

NEUROCYSTICERCOSIS: Updates on Epidemiology, Pathogenesis, Diagnosis, and Management

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■ **Abstract** Neurocysticercosis is now recognized as a common cause of neurologic disease in developing countries and the United States. The pathogenesis and clinical manifestations vary with the site of infection and accompanying host response. Inactive infection should be treated symptomatically. Active parenchymal infection results from an inflammatory reaction to the degenerating cysticercus and will also respond to symptomatic treatment. Controlled trials have not demonstrated a clinical benefit for antiparasitic drugs. Ventricular neurocysticercosis often causes obstructive hydrocephalus. Surgical intervention, especially cerebrospinal fluid diversion, is the key to management of hydrocephalus. Shunt failure may be less frequent when patients are treated with prednisone and/or antiparasitic drugs. Subarachnoid cysticercosis is associated with arachnoiditis. The arachnoiditis may result in meningitis, vasculitis with stroke, or hydrocephalus. Patients should be treated with corticosteroids, antiparasitic drugs, and shunting if hydrocephalus is present.

INTRODUCTION

Neurocysticercosis (NCC), caused by infection of the human central nervous system with the parasite *Taenia solium*, is now recognized as an important public health problem both in developing countries and in the United States (1–3). Three developments led to the recognition of NCC as a major cause of neurologic disease (1).

First, the development of computerized neuroimaging studies [e.g. computed axial tomography (CT) and magnetic resonance imaging (MRI) scanning] provided the first sensitive, noninvasive diagnostic tests for NCC.

Second, large numbers of rural immigrants from developing countries arrived in the United States in the 1970s and 1980s. When this population developed neurologic disease, they were studied by CT or MRI, leading to the frequent diagnosis of NCC. For example, the number of NCC cases diagnosed in Los Angeles increased fourfold between 1977 and 1981, associated with initial use of CT scanning (1). Since the early 1980s, numerous case series of NCC patients

have been reported from the United States. The first series came from the Southwest, the area with the highest concentration of Latin American immigrants (2, 4). Large series have now been reported from throughout the country (2, 5). Furthermore, the immigrant communities have also included tapeworm carriers. The result has been recognition of cases of NCC acquired in the United States (6, 7).

Third, improved serodiagnostic assays allowed accurate seroepidemiologic studies. The result has been a dramatic increase in estimates of the prevalence of NCC throughout Latin America as well as in Africa, India, and Asia (1, 8).

TAENIA SOLIUM AND ITS LIFE CYCLE

T. solium requires two hosts to complete its life cycle. Pigs, the intermediate hosts, contain the cysticerci, primarily in muscle. Humans become infected with the tapeworm form (taeniasis) by ingesting undercooked pork containing *T. solium* cysticerci. After ingestion, the scolex evaginates and attaches to the small intestines. Segments termed proglottids develop from the base of the scolex. The mature proglottids are ~1 cm wide, 1–2 cm long, and 2–3 mm thick. Eggs and/or proglottids are shed intermittently in the stool. Tapeworm carriers usually note few symptoms other than observing proglottids passed with stool. Excretion is intermittent, and stool examinations for ova are usually negative. Pigs become infected by ingesting the ova or proglottids. In the intestines the larvae hatch, penetrate the intestinal mucosa, enter the bloodstream, migrate to the tissues, and mature into cysticerci.

Human cysticercosis also results from ingestion of ova shed by a human tapeworm carrier. Close personal contact with or perhaps food preparation by a tapeworm carrier is noted in most cases. Autoinfection may also occur. Cysticercosis is not acquired directly from ingesting pork, as is illustrated by an outbreak that occurred in an Orthodox Jewish community in New York City (6). This community did not ingest pork. Instead, cysticercosis was acquired from domestic workers, who had immigrated from developing countries and were tapeworm carriers.

EPIDEMIOLOGY

Older data on the prevalence of NCC were flawed by insensitive case-finding methods (3). During the early 1990s, Latin American investigators reported case series of adults with new-onset seizures. They found that up to 50% of cases had CT scan evidence of NCC (9, 10). These studies led to the estimation that NCC is the major reason why the incidence of seizures in Latin America is approximately twice as high as in developed countries (11).

