Neurocysticercosis: A Major Cause of Neurological Disease Worldwide

A. Clinton White, Jr.

Neurocysticercosis is the most likely reason for epilepsy being twice as common in developing as in developed countries of the world [1].

Neurocysticercosis, which is caused by infestation of the human CNS with the tissue cysts of *Taenia solium*, is arguably the most common parasitic disease of the human nervous system. While most of the details of the parasite’s life cycle were known by the nineteenth century and most of the clinical manifestations of *T. solium* infection had been identified by the mid-twentieth century, concepts regarding the prevalence of infection, associated morbidity and mortality, treatment, and epidemiology have changed dramatically over the past 10 years.

The increased ease of international travel, the increasing numbers of immigrants from developing countries, and the widespread use of improved diagnostic techniques have led to widespread recognition of neurocysticercosis as a common infection not only in developing countries but also in the United States. Large case series have been reported in the southwestern United States and other areas with large immigrant populations from Latin America [2–7].

Since neurocysticercosis is not a reportable disease, the total number of cases diagnosed in the United States is not known but is estimated at >1,000 new cases each year [2], and this number appears to be increasing. While most of these cases occur in patients infected in developing countries, the number of locally acquired infections is also increasing.

The ancient Greeks recognized that intestinal tapeworms were associated with ingestion of meat. They referred to the “hailstones” in meat as cysticerci, meaning bladder-tails. During the nineteenth century, Leuckart identified the complete life cycle of *T. solium* and noted that human cysticercosis resulted from ingestion of parasite eggs shed by tapeworm carriers. Neurocysticercosis was a frequent finding in autopsy series reported from Europe in the nineteenth century. Studies from the early twentieth century elucidated most of the clinical manifestations of neurocysticercosis [8]; however, few clinical cases were diagnosed before death until the 1970s. Thus, neurocysticercosis was often regarded as a somewhat obscure “tropical” disease.

Within the past 20 years, three developments have led to the recognition of neurocysticercosis as a common cause of neurological disease worldwide. First, the development of computerized neuroimaging studies—CT and, subsequently, MRI—provided the first sensitive, noninvasive techniques for antemortem diagnosis of neurocysticercosis. The influx of rural immigrants from developing countries into the United States, which occurred during the 1970s and 1980s, resulted in the use of these studies in high-risk populations. Since the early 1980s, numerous case series of patients with cysticercosis have been reported from the United States [2–7].

Second, the development of highly specific serological tests provided a mechanism for performing accurate epidemiological studies [9, 10]. Specific serological tests and neuroimaging studies have subsequently been used in epidemiological studies in countries where the infection is endemic. This has resulted in the third development, which is the increasing recognition of the high prevalence of neurocysticercosis worldwide. In addition, studies have identified an increasing variety of clinical manifestations of the infection.

The Parasite and Its Life Cycle

In the usual cycle of transmission, humans acquire intestinal infection (taeniasis, or tapeworm infection) by ingestion of undercooked pork infected with *T. solium* cysticerci. In the intestine, the protoscolex evaginates and attaches to the intestinal wall by means of suckers and hooks. The adult worm develops in the small intestine by forming segments (proglottids) that arise from the caudal end of the scolex. The proglottids gradually enlarge and mature as they are separated from the scolex by newly produced segments. As they mature, the proglottids form testes and ovaries. The eggs are fertilized within the proglottids.
Each terminal proglottid contains ~50,000 eggs. The eggs are intermittently extruded from the proglottid into the intestine, or the entire proglottid may be shed in the feces. Most patients are asymptomatic. However, they may note the passage of proglottids, which are opaque, off-white in color, and ~1–2 cm long, ~1 cm wide, and 2–3 mm thick. Since excretion of the proglottids is intermittent, stool studies from patients with active tapeworm infections are commonly negative for parasite ova.

The usual intermediate host, the pig, is infected by ingestion of parasite eggs or proglottids in human feces. Thus, porcine infection is limited to areas where animal husbandry practices are such that pigs come into contact with human feces. The eggs are induced to hatch and activate by the action of gastric and intestinal fluids. The hatched larvae, also called the oncospheres, escape from the eggs, attach to the intestines via motile hooks, and penetrate the intestinal mucosa and the vessels in the submucosa. Penetration appears to be facilitated by excretory proteases produced by the oncospheres [11].

After invasion, the oncospheres migrate via the bloodstream throughout the body of the intermediate host. It is not known whether the oncospheres actively migrate to specific tissue or merely passively lodge in the tissues with high blood flow (e.g., the muscles or the brain). Over a period of 3 weeks–2 months, the oncospheres enlarge and mature into cysticerci. The cysts appear in the muscles as translucent, thin-walled cysts that are ~1 cm in diameter and have an eccentric white nodule containing the invaginated scolex. The life cycle of the parasite is completed when humans consume undercooked pork containing the cysts.

Humans can also be infected by ingestion of T. solium eggs. The eggs are sticky and can be found attached to the perianal skin and even under the fingernails of tapeworm carriers; they are likely transmitted by either direct contact with a tapeworm carrier or in food prepared by the carrier. After ingestion of the eggs, the oncospheres are released, penetrate the intestinal mucosa, and migrate throughout the body to produce human cysticercosis. Larval cysts are occasionally found in nearly every tissue, but the larvae do not usually develop into mature cysts in most tissues. Thus, most cysts are found in the CNS, skeletal muscle, subcutaneous tissue, and the eyes.

Pathology and Pathogenesis

T. solium lives in tissues as a fluid-filled cyst (or metacestode). The cyst has a thin, semitransparent wall. The scolex is invaginated and appears as an opaque nodule, 4–5 mm in diameter, on one side of the cyst. The size and shape of the cyst vary with pressure from surrounding tissue. In the brain, the cysts are round, with a diameter of ~1 cm [12]. There may be a surrounding capsule of variable thickness that consists of astrocytes and collagen fibers, but the capsule is usually less thick in the CNS and eye. The bladder wall consists of three layers: a cuticle layer containing microtriches (which are in turn coated by a carbohydrate glycocalyx), a layer of pseudoepithelium and muscularis, and a loose layer of connective tissue containing a network of canaliculi. The mural nodule contains the invaginated scolex and an associated spiral canal, also with a trilaminar membrane. A small excretory pore near the end of the spiral canal connects the digestive canal to the surrounding tissue.

