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University of Nevada, Reno

**Case Study: The clinical manifestations and differential diagnoses of
Neurocysticercosis**

A thesis submitted in partial fulfillment
of the requirements for the degree of

Bachelor of Science in Biology and the Honors Program

By

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RENO**

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**Case study: The clinical manifestations and differential diagnoses of
Neurocysticercosis**

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Abstract

Neurocysticercosis is the most prevalent parasitic brain infection in the human population. It causes many symptoms, and can eventually lead to the death of the human host. Many diagnostic tools have been developed to detect the presence of such an infection. These include serum tests for the detection of elevated white blood cells and antibodies against the neurocysticercosis parasite. In addition, imaging studies, such as CT scans and MRI, can be used to detect the infection. A full case study has been written as well, which presents the illness in a clinical setting. Exams and tests were performed to solve the case of seizures for the patient, K.B. Differential diagnoses for the presented case are sorted until a final diagnosis of neurocysticercosis is proved. The plan of treatment is then established for the patient.

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Introduction

Neurocysticercosis is the most prevalent parasitic infection of the brain. It is caused by the life cycle of the tapeworm *Taenia solium*, where the eggs are excreted through human fecal matter which find their way back into the human body to the brain or muscle tissues. Because of this pathway, this disease is prevalent in regions with low sanitation conditions (Sinha & Sharma, 2009). Although the disease has been known since the time of the ancient Greeks, medical advances have not progressed until the 1970s and 1980s when new diagnostic technology and treatment plans emerged. CT scans and MRI have allowed physicians to diagnose neurocysticercosis accurately as well as to identify any complications that may be associated with the disease (Salgado, Rojas, & Sotelo, 1997).

A major issue with neurocysticercosis is the severity of the many symptoms and complications associated with the infection which must be addressed in order for a complete diagnosis of the disease (Jayaraman et al., 2011). These symptoms include ischemic cerebrovascular disease, arachnoiditis, vasculitis, infarction of brain tissue, ischemic attack, Brun's syndrome, cysticercotic encephalitis, and hemorrhage (Sotelo, 2011). The more prevalent complications are chronic hydrocephalus and epilepsy, depending on the area of infection. All of these follow their own prognosis which means the treatment for each symptom and complication can vary after the primary treatment for neurocysticercosis.

Once the complications have been diagnosed, treatment may begin. Cysticidal drugs like praziquantel and albendazole have been proven to be effective in clearing the neurocysticercosis infections (Escobedo, Penagos, Rodriguez, & Sotelo, 1987; Sotelo,

Escobedo, Rodriguezcarbajal, Torres, & Rubiodonnadieu, 1984). Multiple therapies have been established, all of which are proven to be effective. If drug therapy is chosen, there are some adverse effects when taking them. However, the effects are usually in the form of inflammation which can be treated with a brief course of steroid therapy. Another alternative treatment plan is through the surgical removal of the cysts but this procedure is unnecessary for most cases.

Overall, neurocysticercosis is caused by a simple infection that has many complications attached with it. Some of the steps toward eradicating the disease are outlined here through diagnosis and treatment. Other steps can be toward efforts in sanitation and public education in endemic areas.

Historical background/social context

As outlined previously, the root of neurocysticercosis lies in the deficiencies of sanitation. Because of this, the more prevalent regions are usually third world, developing countries in America, Africa, and Asia (Sotelo, 2011). However, non-pork consuming countries are not affected as much by this disease because of the implications of the helminthic parasite life cycle through pigs (Sotelo, 2011).

It is important to be familiar with the mechanisms of disease, including the modes in which the parasite infects the host and distributes its progeny (Sotelo, 2003). The origin of the disease is due to consuming undercooked pork that is infested with cysterici which houses the larvae. The larvae will then grow and evolve into its adult cestode form, *Taenia solium*, in the digestive tract. This worm will continue to grow for another three months until it is two to three meters long. At this point, the distal proglottids, which are the tail segments of the tapeworm containing eggs, are detached daily where it is excreted through feces. The eggs are then free to contaminate anything they land on, including food prepared under unsanitary conditions. Once the eggs have been ingested by humans or pigs, they will be absorbed by the digestive tract and lodged either in the brain or in the muscle where they will hatch and develop into cysterici. The cysterici in pigs will then repeat the cycle again which is shown in figure 1 (Sotelo, 2003).

This mechanism of transmission is not prevalent in developed countries because of the good sanitation found in those areas. The growth of the worm is a problem in developing countries because of the lack of certain sanitary measures like the existence of a sewage system. However, citizens from developed nations are not exempt from neurocysticercosis itself. People from developed countries traveling to third world areas