Tsang et al (8) reported the development of a specific immunoblot assay for cysticercosis in 1989, termed the enzyme-linked immunotransfer blot (EITB). The EITB test uses parasite glycoproteins, isolated by lectin affinity chromatography and an immunoblot format (8). A reaction to one or more 9–50-kDa bands is nearly 100% specific for *T. solium* infection with an enhanced sensitivity. Studies that used EITB in population-based analyses of endemic villages demonstrated that earlier estimates of the seroprevalence were several-fold too low (12). Endemic villages in Mexico, Guatemala, Bolivia, Peru, and Ecuador have demonstrated a seroprevalence of NCC infection of between 4.9% and 24% of the entire population (1). These numbers also underestimate the prevalence of infection because only about half of cases diagnosed by CT are seropositive (12, 13).

NCC has been recognized in nearly all non-Muslim countries in Africa, but there are few good data on prevalence (3, 14). CT scanning of Africans with seizures led to the frequent recognition of NCC in South Africa (15). Subsequent studies have demonstrated evidence of NCC in up to 38% of patients with seizures in Togo (16). High seroprevalence rates have been noted in Madagascar, Zimbabwe, and Burundi (1, 3, 17).

In India, CT scans of adults with new-onset seizures demonstrated abnormalities in most patients. Many of these scans demonstrated single enhancing CT lesions, which were initially attributed to tuberculosis or to the effects of seizures. Chandy et al (18) performed excisional biopsies on patients with focal enhancing lesions and demonstrated histopathologic evidence of cysticercosis in most patients. Rajshekhar & Chandy (19) proposed to distinguish cysticercal granulomas from other causes based on clinical criteria. Among patients presenting with seizures and a single enhancing CT lesion ≤ 20 mm in diameter, cysticercal granuloma was presumptively diagnosed in those without evidence of raised intracranial pressure or midline shift, progressive neurocognitive defects, or active systemic disease. Among 215 such patients, only 2 were later found to have other illnesses. By contrast, nearly all 185 patients with single lesions not meeting these criteria were found to have malignancies or tuberculosis. In a series of 991 patients with symptomatic localization-related seizures in south India, 40% were found to have either active NCC, calcifications on CT scan consistent with prior cysticercosis, or single enhancing CT lesions (consistent with cysticercal granulomas) (20). Thus, NCC is a major cause of seizures in India, with a prevalence similar to that noted in Latin America. Fewer data are available from elsewhere in Asia. Large case series have been reported from China, Korea, and Indonesia. Serologic surveys with unfractionated antigens suggest a seroprevalence of about 3–4% in rural residents of China and Korea (21, 22).

In the United States, NCC is primarily an imported disease. Based on the numbers of sera tested and studies in smaller populations, an estimated 1000 new cases are diagnosed each year (1, 5). Most cases occur among immigrants, primarily from Latin America. A minority of NCC patients were born in the United States, but most of these patients had traveled to rural areas in endemic countries

(7, 23). However, locally acquired infection has been clearly demonstrated in cases occurring in Los Angeles, New York, Chicago, and elsewhere (1, 6, 7).

PATHOLOGY AND PATHOGENESIS

The cysticerci must survive in the muscle of the pig for weeks to months in order to complete their life cycle. In the muscle, the viable cysticercus appears as a thin-walled, oval cyst, ~1 cm in diameter. A single invaginated scolex is found as a 1- to 2-mm firm, opaque nodule attached to one side of the wall. Histologic examination of the cyst wall reveals a superficial smooth, eosinophilic cuticular layer, a cellular layer with multiple nuclei and musculature, and a loose reticular layer below. Viable parasites have little surrounding inflammation. When present, inflammation consists of discrete areas of lymphocytes and few eosinophils.