Viable cysticerci (obtained from pig muscle) have little surrounding host inflammation—usually only a few mononuclear cells and variable numbers of eosinophils near the excretory pore [13]. To complete its life cycle, the cysticercus must survive in the pig muscle for weeks to months. Thus, it is not surprising that the cysts have developed elaborate mechanisms for evading the host response [14, 15]. Animals actively infected or previously infected with the cyst stage are immune to reinfection with oncospheres. This immunity is mediated by antibody and complement [16, 17]. However, in natural infection, the antibody response develops only after the parasites have transformed into the more resistant metacestode form.

The metacestodes have elaborate means of evading complement-mediated destruction. Parasite paramyosin binds to C1q and inhibits the classic pathway of complement activation [18]. The parasites also secrete a serine protease inhibitor termed taeniaestatin, which inhibits both classic and alternate pathways of complement activation, interferes with leukocyte chemotaxis, and inhibits cytokine production [19, 20]. Sulfated polysaccharides, which coat the cyst wall, activate complement away from the parasite, decrease complement deposition, and limit access of inflammatory cells to the parasites [21]. Similarly, antibody does not seem to be able to kill the mature metacestode. Viable cysts actually stimulate production of cytokines involved in immunoglobulin production, and immunoglobulin is taken up by the cyst, perhaps as a source of protein [22].

In contrast, the cellular immune response is suppressed. Taeniaestatin and other parasite molecules interfere with lymphocyte proliferation and macrophage function. Symptoms of neurocysticercosis are typically associated with a granulomatous response that may result when the cysts are no longer able to modulate the host response. Preliminary evidence suggests that the granulomatous response is mediated via T helper 1 cytokines such as IL-2 and IFN-γ (P. Robinson, R. Atmar, D. Lewis, and A. C. White, unpublished data).

The clinical manifestations of human cysticercosis depend on the location and number of cysts and on the host response. If only a few lesions are located in nonstrategic areas of the body such as the muscle or certain portions of the brain, infection may be asymptomatic. Even in cases of neurological disease, there is usually an asymptomatic period of several years before the onset of symptoms [7, 8]. This incubation period approximates the estimated life span of the tissue cyst [23].

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The histopathology of cysts in humans without symptoms of cysticercosis who died of other causes resembles that observed for viable cysts in pigs [12, 24]. In contrast, most cysts from patients with symptomatic disease are associated with a prominent inflammatory response including lymphocytes, eosinophils, granulocytes, and plasma cells [12]. Thus, the symptoms of parenchymal cysticercosis appear to result from inflammation, which develops when the cyst loses its ability to modulate the host response.

The changes associated with this inflammation progress through a series of stages [23]. In the colloidial stage, the appearance of the cyst is similar to a colloid cyst with gelatinous material in the cyst fluid and hyaline degeneration of the larva. In the granular-nodular stage, the cyst begins to contract, and the walls are replaced by focal lymphoid nodules and necrosis. Finally, the granulation tissue is replaced by collagenous structures and calcification in the nodular-calcified stage [23].

The major manifestation of extraparenchymal neurocysticercosis is hydrocephalus [25]. Cysts in the ventricles may cause a mechanical obstruction of CSF flow, leading to noncommunicating hydrocephalus [26]. Ventricular flow can also be obstructed when granular ependymitis develops, especially at the aqueduct of Sylvia. Cisternal and subarachnoid cysts are often associated with arachnoiditis, which can lead to obstructed CSF outflow and communicating hydrocephalus [26].

**Epidemiology**

Before the 1990s, reliable epidemiological data on the prevalence of neurocysticercosis were limited. Neuroimaging studies, biopsy, or autopsy studies, which were needed to confirm the diagnosis, were not readily available in areas where the infection is endemic. Serological assays performed with unfractionated antigens were both insensitive and nonspecific [27, 28]. In 1989, Tsang and colleagues [9] reported the development of the enzyme-linked immunotransfer blot (EITB) assay, which is performed with parasite glycoproteins. EITB was the first specific assay for *T. solium* infection that could be used in large field studies. With the increasing availability of neuroimaging studies in areas of endemicity and the use of the EITB assay in epidemiological studies, it has become clear that the global morbidity and mortality associated with cysticercosis have likely been grossly underestimated [10].

There are few reliable data on the prevalence of intestinal taeniasis due to *T. solium*. Estimates based on stool studies suggest that 4 million people worldwide harbor the porcine tapeworm [29]. More-recent studies, which have included antigen-detection assays and collection of posttreatment stool samples, have shown that stool examination is an insensitive method of identifying tapeworm carriers [30]. For each patient with a tapeworm, there are thought to be 10 or more persons infected with the cyst stage of the parasite. It has been estimated that ~50 million persons are infected with the cyst stage [31], but this figure is likely also an underestimate [10].

The geographic distribution of cysticercosis is wide, with areas of high prevalence in Mexico, Central and South America, India, and sub-Saharan Africa. Autopsy studies in Mexico have shown that at least one cysticercus has been detected in the CNS in ~2% (range, 0.4%-3.5%) of unselected autopsies [32]. In recent studies in which the EITB assay was performed, seroprevalence rates of 4.9%-22.6% were found among inhabitants of villages in Latin America where the infection is endemic [10, 27, 33, 34]. In contrast to these findings from rural areas, only 1.5% of patients in Lima, Peru, who were referred for endoscopy had evidence of cysticercosis [35]. Furthermore, studies from Mexico, Ecuador, and Peru have all demonstrated that up to half of all patients with evidence of neurocysticercosis on CT scans will have negative EITB assays [27, 36, 37].

Medina and colleagues [38] identified evidence of neurocysticercosis in 50 of 100 consecutive patients in Mexico City who had adult-onset seizures and were examined with use of CT. Similarly, neurocysticercosis was the most common cause of adult-onset seizures in a study from Ecuador [39]. Eighteen percent of patients referred to neurologists in Lima had positive EITB assays [40]. Up to half of all pigs in villages where cysticercosis is endemic are infected [10]. Most studies from Latin America demonstrate higher prevalence among residents of rural areas, especially those areas where pigs are raised [40].

