<td>HIV</td>
<td>negative</td>
<td>negative</td>
</tr>
<tr>
<td>RPR</td>
<td>negative</td>
<td>negative</td>
</tr>
<tr>
<td>HBsAG</td>
<td>negative</td>
<td>negative</td>
</tr>
</tbody>
</table>

**Days Nine and Ten**

On day nine, the patient experienced a spontaneous rupture of membranes. The neurosurgery cleared the patient for a vaginal delivery. They planned to carefully monitor her neurological status and monitor her ICP via the EVD during labor and delivery. She received epidural anesthesia and her labor was augmented with pitocin. The patient precipitously delivered in the labor room and was taken to the labor and delivery OR for the delivery of the placenta and the repair of a second-degree laceration. The infant was a 2884-gram male with Apgar scores of 7 and 9. His estimated gestational age was 37 weeks. The patient had an opportunity to hold the baby before he was taken to the Level I (normal) newborn nursery.

**Days Eleven through Thirteen**
There were no postpartum complications. The infant was able to visit his mother in the intensive care unit. The lactation consultant assisted the mother and infant with breastfeeding.

**Rationale for breastfeeding and the use of levetiracetam.** The World Health Organization recommends that infants be exclusively breastfed the first six months of life to achieve optimal growth, development, and health, unless absolutely contraindicated (World Health Organization, 2002). Levetiracetam is an antiepileptic drug with an undefined mechanism of action used to treat complex partial or generalized tonic-clonic seizures. It is excreted into breast milk, but only in very small amounts. (ACP PIER & AHFS DI® Essentials, 2010; McNamara, 2006). Therefore, the use of levetiracetam is not a contraindication to breastfeeding. It is recommended that the infant be observed for drowsiness, sufficient weight gain, and achievement of developmental milestones, especially in infants less than two months old or if the infant is exclusively breastfed (Levetiracetam, 2010, para 1).

The patient’s husband was supportive during her hospitalization. As the infant could not remain in the hospital until his mother had completed her treatment, a social work consultation was done to complete arrangements for the infant’s care post discharge. The plan was for the patient’s husband to care for the infant at home, with assistance from the baby’s maternal aunt. The maternal grandmother, who lives in Mexico, was attempting to obtain a visa to the United States so she could assist the family.

**Day 14**

The patient was taken to the operating room where a freely mobile green lobular cyst was removed from the third ventricle via neuroendoscopy.
Day 16

The neurosurgeons removed the EVD. Since a repeat CT scan showed no evidence of viable or degenerating cysts, IDS felt there was no reason to treat the patient with an antihelminthic medication.

Day 17

The patient was transferred to the floor.

Day 18

Occupational therapy services (OTS) evaluated the patient, and determined that she would require moderate assistance with aspects of daily living (ADLs). The patient was discharged home later that day.

Her discharge medications included hydrocodone, dexamethasone, docusate, levetiracetam, and ranitidine.

She was instructed not to lift anything heavier than 15 pounds and not to drive until she was seizure-free for six months. She was given patient education material discussing the diagnosis, transmission, prevention, and treatment of cysticercosis, as well as instructions on how to care for her craniotomy incision.

Her follow-up appointments included outpatient speech therapy (to be scheduled), OB/Gyn clinic in one week, and Neurosurgery clinic in two weeks.

Discharge ICD-9 codes.

Cysticercosis 123.1
Obstructive hydrocephalus 331.4
Grand mal status epilepticus 345.3
Normal Pregnancy V22
Spontaneous Vaginal Delivery 650123.1

*Major CPT codes.*

MRI Head w/o contrast 70551

Diagnostic lumbar puncture 62270

CT Head/Brain w/o contrast 70450

Creation of shunt; ventricular-atrial, -jugular, -auricular 62220

Neuroendoscopy, intracranial; with retrieval of foreign body 62163

**Two months post hospitalization**

At the patient’s follow-up appointment with neurosurgery, a CT scan showed the

“Resolution of pneumocephalus without evidence of hydrocephalous, right frontal burr hole with bridging metallic apparatus, and bifrontal punctuate calcifications, likely the sequela of remote neurocysticercosis.” The patient had not had any further seizure activity since her hospitalization and the levetiracetam was discontinued.
References