Experimental models have identified a number of mechanisms used by the cysticercus to modulate the host's immune and inflammatory responses (24). The parasites secrete a serine proteinase inhibitor, termed taeniaestatin, which inhibits complement activation, lymphocyte activation, and cytokine production. The parasite surface is covered with sulfated polysaccharides, which are shed and activate complement away from the cyst wall. Parasite paramyosin is thought to inhibit classical pathway complement activation. The parasites elaborate prostaglandins and low-molecular-weight molecules that decrease inflammation and shift host cytokine production toward T-helper 2 molecules (24, 25). Cysticerci secrete proteases that can degrade interleukin (IL)-2 and immunoglobulin (S Baig, R Hashmey, P Robinson, AC White Jr, manuscript in preparation). Viable cysticerci also stimulate immunoglobulin production. Instead of damaging the cysticerci, immunoglobulin molecules are taken up by the cysts and degraded as a source of amino acids (24). When parasites degenerate, there is a brisk host inflammatory response, primarily composed of lymphocytes but also including some neutrophils and eosinophils. Early stages of degeneration are associated with stimulation of T-helper 1 cytokines, interferon- γ , and IL-2 (26).

Although the cysticerci reach their mature size within a few weeks, there is typically a period of several years between exposure and onset of symptoms (23, 27). Thus, infection of the central nervous system alone does not explain symptoms. Individuals who died of other causes have been found upon autopsy to have viable cysticerci, which appear similar to the viable cysticerci from pig muscle (28). By contrast, cysticerci from patients with seizures invariably demonstrate a prominent inflammatory infiltrate. Thus, the seizures are thought to result not from the parasitic infection per se, but rather from the host response.

Cysticerci pass through a series of stages (29). Initially, viable cysticerci have minimal associated inflammation (vesicular stage). As the cysticerci lose their ability to control the host response, the cyst wall is infiltrated and surrounded by host inflammatory cells composed primarily of mononuclear cells. The inflammatory cells also enter the cyst fluid (colloid stage). This inflammatory response

TABLE 1 Presentation and treatment of different forms of neurocysticercosis

Activity	Location	Typical presentation	Treatment
Inactive	Parenchymal calcification ± enhancement	Seizures	Anticonvulsants ^a
	Chronic hydrocephalus	Symptoms of increase Intracranial pressure	Ventriculoperitoneal shunting
Active	Parenchymal	Seizures	Anticonvulsants, ^a antiparasitic drugs, ^{b,c} or steroids ^{c,d}
	Ventricular	Hydrocephalus	Surgery (shunting versus removal), antiparasitic drugs, ^{b,c} steroids ^{c,d}
	Subarachnoid (cisternal)	Seizures, hydrocephalus, stroke	Steroids, antiparasitic drugs ± shunting ^e
	Ocular	Visual changes	Surgical removal
	Spinal subarachnoid	Radiculopathy or myelopathy	Surgical removal and/or antiparasitic drugs
	Spinal intramedullary	Myelopathy	Surgical removal

^aAnticonvulsant therapy includes phenytoin, phenobarbital, or carbamazepime.

^bAntiparasitic drugs include praziquantel and albendazole.

^cThis treatment is controversial. For example, randomized trials have shown no benefit of antiparasitic drugs in parenchymal disease. The role of either steroids or antiparasitic drugs in ventricular disease is supported by single or poorly controlled trials.

^dCorticosteroids include dexamethasone or prednisone. Their role in parenchymal and ventricular disease is not established.

^eThe management of subarachnoid cysticercosis has not been established from well-controlled trials. However, the best studies support the use of corticosteroids, antiparasitic drugs (especially albendazole), and shunting for hydrocephalus.

is associated with elaboration of type 1 cytokines such as IL-12, interferon- γ , and IL-2 (30). As the host response progresses, fibrosis encompasses the cysticercus with collapse of the cyst cavity (granular-nodular stage). Eventually, the parasite is replaced by progressive fibrosis, which may calcify (calcific stage).

Patients with cysticerci in the ventricles present primarily with symptoms of hydrocephalus. In most cases, the hydrocephalus results from mechanical obstruction of cerebrospinal fluid (CSF) flow by a cysticercus or associated ependymitis (3, 4, 31, 32). When the cysticerci become inflamed, the granular ependymitis and accompanying fibrillary astrocytosis cause the cysticercus to adhere to the walls of the ventricles (33).