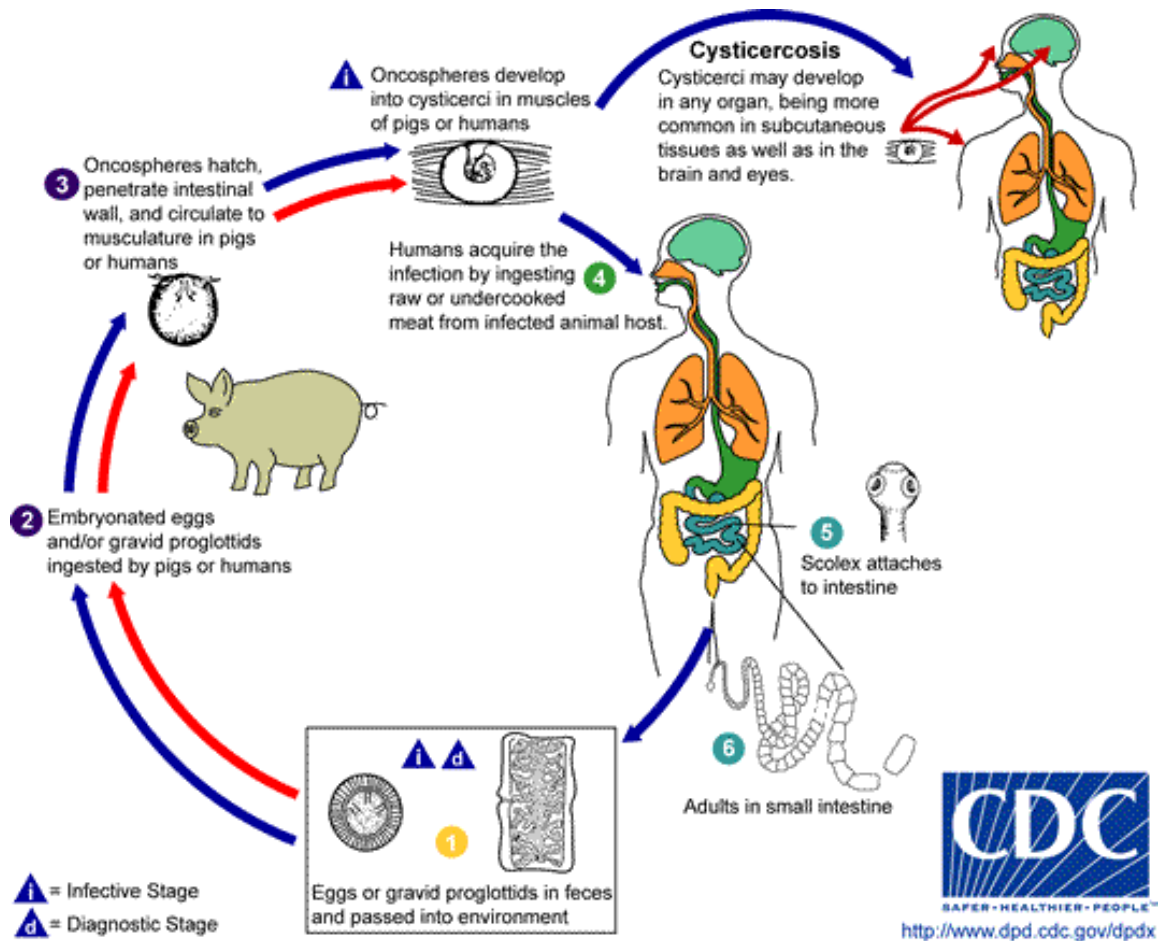


Figure 1. A *Taenia solium*-infected patient will continuously spread the parasite's egg for the life of the worm which can infect either pigs or humans, causing harm to both hosts. Once the embryonated eggs have been ingested by a human being, it can either infect the host in the digestive tract or in soft tissue such as the muscles and brain. Photo courtesy of the Center of Disease Control. <http://www.dpd.cdc.gov/dpdx/html/Taeniasis.htm>

have been found to contract the disease from eating the local food. The ingestion will cause the regular prognosis of neurocysticercosis to proceed, but the individual with the infection will most probably not be able to spread the disease to anyone else once they've returned home because of the implications of the fecal to pig vector transmission mechanism (Sotelo, 2003).

The mechanism of transmission also allows the spread of neurocysticercosis through one individual who has contracted the tapeworm. It explains why there is a

higher prevalence of incidence for cysticercosis compared to intestinal taeniasis, which is the infection of the tapeworms, since a single worm has the capacity to generate several thousand eggs daily (Sotelo, 2003).

Due to the high amount of eggs released from a single worm, areas endemic to this disease have a high rate of incidence for it, but the data collected from epidemiologic studies are incomprehensive. The frequency of prevalence has been estimated to range from 1% to 4% of the entire population of an endemic region (Sotelo, 2011). This estimates for around 50 million people affected a year. The reason for the lack of knowledge for this disease is due to the economic restrictions of diagnosing the disease using CT scans and MRI in the developing countries. However, there are different ways to diagnose patients that are cheaper than CT scans or MRI. A diagnostic tool physicians can use is by extracting the patient's cerebrospinal fluid and analyzing it (Sotelo, 2011). It may be a cheaper alternative way to diagnose patients, but there are complications tied to that method which will be discussed later in the diagnosis section. As a result of the unavailability of accurate diagnostic tools, patients with neurocysticercosis are usually not diagnosed correctly (Sotelo, 2011). For example, epilepsy, which is a common cause of neurocysticercosis, is a common standalone diagnosis in areas susceptible to neurocysticercosis infections. Physicians are not certain of a parasite infection in their patients so they cannot diagnose them with neurocysticercosis.

Although the general patterns of endemic areas are identified, the incidence of the disease is highly variable, even in areas within the same region. This is mainly due to the cultural and economic differences found between the areas. In other words, areas where religious practices inhibit the consumption of pork have relatively low levels of incidence

for neurocysticercosis because of the implications of the pig which is required for transmission. However, non-pork eaters are not completely safe from acquiring the disease. Pigs are only necessary for the transmission of the cysterci but not the eggs of the tapeworm. Foods prepared in unsanitary conditions with an individual infected with the tapeworm are the underlying cause of this disease (Sotelo, 2003).

Review of literature

New treatment methods, both medicinal and surgical, have been developed in the recent years. This includes the synergistic effects of drug therapy with both albendazole and praziquantel and the methodology of ventriculoperitoneal shunting.

Albendazole and praziquantel are very effective drugs with cysticidal effects for the treatment of cysticercosis. However, there are advantages and disadvantages for both drugs. Albendazole is the more inclusive drug that can be used against cysterici in almost any location, but the only treatment plan available is an 8-day course. Praziquantel is only effective against cysterici in the parenchyma of the brain, but there is a one-day treatment plan when using this drug. Both of these treatments are quite effective for their treatment courses but researchers wanted to know if both drugs can be combined to achieve a better effect against the parasite. After a 10-day course, patients were seen with higher cysticidal drugs in their plasma if they were taking both albendazole and praziquantel as opposed to patients taking albendazole and a placebo. This proves the increase in effectiveness of the drug against the parasite (Garcia et al., 2011). However, is it worth taking both medications for a longer period of time to essentially achieve the same healing effect at the end of the day?

Surgical intervention is not necessary in most cases of neurocysticercosis due to the effectiveness of drug therapy but the treatment of hydrocephalus is usually through ventriculoperitoneal shunting which is one of the only surgical procedures due to cysticercosis (Sotelo, Arriada, & Lopez, 2005). Hydrocephalus is when the circulation of the cerebrospinal fluid is disturbed which causes a build-up in pressure on the brain. This can cause damage to the sensitive areas of the brain so surgery is usually performed to

drain the excess fluid from the ventricles of the brain to the abdominal area. A small hole is drilled through the skull to place a catheter into a ventricle of the brain. Another catheter is placed into the abdominal cavity and flow is controlled by a valve which can be set to drain more or less fluid.