The prevalence of neurocysticercosis in pig-raising areas of Africa is similar to that in Latin America. Between 0.45% and 7% of autopsies in these areas reveal evidence of neurocysticercosis [41, 42]. In a large epidemiological study from Togo (West Africa), 2.4% of the population, including 39% of the patients with epilepsy, evidenced cysticercosis [43]. In large case series of black South African patients with epilepsy, 28%–38% had evidence of neurocysticercosis on CT scans [44, 45]. Twenty-one percent of epileptics in Rwanda were found to be EITB positive for *T. solium* [10]. Furthermore, cysticercosis is highly prevalent among pigs in all pig-raising areas of Africa [42].

Neurocysticercosis is also a common cause of neurological disease in India. Up to half of patients with seizure disorders have serological evidence of neurocysticercosis [10, 41]. Gulati and colleagues [46] demonstrated that 24% of 361 epileptic patients in Delhi had unequivocal evidence of neurocysticercosis on MRI scans. Additional patients had evidence of resolving lesions on their scans. Examination of brain biopsy specimens from similar patients elsewhere in India has shown that nearly all of these resolving lesions are due to neurocysticercosis [47].

Few data are available on the prevalence of cysticercosis in China. Large case series of patients with neurocysticercosis continue to be described, with seroprevalence rates of up to 16% in selected populations [48]. Nearly 10% of patients who
had neurological conditions in Beijing were found to be EITB positive [10]. In a nationwide survey of parasitic infections in China, a high prevalence of cysticercosis was identified in 27 of 30 provinces, autonomous zones, and/or municipalities, but some areas were apparently spared [49]. In this survey, the investigators indicated that neurocysticercosis is among the more common serious parasitic diseases, with nearly 9,000 cases identified [49]. Neurocysticercosis is also common elsewhere in Asia, including Bali and Korea, where a large proportion of epileptics have the disease.

Because of the recent influx of immigrants to the United States, neurocysticercosis is being increasingly recognized as a problem here as well [2–4, 6, 7, 50]. Most of the cases occur among Hispanic immigrants—especially Mexican immigrants—who are presumed to have been infected before immigration. There have also been a number of cases in immigrants from Asia, especially Korea [50]. In addition, locally acquired cases have been documented in New York, Chicago, Los Angeles, and elsewhere in the United States [50, 51]. For example, 1.3% of the members of an Orthodox Jewish community in New York City were found to have positive EITB assays [52]; seropositivity was associated with the presence of domestic employees who were from regions of endemicity [51, 52].

Since cysticercosis is not reportable in the United States, except in Southern California, there are no clear figures on the national prevalence of infection. However, on the basis of the number of cases seen at my institution, the number of positive serological tests performed at the Centers for Disease Control and Prevention, and the data on the epidemiology of the disease in Southern California, my colleagues and I have estimated that there are likely >1,000 cases diagnosed each year in the United States [2].

### Clinical Manifestations

Neurocysticercosis has a wide variety of clinical manifestations. Seizures are by far the most common clinical manifestation and occur in 70%–90% of cases (table 1). Less common manifestations include headache, symptoms of elevated intracranial pressure (especially nausea and vomiting), and altered mental status (including psychosis). Only a minority of patients present with cranial nerve palsies or other focal findings.

The clinical spectrum of the disease depends on the location, number, and viability of the cyst(s) as well as host response. Cyst viability and location and the degree of inflammation are also important factors in pathogenesis, sensitivity of serodiagnostic tests, response to treatment, and prognosis. A classification scheme that has been used at our institution is illustrated in table 2. My colleagues and I classify patients on the basis of whether they have active infection (with either viable or dying parasites) or inactive infection (with residual parasites from a prior infection) [2]. The active infections can then be classified on the basis of the location of the cysts (e.g., parenchymal vs. extraparenchymal; the latter location is subcategorized as ventricular, cisternal, ophthalmic, or spinal).

Finally, the viability of the cyst can be assessed based on the degree of inflammation, since viable cysts are able to suppress the host inflammatory response. Patients will frequently present with cysts in multiple locations and in varying stages.

### Inactive Infection

A substantial portion of patients with neurocysticercosis present clinically with the residua of prior active infection. Intraparenchymal calcification can usually be seen on the brain CT scans of these patients. Plain films and MRI scans may also show calcification, but CT is more sensitive. Most patients present with seizures, which are usually generalized. On questioning, most of the patients will provide a history of prior episodes of seizures that often occurred several years before the diagnosis. Another common manifestation is headache.

### Table 1. Symptoms associated with neurocysticercosis in 159 patients evaluated at Ben Taub General Hospital, Houston.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No. of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seizures</td>
<td>127 (79)</td>
</tr>
<tr>
<td>Headache</td>
<td>66 (41)</td>
</tr>
<tr>
<td>Visual problems</td>
<td>27 (17)</td>
</tr>
<tr>
<td>Confusion</td>
<td>25 (16)</td>
</tr>
<tr>
<td>Ataxia</td>
<td>10 (6)</td>
</tr>
<tr>
<td>Symptoms of hydrocephalus*</td>
<td>17 (11)</td>
</tr>
<tr>
<td>Psychosis</td>
<td>9 (6)</td>
</tr>
</tbody>
</table>

* Symptoms of hydrocephalus include nausea, vomiting, and headaches.

### Table 2. Classification scheme for patients with neurocysticercosis, based on the presence of active disease or inactive disease (calcification or hydrocephalus from prior infection), the location of the cyst, and the degree of host inflammation.

<table>
<thead>
<tr>
<th>Category</th>
<th>Approximate percentage of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inactive disease</td>
<td></td>
</tr>
<tr>
<td>Calcifications alone</td>
<td>10%–40%</td>
</tr>
<tr>
<td>Calcification and hydrocephalus</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Active disease</td>
<td></td>
</tr>
<tr>
<td>Parenchymal cysts</td>
<td></td>
</tr>
<tr>
<td>With edema or enhancement</td>
<td>50%–80%</td>
</tr>
<tr>
<td>Without edema or enhancement</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Ventricular cysts</td>
<td>10%–20%</td>
</tr>
<tr>
<td>Subarachnoid cysts</td>
<td>5%–10%</td>
</tr>
<tr>
<td>Spinal cysts</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Ocular cysts</td>
<td>&lt;1%</td>
</tr>
</tbody>
</table>

NOTE. Patients are grouped according to the most active form of infection or least common site of infection; the approximate percentages of cases are based on large case series from the United States.
smaller number of patients present with altered mental status or psychosis that is perhaps related to anoxic injury from prior seizures.