Cysticerci in the basilar cisterns may induce arachnoiditis. Patients may present with meningeal signs or communicating hydrocephalus (4). Hydrocephalus is thought to result from CSF outflow obstruction or obstruction of the ventricular outflow (3, 4, 31). Arachnoiditis may also be associated with vasculitis and may present as lacunar infarctions (3, 34). In some cases, the cysticerci erode into larger vessels causing large-vessel strokes (34). Some of the cysticerci within the subarachnoid space may enlarge and occasionally lose the scolex; cysticerci without a scolex are termed racemose. The term racemose cysticercosis was used to describe subarachnoid disease, but the clinical presentation, pathogenesis, and treatment are the same regardless of whether the scolex is present. Therefore, current classification schemes no longer use the term racemose.

CLASSIFICATION AND NEUROIMAGING STUDIES

Current clinical classification is usually based on the location of cysticerci and the accompanying host response as determined from neuroimaging studies (35–37). Most schemes separate cysticercosis into active and inactive infection. Others separate degenerating or transitional forms from viable cysticerci and reserve the term active for only the latter (36).

Inactive Neurocysticercosis

Inactive infection refers to cases in which there is no longer evidence of either a viable or a degenerating parasite. Instead, neuroimaging studies demonstrate the residua of prior infection and the host response. The most common form of inactive infection is parenchymal calcifications. CT reveals one or more sites of calcification, typically 2–10 mm in diameter.

Other patients with inactive infection may present with hydrocephalus. In this case, prior arachnoiditis or granular ependymitis resulted in obstruction of CSF flow (e.g. aqueductal stenosis) or in CSF outflow obstruction.

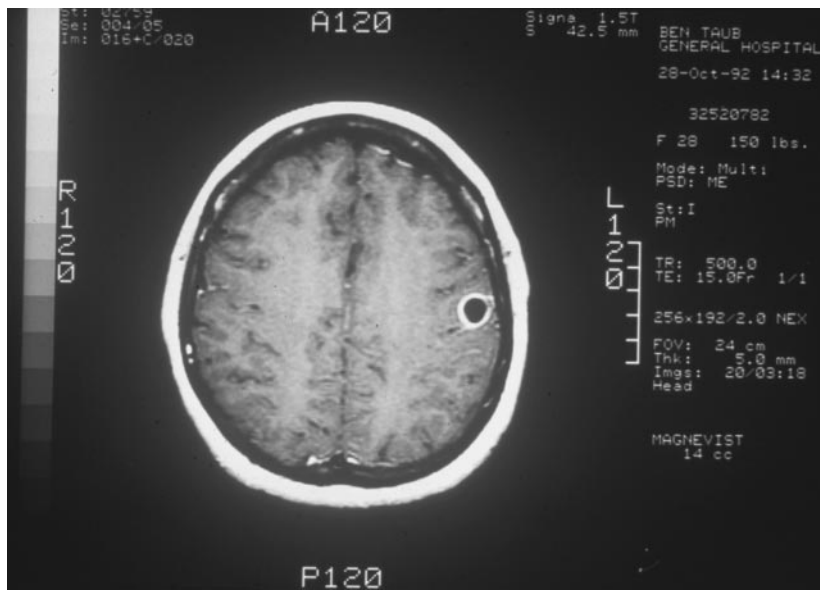
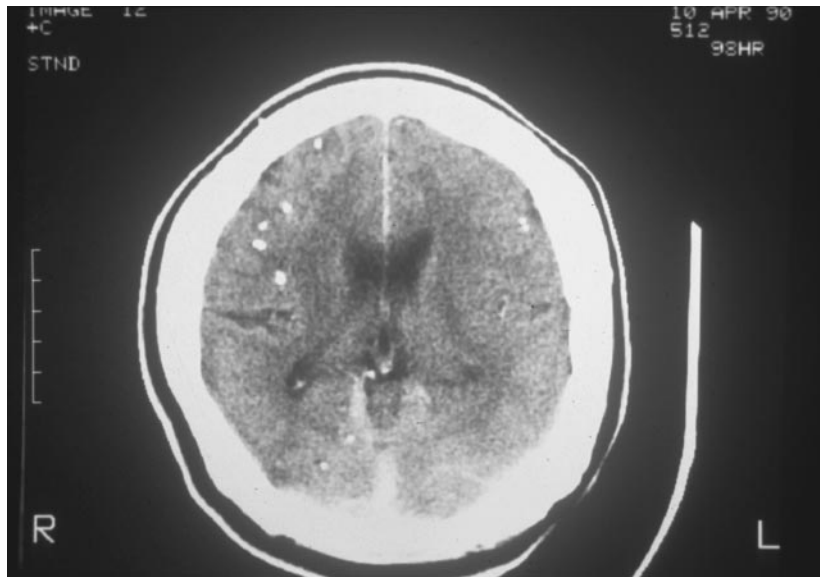
Active Neurocysticercosis