Case manifestations: differential diagnosis, underlying symptoms

As briefly stated previously, there are many symptoms associated with neurocysticercosis with the major ones primarily affecting the brain, described below. However, different areas of infection of neurocysticercosis will lead to different presentable symptoms. Cysts, which are shown in figure 2, can grow almost anywhere within the head. Some of these areas include the brainstem, the pituitary gland, and even the eye (Kelesidis & Tsiodras, 2011). These are the more sensitive areas where the cysts can cause a more severe clinical manifestation than cysts in other locations of the brain. The cysts found in the brain parenchyma, or the bulk of the brain, usually do not affect patients as much since the areas affected are usually not centers of major importance for basic functioning (Salgado et al., 1997). Patients with cysts in these areas may not even be aware of the infection. However, the severity of each symptom widely varies from patient to patient, even if the cysts have grown to 10cm in diameter (Jayaraman et al., 2011; Praet et al., 2010). In addition to those that reside within the skull, there are others that infect the intra spinal compartments. However, these locations are not as common as those found within the parenchyma and subarachnoid space of the brain.

For infections in the brain parenchyma, the prognosis, or the general trend of what is expected to happen, is benign for the most part compared to the prognosis for infections in the subarachnoid space or meninges, which are the membranes separating the nervous system from the rest of the body (Rajshekhar, 2010). Almost any neurological sign or syndrome could be the cause of neurocysticercosis. However, parenchymal neurocysticercosis is most closely associated with granulomas or calcifications and epilepsy. In many cases, patients will form cysts in their brain. These

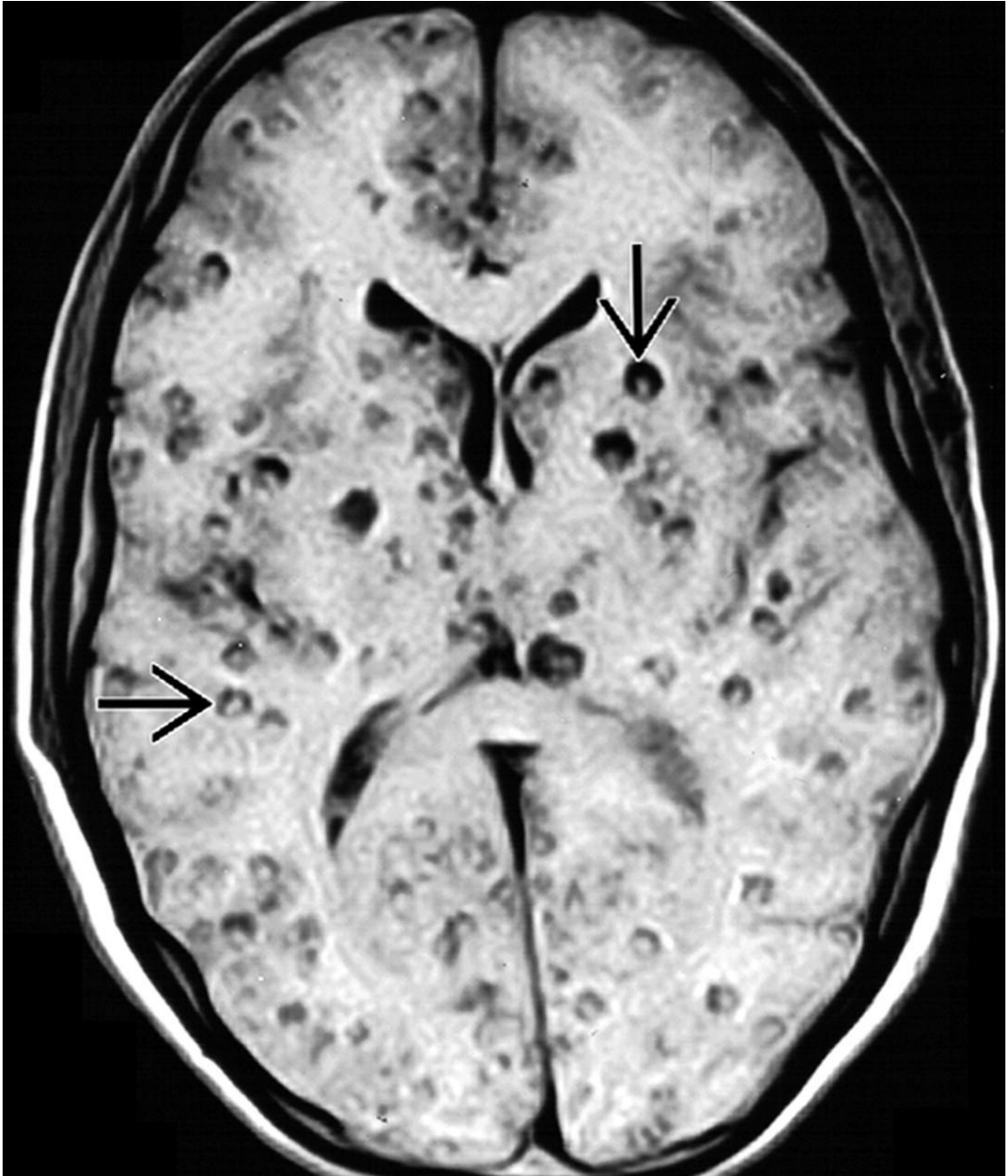


Figure 2. Multiple cysts can be seen in this MR image of a patient with neurocysticercosis. Some of the scolices of the tapeworm are shown by the arrows. Other parts of the parasite can be seen as the white areas within the cysts. Photo courtesy of <http://radiology.rsna.org/content/239/3/650/F25.large.jpg>.

cysts will cause inflammation in the area but they can disappear spontaneously due to the immune system. Those that are not exterminated by the host's immune system will usually turn into granulomas or calcifications. Granulomas are areas where the immune system has quarantined the pathogen. In these cases, the pathogen is not completely destroyed. They are just kept away from the body by forming a layer of lymphocytes around them. Calcifications are the most common findings of neurocysticercosis and they form because of the involution of the cysts when they die. It is interesting that many of the granulomas or calcifications are found in people due to reasons not associated with neurocysticercosis such as a scan of the head for injuries due to an automobile accident. This just strengthens the notion most cases of neurocysticercosis in the parenchyma are not serious since patients were not aware of the cysts beforehand. In addition to the calcifications, many patients also develop epilepsy which is a neurological disorder where patients experience repeated seizures over time. These seizures can affect normal activities and can cause physical harm to oneself as well as mental problems such as learning difficulties.