Pathological studies show that calcifications correspond to the nodular-calcified stage of the disease, with calcified granulomas and scarring. Thus, seizures have been attributed to scarring that results in chronic epileptiform foci. A surprising finding for many of these patients is focal edema or enhancement on imaging studies, and CSF study results will also be abnormal in many cases [2]. Thus, acute inflammation may also play a role in the pathogenesis of seizures, perhaps from the intermittent release of antigens from the site of prior infection. Diagnosis depends primarily on CT findings, since the serological tests for many of these patients will be negative [27, 36].

A small minority of patients present with chronic hydrocephalus in the absence of active infection. Multiple parenchymal calcifications are usually apparent on CT scans, suggesting prior infection with multiple cysts. The hydrocephalus is thought to result from the obstruction of CSF outflow secondary to prior arachnoiditis or from obstructive hydrocephalus secondary to granular ependymitis with scarring (typically presenting with aqueductal stenosis) [26]. These patients usually present with symptoms of hydrocephalus (e.g., headache, nausea, vomiting, altered mental status, and disequilibrium) that are of gradual onset. Some may present with symptoms such as seizures that are related to coexisting parenchymal disease.

**Active Infection**

*Active parenchymal neurocysticercosis.* This is the most common form of disease, representing >60% of cases in most recent series. The vast majority of patients with active parenchymal disease present with seizures. Seizures are usually generalized or focal with secondary generalization [39, 53]. One-third of patients have simple partial seizures. A small minority develop partial complex seizures. Other symptoms include headaches and confusion. The neurological examination usually reveals nonfocal findings, but a minority will present with focal neurological abnormalities. Studies of CSF may reveal elevation of the protein level or pleocytosis, but the results are generally nonspecific and only mildly abnormal. The proportion of the WBCs that are eosinophils ranges from none to the majority of cells. The results of electroencephalographic studies usually are either normal or indicate nonspecific abnormalities.

Natural history studies of patients with parenchymal cysticercosis have revealed that patients do well even when they do not receive antiparasitic drugs [5, 54]. Studies from the United States have shown that seizures are generally well controlled with a single anticonvulsant. Neuroimaging studies reveal that cysts usually resolve within 2 years. When my coworkers and I carefully reviewed neuroimaging studies of patients from our center, we identified evidence of an inflammatory response to the parasites (edema surrounding the parasites, enhancement of either the cyst wall or the surrounding brain parenchyma, or calcifications from prior acute infection) in all patients with symptomatic disease (figure 1) [2]. This finding contrasts with the appearance of cysts in patients who undergo neuroimaging studies for conditions other than neurocysticercosis; in this case, there is no edema or enhancement (figure 2) [55]. Thus, most, if not all, patients with symptomatic parenchymal neurocysticercosis are infected with parasites that have lost their ability to suppress the host response and are in the process of dying.

Investigators, primarily from Latin America, have argued that there is a subgroup of patients who present with symptomatic parenchymal cysticercosis without evidence of an inflammatory response [56–59]. These researchers have assumed that the cysts will not resolve without antiparasitic therapy and that any response to therapy represents a clinical response to antiparasitic drugs. This assumption contradicts the uniform findings of autopsy studies, in which symptomatic disease has invariably been associated with an inflammatory infiltrate or scarring [12, 24].

![Figure 1. Contrast-enhanced CT scan of the brain of an 11-year-old boy from India who presented with a recurrence of seizures; a cyst in the right parietal lobe, with enhancement of the cyst wall and surrounding brain, as well as edema are apparent. Reprinted with permission from Medicine [2].](image-url)
Furthermore, investigators have not always performed contrast studies on their patients, have rarely assessed patients by means of contrast MRI (which is more sensitive than CT for showing inflammation in the host [7]), and have not uniformly included pericystic edema as evidence of inflammation. Thus, cases of symptomatic parenchymal cysticercosis in the absence of an inflammatory response are extremely rare, if they occur at all. In addition, natural history study findings suggest that most of these lesions will resolve within a period of a few years, even in the absence of antiparasitic therapy.

Some patients with parenchymal cysticercosis have prominent pleocytosis. While cysts may not be visible outside the parenchyma on neuroimaging studies, I believe that most of these patients responded as did patients with cysts in the subarachnoid space or basilar cisterns.

Cysticercal encephalitis. Another variant of parenchymal disease is the presentation referred to as cysticercal encephalitis. In this form of the disease, patients (primarily young girls) present with numerous parenchymal cysts and an associated inflammatory response and elevated intracranial pressure secondary to diffuse cerebral edema [60]. If these patients are not treated, they are at high risk of developing severe neurological sequelae.

The key to therapy is aggressive management of hydrocephalus. Since hydrocephalus is primarily a response to inflammation, high doses of corticosteroids (e.g., dexamethasone at a dose of 24–32 mg/d) are the primary initial therapy. Some patients may require osmotic diuresis or even decompressive craniotomy in cases of immediately life-threatening elevated intracranial pressure. Antiparasitic drugs should not be given until the elevated intracranial pressure has resolved.

Extraparenchymal Neurocysticercosis

Ventricular neurocysticercosis. About 10% to 20% of patients with cysticercosis present primarily with cysts in the ventricles. In about 20% of cases, patients will present with seizures, which are invariably associated with coexisting parenchymal disease (either active cysts or calcifications from inactive disease). Another 20% of patients also have cisternal disease and may present with symptoms of meningeal inflammation.

Most patients with ventricular cysticercosis present with symptoms of obstructive hydrocephalus, including headache, nausea, vomiting, dysequilibrium, and altered mental status. The onset of these symptoms is quite variable. For example, some patients’ mental status deteriorates rapidly while they are in the emergency department for evaluation of headache, while others experience the onset of symptoms gradually over a period of years [2].