Although some authors reserve the term active NCC for infection with viable-appearing cysticerci, I include both viable and degenerating cysticerci in this category. In our experience at Ben Taub General Hospital in Houston, nearly all patients with symptomatic NCC show at least some evidence that parasite degeneration and/or the host inflammatory response plays a role in their illness (2, 5). Active infection should be further classified based on the location of the cysticerci. Active parenchymal infection typically results from direct effects of the host inflammation and carries a benign prognosis (38). In contrast, extraparenchymal disease is often associated with hydrocephalus, often requires surgical therapy, and can be fatal if not properly managed (32, 38).

Active Parenchymal Neurocysticercosis Active parenchymal infection is the most common form of disease. Viable cysts, as described above, rarely cause symptoms. Instead symptomatic infection develops when the cysticercus loses the ability to control the host inflammatory and immune responses. Neuroimaging studies reveal a progression similar to the pathologic stages (39, 40). Invasion of the central nervous system and early development of the cysticercus may present as an area of focal edema or enhancement. The viable cysticercus appears as an area of decreased density, 4–20 mm in diameter. The cyst fluid is isodense with CSF. The cyst wall is thin (<1 mm thick), isodense with brain tissue, and not usually visible. There is no surrounding edema or contrast enhancement (vesicular stage). As the cysticercus becomes inflamed, the cyst wall becomes denser and may display contrast enhancement of the wall (ring enhancement), cyst fluid, or surrounding tissues (Figure 1). There is often associated edema and/or enhancement in the brain parenchyma, especially on T2-weighted MRI scans. The cyst fluid may be isodense with CSF or develop increased density (colloid stage). As the cysticercus becomes fibrotic or collapses, neuroimaging studies reveal an area of focal enhancement, suggestive of a granuloma (granular-nodular stage). Finally, a focal area of calcification appears, typically several millimeters in diameter (calcific stage) (Figure 1).

In a subtype of active parenchymal NCC, patients present with encephalitis-like symptoms (4). This presentation is more common in children than adults. Cysticercal encephalitis results from infection with large numbers of inflamed cysticerci in the brain parenchyma. The accompanying inflammatory response causes diffuse cerebral edema.

Active Ventricular Neurocysticercosis Of all patients with NCC, 10–20% have cysticerci in the ventricles. Symptoms usually result when the cysticerci obstruct CSF flow, causing obstructive hydrocephalus. The cysticerci can be found in any of the ventricles and may be viable or inflamed. The viable cysticerci have thin walls, and the cyst fluid is isodense with CSF. Thus, CT scanning may only reveal evidence of obstructive hydrocephalus or distortion of the shapes of the involved ventricle. The cysticerci are usually visible on MRI (33, 41). However, the find-