For infections within the meninges, more serious symptoms may appear (Sotelo, 2011). This is mainly due to the cerebrospinal fluid surrounding the cysts in the subarachnoid space. Cerebrospinal fluid has many functions including the circulation of wastes and the protection of the brain. As a result, any foreign matter that is found in the fluid will find its way around the different regions of the brain. Cysterci that have found its way in the subarachnoid space will induce a major amount of inflammation wherever it goes. This will cause widespread damage which is linked to numerous symptoms, including, but not limited to, arachnoiditis, vasculitis, brain infarctions, transient ischemic

attacks, brain hemorrhage, and hydrocephalus, which is the most frequent symptom associated with meningeal neurocysticercosis (Sotelo, 2011). Arachnoiditis and vasculitis are inflammations of the arachnoid mater and of the blood vessels, respectively. An infarction is tissue death due to the lack of local oxygen. A transient ischemic attack is the loss of blood flow to the brain for a short amount of time that does not cause tissue death due to disturbances in the circulation of cerebrospinal fluid. Hydrocephalus is the accumulation of cerebrospinal fluid in the ventricles of the brain due to improper circulation. This causes an increase in pressure exerted on the brain which can cause many problems, like visual loss, speech dysfunction, and memory loss (Sotelo, 2011). In addition, patients with hydrocephalus usually suffer from Bruns' syndrome, too. It is a condition where the patient will experience an acute headache followed by vomiting and vertigo.

An important aspect of the symptoms produced is that most of these symptoms are usually caused by the reaction of the host's immune system. As the immune system is fighting the infection, it is producing many inflammatory signals that will cause inflammation which is the root of most of these symptoms. Without the inflammation, some patients will be unaware of the infection because of the lack of symptoms (Rajshekhar, 2010).

In order to diagnose a patient with neurocysticercosis, certain diagnostic tools and tests are necessary for an accurate confirmation of the disease (Sotelo, 2011).

Cerebrospinal fluid studies and neuroimaging studies are the more reliable ways to diagnose patients. A cheap, yet ineffective method is to run serologic tests which will test for antibodies in a patient's bodily fluids. However, these tests will produce many false-

negatives and false-positives due to the implications of the disease so they are not used for diagnosis. Instead, physicians turn to the cheaper alternative of cerebrospinal fluid testing. This is reliable when identifying living cysterci, and is useful for determining a cysticidal treatment for the patient. However, the patient's consent for the lumbar puncture procedure is frequently refused because of its painful experience. This leaves neuroimaging as one of the only diagnostic tools for identifying cysticercosis. In general, CT scans and MRI are the best tools to diagnose patients with neurocysticercosis (Salgado et al., 1997). However, there is a major dilemma because of the high cost of such scans especially since the majority of patients in need of scans have low economic statuses. It is an unfortunate coincidence which may be relieved as new procedures are conceived along with the better availability of medical care (Sotelo, 2011).

Once the CT scan or MRI has been obtained, the physician can then determine the location of the cysts as well as the severity of the infection. The diagnosis for a patient with cysts in either the parenchyma or meninges is the same and both will be treated in a similar fashion as well. However, different complications and symptoms must be treated individually to cover the complete disease (Sotelo, 2011).

Plan for care/treatment

For the treatment of neurocysticercosis, most patients will undergo drug therapy to eliminate any live cysticerci in the brain. There are currently at least two drugs that can be used for this treatment. Praziquantel, which was first discovered as an effective drug for porcine cysticercosis, and albendazole are both used for cysticidal drug therapy with albendazole as the more preferred drug (Sotelo et al., 1984). It is the preferred drug because of many reasons, including its effectiveness in all areas of the body as opposed to praziquantel which does not spread in places like the cavities of the eye.

As with all treatments, there were some skeptical issues that were presented by using these drugs. The drugs did not guarantee the restoration of function in the patients (Sotelo, 2004), they caused inflammation because of the acute destruction of the parasite which could cause many other problems (Uddin et al., 2010), and infections that produced mild symptoms could be managed through symptomatic therapy which would eliminate the need for a powerful drug. These were the considerations of the physicians and researchers involved with the disease. However, it was shown through 25 years of observations that the drugs were reliable and safe (Julio Sotelo & Diaz-Olavarrieta, 2010). When taken early in the prognosis, the drugs would improve the neurologic dysfunctions caused by the infection. In addition, the inflammations caused by the use of the drug could be controlled through the use of anti-inflammatory agents like steroids. All in all, the drugs are safe and effective which leaves no reason to stop infections in patients with little to no symptoms (Sotelo, 2011).

There are several courses for each drug which the patients can take to get rid of the infection. Of course, these drugs target living cysts which means they will have no

effect on calcified cysts. However, they will work on granulomas because the parasite is effectively alive within the granuloma (Sinha & Sharma, 2009). The most common treatment is an 8-day course of albendazole at 15 mg/kg/day separated into two daily doses (Sotelo, 2011). This will effectively kill the parasites located in the parenchyma, ventricular system, subarachnoid space, and retina. In cases where the cysts have reached the ocular cavities, surgical extirpation must follow the therapy to remove any dead cysts there since lymphocytes won't be able to reach those places to clear the cysts like in the other locations. An alternative course of praziquantel is also used. This 2-week course is distributed in three daily doses of 50 mg/kg/day and it is primarily used to treat cysts located in the parenchyma (Sotelo et al., 1984). This is a longer treatment plan than albendazole and because of that, patient cooperation is usually lower. Lower patient cooperation, higher dosage, and lower area of effect are some reasons physicians prefer albendazole rather than praziquantel.

However, there is a different treatment course involving praziquantel. Researchers have identified the peak of plasma levels at two hours after the ingestion of praziquantel. As a result, a new single-day course was planned for patients which consists of three doses administered once every two hours (Sotelo et al., 1984). This was seen to be as effective as the traditional two-week course which was confirmed with CT scans or MRI. In addition to its effectiveness, it greatly reduced the cost of treatment as well as the length and total drug dose. This was also a good treatment plan for patients as well since the whole therapy can be accomplished within four hours which increased compliance. However, as mentioned earlier, praziquantel is only effective for cysts found in the parenchyma so it is far less effective at treating infections in ventricular and subarachnoid

locations. This is why there have been research suggesting a combined therapy with praziquantel and albendazole (Mahanty et al., 2011).

In addition to the treatment of the infection itself, there are treatments plans for the inflammation that induced due to the acute destruction of the parasites if necessary (Sotelo, 2011). It is usually simultaneously prescribed with albendazole or praziquantel with the dosage depending on the patient's reaction to the treatment. However, studies have shown steroids have different effects on the actual drugs themselves. It has the tendency to increase the plasma concentrations of albendazole while decreasing the concentration of praziquantel. Because of this effect, the steroid prednisone or dexamethasone is usually prescribed with the albendazole as a daily single dose intramuscularly. For praziquantel, it is usually prescribed to patients to take whenever they experience any discomfort related to inflammation such as headaches. In these cases, the steroid will not affect the performance of the drug since the development of the neurologic reactions indicates the drug has already done its job.