The main pathological event appears to be the mechanical obstruction of CSF flow, either by the cyst itself or by accompanying inflammation [26]. Obstruction is most likely to occur at sites such as the foramina of Luschka or Magendie or the aqueduct of Sylvia, where the CSF passages have narrowed. In the absence of significant inflammation, cysts in the ventricles are mobile and thus symptoms can be intermittent or may vary with positioning of the patient’s head. Cysts are most commonly identified in the fourth ventricle (figure 3). In recent cases in which MRIs were performed, cysts were also frequently identified in the third and lateral ventricles.

Subarachnoid (cisternal) cysticercosis. In addition to the seizures associated with coexisting parenchymal cysts and to obstructive hydrocephalus due to ventricular cysts, patients with cysts in the basilar cisterns may present with basilar arachnoiditis. In some cases, patients will present with meningeal signs and CSF findings suggestive of meningitis. The predominant cell type in CSF samples can be lymphocytes, neutrophils, or eosinophils.

Some patients develop communicating hydrocephalus due to obstruction of CSF outflow [26]. Others may present with vasculitis and stroke due to accompanying angiitis. Case-fatality rates of ~50% were documented in studies from Mexico, even when shunting was performed [61]. However, better outcomes have been documented in most series from the United States [4, 25].
MRI, myelography, or CT myelography. My colleagues and I, as well as other investigators, have observed a few patients with leptomeningeal spinal cysts who responded when treated with antiparasitic drugs instead of surgery [25]. However, there has been too little experience with this approach to recommend it routinely. Surgical resection is still the standard approach for patients with intramedullary spinal cysts.

**Ophthalmic cysticercosis.** *T. solium* can also infest the eye. Most of the parasites are found in the vitreous humor, about one-third are found in the subretinal space, and a smaller number are found in the subconjunctiva or anterior chambers [62]. *T. solium* infection is usually diagnosed while the parasites are still viable (many are observed to be actively motile on fundoscopic examination). Although the standard therapeutic approach is surgical removal of the cysts, there are case reports of therapy with antiparasitic drugs. However, there is a substantial risk of inducing a brisk inflammatory response that would compromise visual acuity. Therefore, most patients should be treated surgically.

**Cerebrovascular disease.** A minority of patients with neurocysticercosis can present with cerebrovascular accidents.

In contrast to parenchymal cysts, which reach sizes of only 1–2 cm in diameter, subarachnoid cysts in some patients enlarge to 10 cm in diameter and may cause CNS mass effect. This is particularly true for cysts found in the sylvian fissure. Some of these cysts no longer have scolexes and have been described as racemose. However, patients have identical clinical presentations whether scolexes are present or not. Patients with subarachnoid cysticercosis typically have marked pleocytosis and tend to have recurrent symptoms.

**Unusual and Recently Recognized Presentations**

**Spinal cysticercosis.** A minority of patients present with symptoms primarily related to the spinal cord. The cysts are usually located in the leptomeningeal space but rarely may be found within the cord. Patients typically present with radicular pain or paresthesias; in some patients, these findings may progress to signs and symptoms of spinal cord compression.

In most cases, spinal cysticercosis is not diagnosed before surgical removal of the cysts, which remains the best documented therapy. However, it is possible to identify cysts by MRI, myelography, or CT myelography. My colleagues and I, as well as other investigators, have observed a few patients with leptomeningeal spinal cysts who responded when treated with antiparasitic drugs instead of surgery [25]. However, there has been too little experience with this approach to recommend it routinely. Surgical resection is still the standard approach for patients with intramedullary spinal cysts.

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**Cerebrovascular disease.** A minority of patients with neurocysticercosis can present with cerebrovascular accidents.
Lacunar infarctions that result from angiitis secondary to the basilar arachnoiditis of cisternal disease typically occur in young females (figure 4) [63, 64]. These infarctions are indistinguishable from lacunar infarctions caused by atherosclerosis. However, most patients will have evidence of basilar arachnoiditis on neuroimaging studies as well as CSF (pleocytosis). Findings on cerebral angiograms are usually normal, since only small vessels are involved. Patients typically recover completely [63, 64]. Patients can also have large-vessel strokes related to cysticercosis. In one recent series, large-vessel strokes were actually more common than lacunar infarctions [64]. In most of these cases, large cysticerci have been found associated with the affected vessel.

A third group with cysticercal cerebrovascular disease includes patients with chronic cysticercal meningitis [64]. These patients present primarily with symptoms of communicating hydrocephalus, but their courses are often complicated by multiple cerebral infarctions, deteriorating mental status, and, in some cases, death [64].

Cysticercosis and migraine headaches. Cruz and colleagues [36] conducted a neuroepidemiological study in Ecuador to define the prevalence and morbidity associated with neurological disease in a village where cysticercosis is endemic. They discovered a prevalence of migraine headaches nearly double that identified in other areas. The prevalence of neurocysticercosis diagnosed by either CT (33% of cases) or EITB assay (21% of cases) was higher among patients with migraines than among patients without migraines from the same village. Thus, in areas of endemicity, cysticercosis may be an important cause of migraine.

Neurocognitive defects. Levav and colleagues [65] performed neurocognitive testing on patients with neurocysticercosis and compared the findings with those for patients with other forms of brain damage and those for unaffected relatives. They found a significant association between neurocysticercosis and altered visual-motor control, attention, impulsiveness, and motor control. These data suggest that cysticercosis may be associated with neurocognitive effects, even in patients with no apparent symptoms.

Extraneural forms of cysticercosis. Cysticercosis can also be present outside the nervous system. Cutaneous cysticercosis is only rarely identified in patients infected in the western hemisphere but is apparently more common in Africa and Asia. Patients typically present with soft subcutaneous nodules that resemble sebaceous cysts. The infection is usually suspected only when the nodules are resected or histopathologic examination is performed. Cysts commonly infect the muscle, and the infection is typically asymptomatic. However, patients occasionally present with a hypertrophic mass. In cases of heavy infestation, patients may develop myopathy.