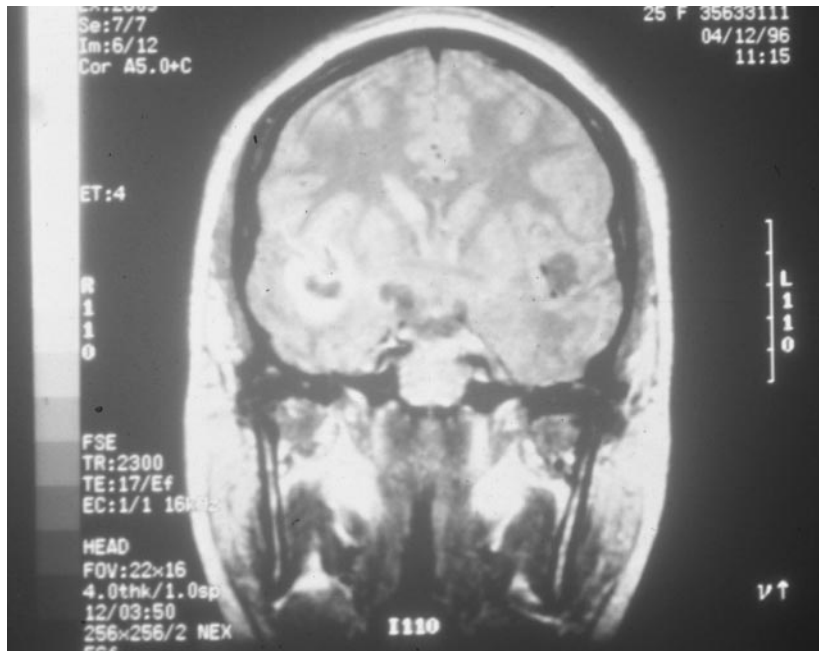


Figure 1 Neuroimaging studies from patients with neurocysticercosis. (*left, top*) CT scan revealing multiple calcifications of inactive parenchymal disease. (Reprinted from Reference 5 with permission.) (*left, bottom*) MRI with gadolinium contrast of a parenchymal cyst in the colloid stage. The hypodense cyst fluid is surrounded by the cyst wall, which enhanced with contrast. (*above*) MRI with gadolinium contrast revealing inflamed giant cysticerci in the Sylvian fissures. A lacunar infarction is visible as an enhancing lesion in the right basal ganglion. The basilar cisterns are also packed with thin-walled cysticerci.

ings are subtle and can be missed by neuroradiologists not familiar with cysticercosis. Often these patients have parenchymal cysticerci as well.

Active Subarachnoid Neurocysticercosis There are three types of subarachnoid cysticercosis. Some patients have cysticerci in the gyri of the cerebral convexities. The appearance, clinical presentation, and pathogenesis are similar to those of active parenchymal NCC (37).

When the cysticerci are found in the fissures (especially the Sylvian fissure), the cysticerci can enlarge to several centimeters in diameter and are termed giant cysticerci (42). Isolated cysticerci in the fissures carry a similar prognosis to parenchymal cysts, and many of the symptoms result from parenchymal inflammation. In some cases, however, the cysts may enlarge to the point of causing midline shift (43). Giant cysticerci are usually accompanied by cysticerci in the parenchyma or basilar cisterns (Figure 1). Although the giant cysticerci are readily

visualized by CT or MRI, the accompanying cisternal cysticerci are often not seen on CT and present with subtle findings on MRI (41, 44).

Cysticercosis of the basilar cisterns carries a graver prognosis (32). A few patients may present with focal problems from a limited number of cysticerci. However, most patients are infected with numerous cysticerci that pack the basilar cisterns. Cisternal cysticercosis is characterized by arachnoiditis, which may be seen as focal or diffuse meningeal enhancement or vasculitis with strokes (45). Patients often develop communicating hydrocephalus (4).

Other Forms of Cysticercosis Spinal NCC results from cysticerci in the sub-arachnoid space (46). Initially, they are free floating and may move between levels. Degenerating cysticerci may eventually become fixed at one level. The accompanying inflammation may result in mass effect. Cysticerci can be intramedullary. These cysts often cause cord compression from mass effect or accompanying inflammation. Most patients with spinal cysticercosis also have evidence of cerebral disease (46). Cysticercosis can also involve the eye; the cysticerci can be either intravitreal or subretinal (47). Cysticerci frequently involve the muscles but rarely cause more than minor symptoms. Cysticerci involving the subcutaneous tissues present as a palpable cystic lesion that is often confused with a sebaceous cyst.

The above discussion has presented cases as if the cysticerci were found in only a single location. Clinical cases, especially those involving large numbers of cysticerci, are often mixed. Patients with mixed forms of disease are usually classified as having the most severe form.

CLINICAL MANIFESTATIONS

The most common clinical manifestation of NCC is seizures (2–4, 48). The seizures may be focal, focal with secondary generalization, or generalized (49). Most patients with seizures have evidence of parenchymal cysticerci with associated edema or enhancement (corresponding to the colloid or granular-nodular stages). Electroencephalogram (EEG) studies in active disease may reveal focal abnormalities (49). The seizures associated with active parenchymal cysts are thought to be provoked seizures, occurring with the active parenchymal inflammation (48). The seizures are usually easily controlled and often resolve as the inflammation subsides.