In some cases, surgical intervention is necessary but it is not needed in most circumstances (Sotelo et al., 2005). This is due to the reliability of the cysticidal drug therapy. However, one of the only surgical treatments is for hydrocephalus by ventriculo-peritoneal shunting which was previously described.

Discussion

Neurocysticercosis is a complex disease which has many complications and symptoms linked to it. As stated previously, all of these complications have some neurological impact on the affected person which can ultimately lead to the malfunction of the sensitive parts of the brain.

In order to avoid any of these problems, one must look toward the causes of the disease which can be preventative. The mechanism of infection starts with the entrance of the tapeworm through poorly prepared food in unsanitary conditions so that is the first place to address. Neurocysticercosis can be eradicated if the unsanitary conditions of the environment can be addressed. This was seen in the case of the disappearance of cysticercosis at the beginning of the 20th century in Europe, where it was endemic (Sotelo, 2011). During this time, medical advances were not significant enough to impact the infection rate. Instead, the sanitary conditions were improved which lead to a decrease in prevalence of neurocysticercosis. If the same principle was used in the endemic areas, the prevalence of the disease should decrease significantly. Education on the issue is also a big factor that could lead to the decrease in incidence of neurocysticercosis. If people in the endemic areas knew about the modes of transmission of the disease, the incidence should decrease.

Of course, increasing the sanitary conditions of the endemic areas will take some time and resources but it is not impossible. However, educating the residents on how to properly cook food and to maintain better hygiene will not require a lot of resources so eradication of the disease is still within reach over the next several decades.

Clinical problem solving: Neurocysticercosis

Patient: Kevin Brown

CC: The patient complains of recurring seizures.

HPI:

K.B. is a 32 year old male pig farmer who complains of spontaneous seizures, which have occurred twice within the past week. The most recent seizure was 2 days ago. His wife, who was with him during the examination, stated he fell to the ground with jerking movements and urinary incontinence for 45 seconds. Several bruises were noted. Additionally, he reported a severe headache for the past 3 weeks as well. The patient described it as being located more towards the right side of his cerebrum, and that it had progressively worsened.

He also complained of being unaware of his location (confusion) occasionally and felt nauseated several days of the week. He had been experiencing these symptoms for the past 3 weeks, but they have not been increasingly worsened.

The patient denied any exacerbating factors for the symptoms described above. No alleviating factors were noted as well besides aspirin which is described in detail below.

When asked if he had traveled out of the country, he said he went on a business trip to Mexico 4 months ago to observe the methodology that farmers utilize to raise their livestock.

It was difficult to verbally communicate with K.B. so he wrote his responses on paper. He had experienced slurred, slowed speech that was progressively intensifying for the last 3 days.

When asked, he denied any injuries to the body, particularly to the head, within the past 6 months.

PMH:

The patient denied any past medical history of hospitalization or surgery. He also denied any past medical history of seizures.

Family History:

The patient denied any familial diseases for his immediate family. He denied any epilepsy in his immediate family.

Social History:

He had neither smoked cigarettes nor drank alcohol in his entire life. He denied illicit drug use as well. He does not exercise but works on the ranch from sunrise to sunset which involves strenuous physical activities.

Medications:

He had been taking aspirin (600 mg of Bayer) once a day to alleviate pain caused by headaches. It was effective in reducing the amount of pain from a 6 to a 4 on a scale of 1-10, where 10 is extremely painful.

Allergies:

He denied having any allergies.

Vitals:**Height:** 5'11"**Weight:** 160 lbs**BP:** 129/81 mm Hg**Temp:** 98.8° F**HR:** 79 bpm**RR:** 15 breaths/minute**BMI:** 22.3**Pox:** 98%

Review of systems:

- General:** No weight change; **no fatigue or no weakness; no fever, chills, or night sweats; confused occasionally.**
- Skin:** No changes in skin, hair, or nails; no itching; no rashes; no sores, lumps, or moles.
- Head:** **No trauma; severe headache on the right side of head, daily; nauseated several times a week but no vomiting;** no visual changes.
- Eyes:** No glasses or contact lenses; no blurriness, tearing, or itching; no visual loss.
- Ears:** No hearing loss; no tinnitus; no vertigo; no discharge or earache.
- Nose, sinuses:** No rhinorrhea or no stuffiness; no sneezing, itching, or allergy; no epistaxis.
- Mouth, throat, neck:** No bleeding gums; no hoarseness, sore throat, or swollen neck.
- Urinary:** No frequent need to urinate; no hesitancy or urgency; no polyuria or dysuria; no hematuria, nocturia, or incontinence; no stones or infection.
- Vascular:** No leg edema; no claudication; no varicose veins; no thrombosis/emboli.
- Musculoskeletal:** **No muscular weakness;** no pain; no joint stiffness, full range of motion; no instability; no redness or swelling; no arthritis or gout.

Neurologic: No loss of sensation/numbness or tingling; no tremors; no weakness/paralysis; no fainting/blackouts; **two seizures within the past week.**

Endocrine: No heat/cold intolerance; no excessive sweating; no polyuria; no polydipsia or polyphagia; no thyroid problems; no diabetes.

Psychiatric: No changes in mood; no anxiety or depression.

Physical exam:

- HEENT:** Normocephalic, atraumatic, no masses, contusions or hematomas; pupils equal, round and reactive to light; extra-ocular movements intact; tonsils visualized with no exudates; no erythema present; tympanic membranes clear; no discharge or inflammation of the nose.
- Neck:** **Neck is supple with full range of motion; non-tender to palpation; no bruits were audible;** no lymphadenopathy; no thyromegaly.
- Back:** No paraspinal tenderness upon palpation; no costovertebral angle tenderness; **negative Kernig's and Brudzinski's signs.**
- Lungs:** Lungs are clear to auscultation bilaterally; no crackles, wheezing, or rhonchi audible.
- Chest:** Regular rate and rhythm. No murmurs, rubs, or gallops.
- Abdomen:** Nonprotuberant; Bowel sounds audible in all 4 quadrants; borborygmi present; Nontender to light and deep palpation; No organomegaly; no palpable masses; No aortic bruits noted.
- Extremities:** No pitting edema or cyanosis; normal capillary refill.
- Integument:** Warm and dry; no rashes.
- Neurological:** Cranial nerves: II – Visual acuity 20/30 in both eyes (not corrected); no abnormalities detected on fundoscopic exam; visual fields with no detected scotoma.
III, IV, VI – Extraocular movements intact; pupils equal, round, and reactive to light, accommodate.