Diagnosis

Neuroimaging studies are the main methods for diagnosing neurocysticercosis. CT is the best method for detecting calcification associated with prior infection. The calcifications vary in size but are often several millimeters in diameter. CT will usually reveal parenchymal lesions. Most parenchymal cysts will appear as low-density cysts, with enhancement of the cyst wall or surrounding tissues, usually accompanied by surrounding edema (figure 1). In some cases, CT will reveal only focal areas of enhancement that may resolve spontaneously.

Most ventricular and cisternal cysts cannot be visualized directly on CT scans, although their presence can often be surmised in the setting of obstructive hydrocephalus. Intraventricular and cisternal cysts can sometimes be identified after metrizamide is injected into ventricular or lumbar CSF; however, this procedure is rarely necessary. Spinal cysts can be identified by MRI, myelography, or CT myelography.

MRI is more sensitive than CT for revealing cysts in the brain parenchyma and in extraparenchymal sites. In some cases, the invaginated scolex can be identified as a mural nodule (figures 2 and 3). The presence of a mural nodule is pathognomonic of neurocysticercosis. Recent studies have also shown that MRI is better for identifying inflammation associated with the cysts [7]. Most cysts in the ventricles can also be identified by MRI (figure 3) [66]. In addition, MRI is much better than CT for identifying cysts in the basilar cisterns [67].

Serological tests. A wide range of serological tests have been used in diagnostic and epidemiological studies of cysticercosis. Unfortunately, most of the tests that use unfractionated antigen are associated with high rates of false-positive and false-negative results [27, 28]. The high rate of false-positive results may be due in part to the avidity of the cysts for immunoglobulin. For example, parasite paramyosin specifically binds the Fc portion of immunoglobulin [68]. False-negative tests may result from the high cutoffs required to avoid false-positive results.

The EITB assay has proved highly specific for T. solium infection; no definite false-positive tests have been observed [9, 10, 69]. The EITB assay is nearly 100% sensitive for patients with either multiple active parenchymal cysts or extraparenchymal neurocysticercosis [69]. However, the sensitivity is lower for patients with either single parenchymal cysts or calcifications alone [27, 69]. The EITB assay is more likely to be positive when serum samples are tested than when samples of CSF are tested. Thus, patients can often be treated without prior lumbar puncture. The EITB assay is currently available commercially.

In the past, plain films of the skeletal system were used to identify calcifications suggestive of prior T. solium infection. However, findings on skeletal radiographs add little to results of neuroimaging studies. Some investigators recommend stool examination for the presence of Taenia ova or proglottids. However, these tests are insensitive, even for tapeworm carriers. It may be important to screen close contacts of infected patients who have not lived in or visited countries where the parasite is endemic, since a minority of these families will include tapeworm carriers [50].
Treatment

The treatment of cysticercosis should be individualized on the basis of the pathogenesis of disease in each patient. Factors used to tailor therapy should include the location of the cysts, symptoms such as seizures or hydrocephalus, the viability of the cysts, and the degree of host inflammatory response.

Inactive Infection

Patients with seizures and calcifications alone on neuroimaging studies are not thought to have viable parasites. Therapy in such cases is limited to symptomatic treatment with anticonvulsants for patients with seizures. In general, the seizures are fairly well controlled in patients who adhere to treatment regimens [39]. For patients with inactive infection and hydrocephalus, the symptoms of hydrocephalus can usually be cured by diversional procedures such as ventriculoperitoneal shunting. Since these patients do not have evidence of viable parasites, there is no role for antiparasitic drugs in treatment.

Active Parenchymal Neurocysticercosis

The presence of viable cysts in the brain parenchyma is usually not associated with symptoms. The disease is usually characterized by a prepatent period of several years [7, 8], a time span approximating the life expectancy of tissue cysts. Neuroimaging studies reveal viable cysts as small rounded areas of low density, corresponding to the cyst fluid, without associated edema or enhancement (figure 2). The cyst wall is isodense with the brain parenchyma, and the two structures cannot be distinguished. This finding corresponds to the vesicular stage observed on histopathologic examination.

In contrast, nearly all patients with symptomatic parenchymal disease have evidence of inflammation (edema, enhancement, or a visible cyst wall) on neuroimaging studies (figure 1), which corresponds to the colloidal or granular-nodular stages observed histopathologically. Natural history studies have shown that the lesions in nearly all of these patients will resolve within 2 years [5, 54].

Patients with parenchymal neurocysticercosis typically present with seizures, and treatment with anticonvulsants is the main therapeutic measure. For most patients, seizures can be controlled with a single anticonvulant [5, 39, 53]. Many of these patients can eventually discontinue anticonvulsant therapy, and the seizures will not recur. Recurrent seizures appear to be more common in patients who develop calcifications or who have multiple lesions [39, 53, 70].

Reports on the use of antiparasitic agents in neurocysticercosis first appeared in the 1970s [71]. Praziquantel was the first antiparasitic agent reported to be active against cysticercosis. It is an orally administered isoquinoline with activity against a broad range of trematodes and cestodes. It diffuses into the CSF at a level approximating that of free drug in serum (~10% of the total). It has usually been used for patients with neurocysticercosis at a dosage of 50–60 mg/(kg · d) in three daily doses for 15 days. Even with standardized dosages, serum concentrations are quite variable.

Additional drugs commonly administered to patients with neurocysticercosis including phenytoin, phenobarbital, and corticosteroids increase cytochrome P-450 mediated metabolism of praziquantel and significantly lower levels of the drug in serum. Praziquantel can be administered safely at doses of up to 100 mg/(kg · d) [72], and higher doses may be more effective in patients who are receiving the other medications. Alternatively, levels of praziquantel can also be increased by coadministering it with cimetidine [73].

Albendazole is a broad-spectrum antihelminthic agent that was subsequently studied in patients with cysticercosis. It was approved by the U.S. Food and Drug Administration (FDA) in 1996 for treatment of hydatid disease and parenchymal cysticercosis in patients found to have nonenhancing cysts. Albendazole is a benzimidazole antiparasitic agent with activity against most nematodes and cestodes. It has been used extensively outside the United States for patients with intestinal nematodes, strongyloidiasis, cutaneous larva migrans, and hydatid disease.