V, VII, XII – Facial sensation is present in the V-I, II, III distribution; masseter strength intact; no dysarthria noted.

VIII – Hearing is intact (not corrected).

XI, X – Gag reflex present.

XI – Muscle strength present and is bilaterally equal.

XII – Protrudes tongue and waggles; no tongue deviation.

Motor: Normal muscle bulk with high definition; muscle strength with all muscle groups – 5/5.

Gait: Pt. could perform a tandem gait, walking on heels and toes; negative Romberg's test

Cerebellar: Pt. could perform finger-to-nose, heel-to-shin, and rapid alternating movements.

Deep Tendon Reflexes: 2+, brisk responses

Differential Diagnoses:

Neurocysticercosis*, stroke, brain tumor, mass or lesion, infection, encephalitis, meningitis, or multiple sclerosis

*most likely diagnosis

Labs:

A CBC and a CMP was ordered to verify the presence of infection through abnormal values. A spinal tap was also ordered to test for serum antibodies.

Complete Blood Count:

Red blood cell count: 4.6×10^9 cells/L

White blood cell count: 13.5×10^9 cells/L

Neutrophils 53%

Lymphocytes 33%

Monocytes 6%

Eosinophils 7%

Basophils 0.7%

Hemoglobin: 15.4g/dL

Hematocrit: 46.2%

Mean corpuscular volume: $100.4 \mu\text{m}^3$

Mean corpuscular hemoglobin: 33.5 pg

Comprehensive Metabolic Panel:

Glucose:	94.3 mg/dL
Calcium:	9.1 mg/dL
Albumin:	4.6 g/dL
Total protein:	7.1 g/dL
Sodium:	141 mEq/L
Potassium:	4.3 mEq/L
CO ₂ :	21 mmol/L
Chloride:	105 mmol/L
Blood urea nitrogen:	9.2 mg/dL
Creatinine:	1.1 mg/dL
Alkaline phosphatase:	97.2 IU/L
Alanine amino transferase:	21.1 IU/L
Aspartate amino transferase:	26.3 UI/L
Total bilirubin:	1.1 mg/dL

Spinal tap

A spinal tap showed a positive test for antibodies against cysticercosis.

A CT Scan and MRI were then performed to check for the presence of cysts within the cerebrum – as an infection was noted with the elevated leukocyte values seen in the CBC.

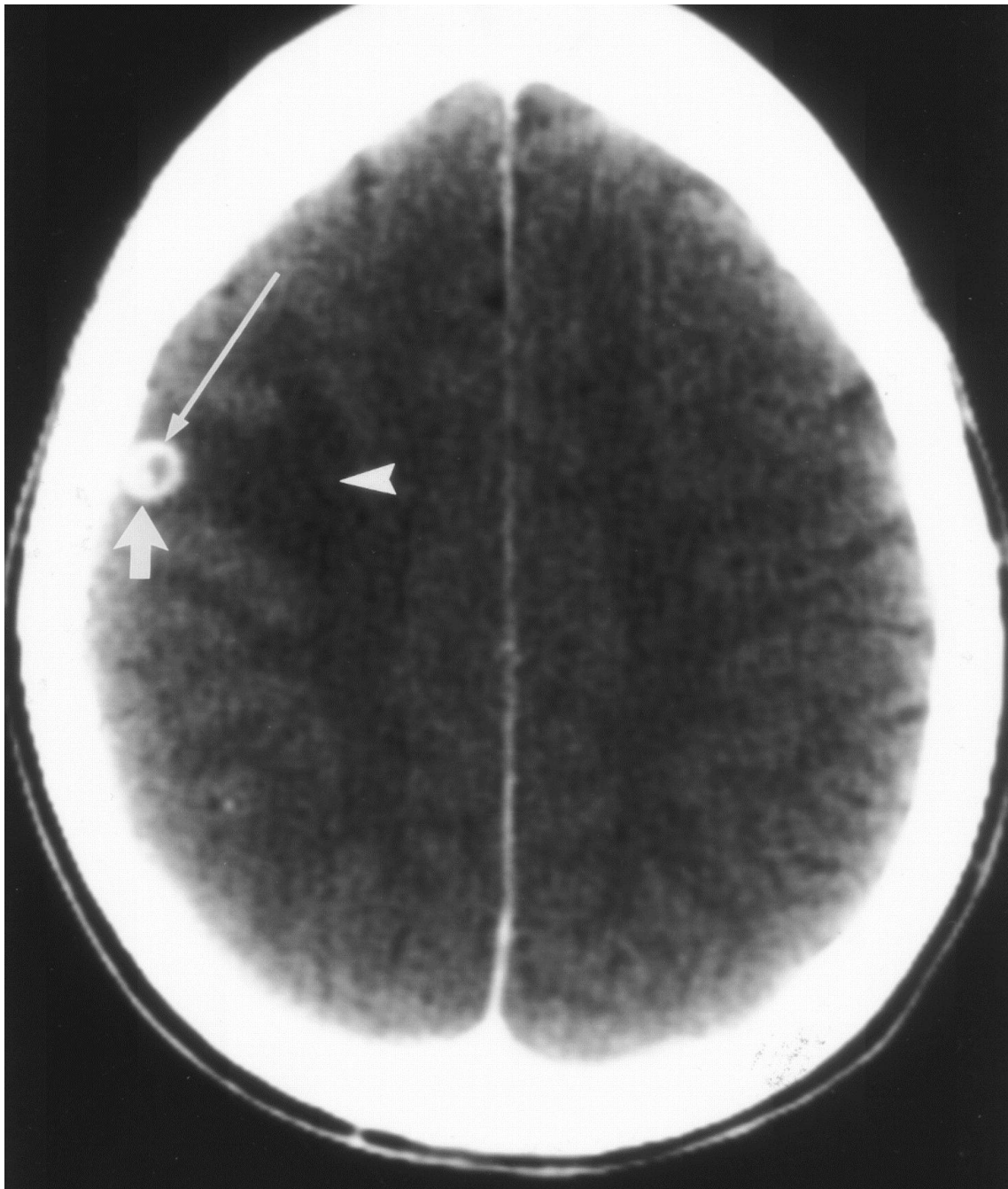


Figure 3. A CT scan of the patient confirms the existence of a cyst in the brain parenchyma. The arrows show a cyst ring forming while the arrowhead shows some edema in the area.

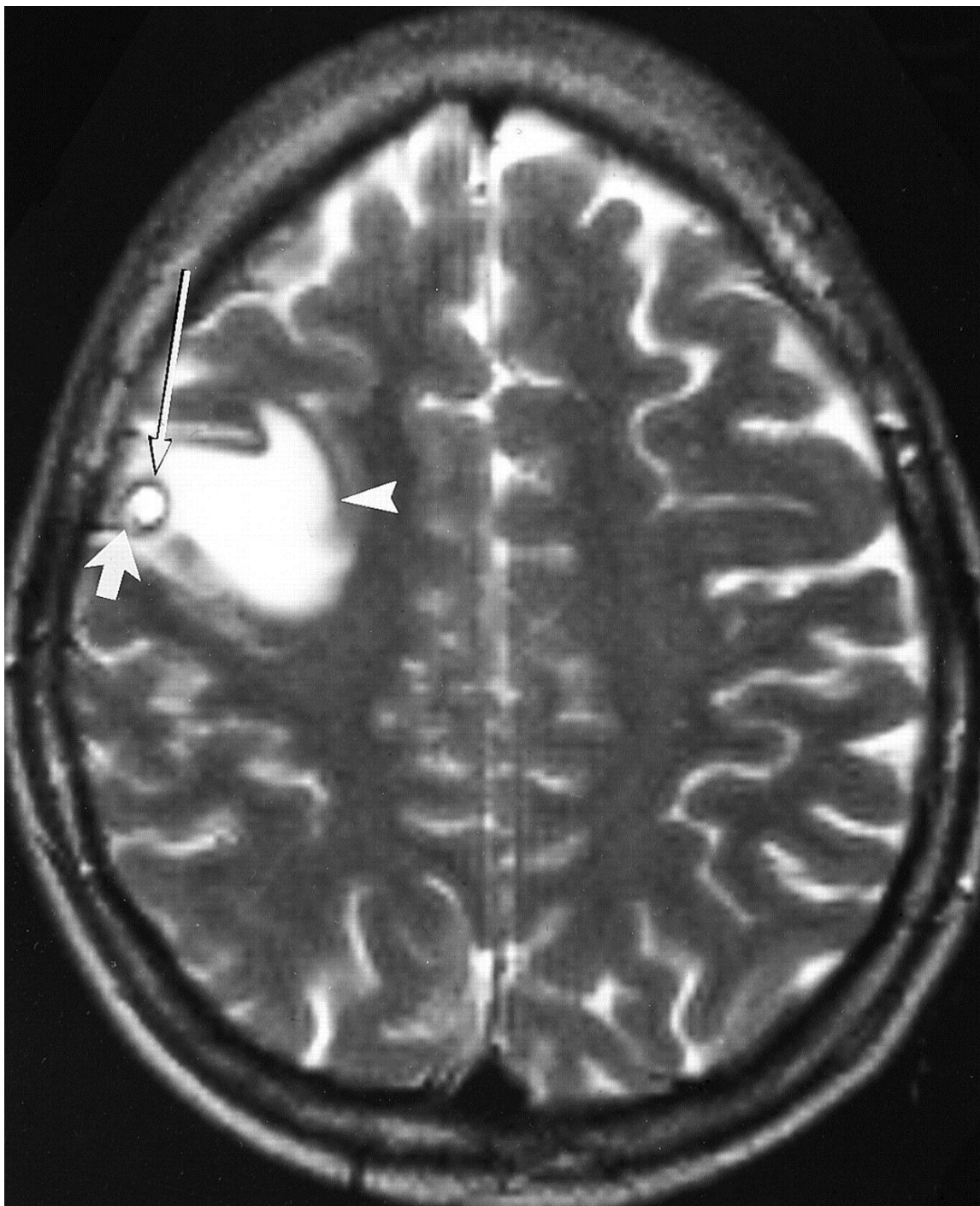


Figure 4. A postcontrast axial T1-weighted spin-echo MR image (600/14 [repetition time msec/echo time msec], 5 mm section thickness, 250 mm field of view, 250x256 matrix, acquisition time of 4 minutes, 9 seconds) was taken for the patient which also acknowledges the existence of a cyst in the brain parenchyma. The long arrow points to the emerging cyst while the short arrow points to a thickened portion of the cyst. The arrowhead points to vasogenic edema.

Assessment:

Based on the patient's history of present and past medical illness, an initial diagnosis of neurocysticercosis was suspected. The patient's presentation of seizures paired with his occupation and travel out of the country to Mexico pointed towards a parasitic infection most likely in the brain. The slowed, slurred speech pointed towards of a mass or lesion located in the brain especially when the all of the cranial nerves were found to be intact.

A review of systems and a physical exam also pointed out the absence of abnormalities in the neurological function of the patient. The cranial nerves were all intact and functional which meant the speech impairment was due to an abnormality in the brain rather than an abnormality in nerve input. In addition, Kernig's and Brudzinski's signs were found to be negative, thus decreasing the chance of meningitis. The neck was supple upon palpation with full range of motion, further decreasing the chance of meningitis. The presence of brisk reflexes and full, bilaterally equal muscle strength decreases the chance of stroke, encephalitis, and multiple sclerosis as the diagnosis. Further analysis of lab tests and brain scans would help determine the cause of the symptoms.

A complete blood count and comprehensive metabolic panel were ordered. The CBC indicated an elevated level of white blood cells with a high level of eosinophils. This suggested a parasitic infection in the patient. This could also be due to an allergic reaction. However, the patient denied having any allergies. The CMP showed no signs of abnormal values which suggest the cause of the patient's symptoms were most likely due to an infection or lesion.

A CT scan and MRI were ordered which showed signs of a cyst in the parenchyma of the brain, confirming the initial suspected diagnosis of neurocysticercosis. It was located near Broca's area. Broca's aphasia was observed when the patient was not able to speak while able to write. Edema was also noted on the CT scan as well as the MRI. The swelling would account for all of the symptoms presented by the patient and a spinal tap showed an infection due to neurocysticercosis.

The patient is diagnosed with neurocysticercosis.

Plan of treatment:

A drug therapy approach was preferable for this patient since it was non-invasive and was seen to be effective from many clinical trials in the past years.