In studies of patients with cysticercosis, the usual dosage has been 15 mg/(kg · d) given in two or three daily doses. The duration of treatment has ranged from 8 to 30 days; no apparent difference in outcome has been observed with increased duration of therapy. In contrast to praziquantel, albendazole can be given in combination with steroids and anticonvulsants without a significant effect on albendazole’s serum levels.

Initial reports described the resolution of cysts on neuroimaging studies after praziquantel and, subsequently, albendazole were administered [74, 75]. Scan findings normalize within 3 months for 54%–96% of patients who receive these agents. However, some patients have developed worsening inflammation surrounding the cysts, with associated symptoms (e.g., nausea, vomiting, and headache); some patients even developed cerebral edema. Some investigators have recommended that corticosteroids be used routinely as an adjunct to antiparasitic therapy [76]; others recommend that corticosteroids be given only to patients who develop symptoms [71].

While antiparasitic drugs have controlled seizures in most patients with neurocysticercosis, the results have not obviously differed from the observed natural history of neurocysticercosis. Some investigators have assessed outcomes of seizures in patients treated with antiparasitic drugs compared with outcomes in patients who refused therapy [39, 58]. While there was a decrease in the frequency of seizures in patients who received antiparasitic therapy, it is not clear that this was a result of therapy rather than patient selection bias.

Comparative studies have generally shown that the cysts resolved at least as quickly with albendazole therapy as with
praziquantel therapy [59, 75, 77]. Differences in the results with albendazole vs. praziquantel may be due to the suboptimal use of praziquantel (e.g., steroids or anticonvulsants were coadministered with praziquantel without dose adjustment or the addition of cimetidine).

Two randomized, placebo-controlled trials have been conducted to examine the effects of antiparasitic drugs in patients with parenchymal cysticercosis. In a small trial from India, patients with parenchymal cysts and associated inflammation were treated with albendazole or placebo and there was no difference in the rate of resolution of cysts [78]. In a larger trial from Ecuador, patients who had cysts without inflammation were all given corticosteroids and randomized to treatment with albendazole, praziquantel, or placebo [79].

There was a trend towards faster resolution of the cysts in patients treated with antiparasitic drugs, but there was no difference in the frequency of seizures. Patients treated with antiparasitic drugs developed hydrocephalus more frequently, but this difference was not statistically significant.

Overall, the current literature suggests that antiparasitic drugs exert an effect by slightly improving the rate of resolution of cysts. However, there appears to be little clinical benefit for patients who are taking these medications and in whom anticonvulsant levels can be monitored. The increased rate of resolution may be advantageous for selected patients, but there is also a small risk of acutely inducing inflammation and cerebral edema and, perhaps, of causing chronic hydrocephalus. Carefully controlled trials of these agents are still needed.

**Extraparenchymal Neurocysticercosis**

In contrast to patients with parenchymal cysticercosis, for whom the outcome is generally good regardless of therapy, patients with extraparenchymal disease are at higher risk for developing permanent neurological sequelae and of dying if they are not treated properly [25]. Hydrocephalus usually requires surgical therapy. The roles of other therapies, including corticosteroids and antiparasitic drugs, are less well defined.

**Ventricular neurocysticercosis.** Most patients with ventricular neurocysticercosis present with obstructive hydrocephalus. Rapid correction of hydrocephalus, which is the mainstay of therapy for ventricular disease, usually involves surgical therapy—either CSF shunting or removal of the cyst(s).

Surgical excision has been the traditional approach to therapy for ventricular cysts, particularly when the cysts are not associated with significant ependymitis [71, 80]. If ependymitis is not present, there is no risk of the hydrocephalus recurring after the cysts are excised, even without placement of a shunt. A transcortical approach is used for resecting cysts in the lateral ventricles; a suboccipital approach is used for fourth-ventricle cysts; and a transcallosal approach is used for third-ventricle cysts.

There are anecdotal reports of cyst removal by ventriculoperitoneal shunting. In the absence of inflammation, the cysts are usually freely mobile, and their location should be confirmed in the immediate preoperative period. Excision is more difficult if the cyst is associated with significant inflammation. Rupture of cysts has not been associated with adverse sequelae [71, 80].

Diversional procedures such as ventriculoperitoneal shunting are therapeutic alternatives for patients with hydrocephalus and ependymitis [80]. In earlier studies, investigators noted failure of shunts, which required revision in two-thirds of patients who were treated with shunting alone [25]. In my institution, treatment with antiparasitic drugs, primarily praziquantel, in combination with shunt placement has resulted in a low rate of shunt failure [2, 25].

Other investigators have noted low rates of shunt failure when volume-regulated shunts are placed [81]. Thus, placement of shunts can be a less invasive alternative for the treatment of ventricular cysticercosis. There are a few reports of patients with ventricular cysts and no hydrocephalus who have been treated with albendazole alone [82]. However, there are presently too few published data to determine whether this approach is either safe or effective.

**Subarachnoid neurocysticercosis (cisternal or racemose neurocysticercosis) and giant subarachnoid cysts.** There are no controlled trials addressing treatment of subarachnoid cysticercosis. Diversion of the CSF should be undertaken in patients with communicating hydrocephalus. There is a high rate of shunt failure among these patients, as has been noted among patients with ventricular disease. In some cases in which giant cysts and mass effect are present, surgical decompression and aspiration of cyst contents have resulted in dramatic improvement in the patient’s condition [83].

Since the major pathogenic mechanism is arachnoiditis, many investigators have argued that corticosteroids have a primary role in the management of cysterceral arachnoiditis. Patients who have had strokes or have angiitis appear to benefit from corticosteroid therapy, but no controlled studies and few carefully documented studies with long-term follow-up have been conducted to confirm this.

The role of antiparasitic drugs in the treatment of subarachnoid cysticercosis is also not clear [25]. Initial studies of praziquantel therapy showed that patients with pleocytosis responded more poorly to therapy than did patients with parenchymal cysticercosis and normal CSF profiles [56]. There are reports of dramatic responses to praziquantel therapy [25], but most patients require repeated courses. In some cases, administration of antiparasitic drugs may precipitate cerebrovascular complications. On the basis of anecdotal reports of some dramatic responses, some authorities argue that albendazole should be administered in all cases of subarachnoid cysticercosis and that all patients should be pretreated with corticosteroids [71].
At my center, most patients are treated with corticosteroids, then ventriculoperitoneal shunting, followed by antiparasitic drugs. Praziquantel has usually been administered, since it was more readily available before the FDA approval of albendazole in late 1996. However, albendazole may be more effective for subarachnoid cysticercosis [71]. In some cases, cysts have been removed in an attempt to decrease the antigen load, and the outcomes have generally been favorable. However, it is unclear which components of this approach are most important.