For neurocysticercosis, the patient was given an anti-parasitic, an anti-inflammatory, and an anticonvulsant drug. The anti-parasitic drug Albendazole was prescribed since it was cheaper and more effective in the presence of steroids than Praziquantel. Steroids were prescribed to decrease the amount of inflammation caused by the combination of Albendazole and the immune system eliminating and clearing the infection. An anticonvulsant was also prescribed to prevent the occurrence seizures. The specific drugs and dosages are as follows:

- Albendazole: 400mg, orally twice a day with meals
- Dexamethasone: 4mg, orally twice a day
- Carbamazepine: 200mg, orally twice a day

References

- Escobedo, F., Penagos, P., Rodriguez, J., & Sotelo, J. (1987). Albendazole therapy for neurocysticercosis. *Archives of Internal Medicine*, *147*(4), 738-741. doi: 10.1001/archinte.147.4.738
- Garcia, H. H., Lescano, A. G., Lanchote, V. L., Pretell, E. J., Gonzales, I., Bustos, J. A., . . . Cysticercosis Working Grp, P. (2011). Pharmacokinetics of combined treatment with praziquantel and albendazole in neurocysticercosis. *British Journal of Clinical Pharmacology*, *72*(1), 77-84. doi: 10.1111/j.1365-2125.2011.03945.x
- Jayaraman, T., Prabhakaran, V., Babu, P., Raghava, M. V., Rajshekhar, V., Dorny, P., . . . Oommen, A. (2011). Relative seroprevalence of cysticercus antigens and antibodies and antibodies to *Taenia ova* in a population sample in south India suggests immunity against neurocysticercosis. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, *105*(3), 153-159. doi: 10.1016/j.trstmh.2010.10.007
- Kelesidis, T., & Tsiodras, S. (2011). Hypopituitarism caused by neurocysticercosis. *American Journal of the Medical Sciences*, *341*(5), 414-416. doi: 10.1097/MAJ.0b013e3182056438
- Mahanty, S., Paredes, A., Marzal, M., Gonzalez, E., Rodriguez, S., Dorny, P., . . . Nash, T. (2011). Sensitive in vitro system to assess morphological and biochemical effects of praziquantel and albendazole on *Taenia solium* cysts. *Antimicrobial Agents and Chemotherapy*, *55*(1), 211-217. doi: 10.1128/aac.00761-10
- Maxwell, Robert W. *Maxwell Quick Medical Reference*. 5th ed. Maxwell, 2006. Print.

MDConsult. Brain tumors, benign. <http://0->

www.mdconsult.com.innopac.library.unr.edu/das/pdxmd/body/329095189-7/1294989708?type=med&eid=9-u1.0-_1_mt_1016551.

Last updated: June 2011. Accessed: 9 April 2012.

MDConsult. Encephalitis. <http://0->

www.mdconsult.com.innopac.library.unr.edu/das/pdxmd/body/329095189-5/1294989128?type=med&eid=9-u1.0-_1_mt_1014457.

Last updated: Aug. 2011. Accessed: 9 April 2012.

MDConsult. Epilepsy in adults. <http://0->

www.mdconsult.com.innopac.library.unr.edu/das/pdxmd/body/329095189-8/1294998007?type=med&eid=9-u1.0-_1_mt_1016545.

Last Updated: Sep. 2011. Accessed: 9 April 2012.

MDConsult. Multiple sclerosis. <http://0->

www.mdconsult.com.innopac.library.unr.edu/das/pdxmd/body/329095189-6/1294989487?type=med&eid=9-u1.0-_1_mt_1014469.

Last updated: Jan. 2011. Accessed: 9 April 2012.

MDConsult. Stroke. <http://0->

www.mdconsult.com.innopac.library.unr.edu/das/pdxmd/body/329095189-4/1294988943?type=med&eid=9-u1.0-_1_mt_1014487.

Last updated: Mar. 2011. Accessed: 9 April 2012.

MDConsult. Viral meningitis. <http://0->

www.mdconsult.com.innopac.library.unr.edu/das/pdxmd/body/329095189-3/1294988387?type=med&eid=9-u1.0-_1_mt_1014562.

Last updated: Nov. 2011. Accessed: 9 April 2012.

- Praet, N., Speybroeck, N., Rodriguez-Hidalgo, R., Benitez-Ortiz, W., Berkvens, D., Brandt, J., . . . Dorny, P. (2010). Age-related infection and transmission patterns of human cysticercosis. *International Journal for Parasitology*, *40*(1), 85-90. doi: 10.1016/j.ijpara.2009.07.007
- Rajshekhar, V. (2010). Surgical management of neurocysticercosis. *International Journal of Surgery*, *8*(2), 100-104. doi: 10.1016/j.ijsu.2009.12.006
- Salgado, P., Rojas, R., & Sotelo, J. (1997). Cysticercosis - clinical classification based on imaging studies. *Archives of Internal Medicine*, *157*(17), 1991-1997. doi: 10.1001/archinte.157.17.1991
- Sinha, S., & Sharma, B. S. (2009). Neurocysticercosis: a review of current status and management. *Journal of Clinical Neuroscience*, *16*(7), 867-876. doi: 10.1016/j.jocn.2008.10.030
- Sotelo, J. (2003). Neurocysticercosis - eradication of cysticercosis is an attainable goal. *British Medical Journal*, *326*(7388), 511-512. doi: 10.1136/bmj.326.7388.511
- Sotelo, J. (2004). Neurocysticercosis - is the elimination of parasites beneficial? *New England Journal of Medicine*, *350*(3), 280-282. doi: 10.1056/NEJMe038218
- Sotelo, J. (2011). Clinical manifestations, diagnosis, and treatment of neurocysticercosis. *Current Neurology and Neuroscience Reports*, *11*(6), 529-535. doi: 10.1007/s11910-011-0226-7
- Sotelo, J., Arriada, N., & Lopez, M. A. (2005). Ventriculoperitoneal shunt of continuous flow vs valvular shunt for treatment of hydrocephalus in adults. *Surgical Neurology*, *63*(3), 197-203. doi: 10.1016/j.surneu.2004.07.040

- Sotelo, J., & Diaz-Olavarrieta, C. (2010). Neurocysticercosis: changes after 25 years of medical therapy. *Archives of Medical Research*, *41*(1), 62-63. doi: 10.1016/j.arcmed.2009.12.003
- Sotelo, J., Escobedo, F., Rodriguezcarbajal, J., Torres, B., & Rubiodonnadieu, F. (1984). Therapy of parenchymal brain cysticercosis with praziquantel. *New England Journal of Medicine*, *310*(16), 1001-1007. doi: 10.1056/nejm198404193101601
- Uddin, J., Gonzalez, A. E., Gilman, R. H., Thomas, L. H., Rodriguez, S., Evans, C. A. W., . . . Friedland, J. S. (2010). Mechanisms regulating monocyte CXCL8 secretion in neurocysticercosis and the effect of antiparasitic therapy. *Journal of Immunology*, *185*(7), 4478-4484. doi: 10.4049/jimmunol.0904158