Control and Prevention of Taenia Infection

The International Task Force for Disease Eradication has identified neurocysticercosis as a target for eradication efforts on the basis of feasibility and public health importance [31]. Eradication is believed possible since the parasite has two obligate hosts: humans and pigs. The main method of control in developed countries has been the eradication of porcine cysticercosis through improved animal husbandry and meat inspection procedures. This approach has resulted in the successful interruption of transmission of intestinal *T. solium* in the United States and western Europe. These approaches have typically failed in developing countries. Raising animals in pens or areas where they cannot scavenge for food requires that they be given feed, which is often unaffordable for a poor farmer. Infected pigs can often be detected by the presence of cysts in the tongue; however, rather than sustaining the economic loss that would result from condemnation of the animals, poor farmers often sell the meat informally rather than to abattoirs, where it will be inspected [84].

Mass administration of chemotherapy to populations in areas of endemicity has been tried in an attempt to eradicate human tapeworm carriage [85]. In a pilot study in Ecuador, mass chemotherapy with praziquantel resulted in elimination of intestinal tapeworm carriage and marked reduction in the rate of porcine cysticercosis [85]. A similar approach in Mexico was less successful.

There is a solid protective immune response against the oncosphere stage in animals with active or cured cysticercosis [15]. Population studies of ovine cysticercosis have suggested the immune response in the intermediate host is critically important in limiting infection. Johnson and colleagues [86] took advantage of this response by using antibody to clone a protective antigen from a *Taenia ovis* oncosphere-stage cDNA expression library. This antigen has now been developed as a recombinant vaccine for ovine cysticercosis and is available commercially for veterinary use in New Zealand [87]. On the basis of evidence of a similar immune response to *T. solium*, it should also be possible to develop an effective vaccine to prevent both human and porcine cysticercosis [16]. However, little work in this direction has been reported to date.

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1. Which of the following is true concerning transmission of Taenia solium in the United States?
   A. There has never been transmission of cysticercosis (infection with the cyst stage) in the United States.
   B. Patients acquire cysticercosis from ingesting contaminated pork.
   C. Patients formerly could acquire cysticercosis in the United States, but transmission has been eliminated.
   D. Patients acquire intestinal tapeworms via contact with other Taenia solium tapeworm carriers.
   E. Patients continue to acquire cysticercosis in the United States via contact with Taenia solium tapeworm carriers.

2. The major mechanism of disease in ventricular cysticercosis is
   A. Seizures due to parenchymal inflammation
   B. Obstructive hydrocephalus from mechanical obstruction
   C. Cerebral edema due to inflammation
   D. Communicating hydrocephalus due to CSF outflow obstruction

3. Which of the following drugs have been proven in randomized controlled trials to improve the clinical outcome in parenchymal cysticercosis:
   A. Praziquantel
   B. Albendazole
   C. Both
   D. Neither

4. Which of the following statements is true of cysternal cysticercosis?
   A. Most patients do well with only symptomatic therapy.
   B. Patients with cysternal cysticercosis respond better to antiparasitic agents.
   C. Patients often develop hydrocephalus as a result of chronic arachnoiditis.
   D. Patients usually develop obstructive hydrocephalus.

5. The main component of standard therapy for ventricular cysticercosis with hydrocephalus is:
   A. Either CSF shunting or excision of the cyst to control hydrocephalus
   B. Antiparasitic drug therapy with praziquantel to eliminate the cysts
   C. Antiparasitic drug therapy with albendazole to eliminate the cysts
   D. Corticosteroid therapy to relieve cerebral edema

6. Which of the following statements is true concerning efforts to control Taenia solium?
   A. Meat inspection has been the most successful technique used in developing countries.
   B. It is unlikely that Taenia solium will ever be eliminated because of the large number of hosts involved.
   C. Mass chemotherapy for tapeworm carriers can decrease the prevalence of infection in developing countries.
   D. As is the case for other helminths, it is unlikely that vaccination will ever play an important role in control because the hosts do not develop a solid protective immune response.

7. Which of the following statements is true for patients with parenchymal neurocysticercosis?
   A. Most patients require anticonvulsants for a few years, but many can then discontinue anticonvulsant therapy.
   B. Seizures usually occur only once, so patients do not need anticonvulsant drugs.
   C. All patients require lifelong therapy with anticonvulsants.
   D. Lesions seen on neuroimaging studies usually persist unless the patient is treated with antiparasitic drugs.
E. Most patients should undergo surgical excision of the lesions to prevent recurrent symptoms.

8. Which of the following statements is true for patients who present with seizures and intraparenchymal calcification alone on a CT scan?
   A. All such patients should be treated with antiparasitic drugs such as albendazole to prevent recurrent symptoms.
   B. Patients should be treated with anticonvulsants but do not need antiparasitic drugs, since they no longer have viable cysts.
   C. Patients should be treated with corticosteroids to decrease inflammation.
   D. Many patients will require placement of a ventriculoperitoneal shunt for hydrocephalus.

9. The single diagnostic test most likely to lead to the diagnosis of active parenchymal neurocysticercosis with a single cyst is
   A. Contrast MRI to visualize the cyst and associated inflammation
   B. Lumbar puncture to obtain a CSF sample for profile and detection of antibodies to T. solium
   C. Enzyme-linked immunotransfer blot (EITB) assay of serum for antibodies to T. solium
   D. Electroencephalography

10. The most common presentations of neurocysticercosis in series from the United States are
    A. Communicating hydrocephalus secondary to chronic arachnoiditis
    B. Obstructive hydrocephalus from cysts in the ventricles
    C. Seizures due to parenchymal cysts, without associated edema or enhancement on neuroimaging studies
    D. Seizures due to parenchymal cysts, with associated edema or contrast enhancement of the cysts
    E. Visual changes due to ocular cysticercosis