Some patients have seizures from inactive parenchymal NCC. The calcified lesions are thought to represent scarring from prior active infection (calcific stage). Despite the presentation with seizures, few will have focal abnormalities on EEG studies (49). Patients who develop calcifications are more likely to have recurrence of seizures if anticonvulsants are withdrawn (50). Thus, patients with calcifications are classified as having remote symptomatic, unprovoked seizures (48). They usually require continuous treatment with anticonvulsants. Recent

MRI studies have documented that some patients with NCC and seizures have calcified lesions with associated enhancement (51). There is no evidence that these lesions are associated with viable parasites. Instead, the enhancement may result from stimulation of the host immune response by antigen released from the calcified granuloma.

Headaches are common in parenchymal, ventricular, and cisternal NCC (3, 4). Headaches may be hemicranial or bilateral. They can be confused with uncomplicated migraines or tension headaches. The pathogenesis is variable. In some cases, headache is the initial symptom of raised intracranial pressure (4). The strong association of parenchymal NCC with migraine-like headaches suggests vascular involvement (13). Headaches may also reflect vasculitis associated with cisternal cysticercosis.

Patients may present with symptoms or signs of raised intracranial pressure. Symptoms may include nausea or vomiting, altered mental status, visual changes, or dizziness (4, 5, 32). In adults, raised intracranial pressure usually results from obstructive hydrocephalus caused by cysticerci obstructing CSF flow in the ventricles (4, 31). Patients with numerous cysticerci in the basilar cisterns may also present with communicating hydrocephalus. Some pediatric cases present with large numbers of inflamed cysticerci, causing cerebral edema.

Neurocognitive defects have been described in patients with cysticercosis (52). Infected children are thought to suffer from learning disabilities (53). Cysticercosis also appears to be associated with depression and perhaps psychosis (54). By contrast, acute alterations of mental status usually reflect ongoing seizures or hydrocephalus (4, 5). We have observed that altered mental status that does not resolve after a brief post-ictal period usually results from hydrocephalus (5).

DIAGNOSTIC CRITERIA

Diagnosis of NCC is frequently problematic. Diagnostic certainty depends on conclusive demonstration of *T. solium* infection (2, 55). Histopathology of biopsy or autopsy material is usually diagnostic but is rarely available. When the eye is involved, parasites may be seen directly (47). In some cases, neuroimaging studies will reveal a cystic lesion with an associated 1- to 3-mm mural nodule (i.e. the invaginated scolex), which is pathognomonic of cysticercosis (44). However, in most cases, neuroimaging studies are not pathognomonic. Thus, diagnosis usually depends on a constellation of clinical history, exposure history, laboratory studies, and X-ray findings.

Del Brutto et al (55) proposed diagnostic criteria based on neuroimaging studies, serologic tests, clinical history, and exposure. Major criteria included neuroimaging studies consistent with NCC (e.g. cystic lesions or large calcification), positive immunoblots of serum, or typical muscular calcifications. Minor criteria included consistent symptoms, punctate calcifications, disappearance of lesions with treatment, or subcutaneous nodules (which were not biopsied). Epidemio-

logic criteria included residence in or extensive travel to an endemic area or exposure to a tapeworm carrier. Two major criteria or one major criterion with two minor criteria and exposure were considered diagnostic. A single major criterion or two other criteria or three minor criteria with exposure constituted a possible diagnosis.

Older serologic tests that used unfractionated antigens [including enzyme-linked immunosorbent assays (ELISA) and other techniques] were associated with high rates of false positive and false negative reactions (12, 56). ELISA is considered reliable only when performed on CSF. With EITB, serum provides sensitivity and specificity as good as or better than that noted with CSF. Initial reports suggested that the assay was over 90% sensitive. Subsequent studies demonstrated that EITB is positive in only 28% of subjects with only a single parenchymal lesion (57). Furthermore, the EITB may revert to negative after the cysticercus dies (58). There is also poor sensitivity in patients with only calcified lesions from prior infection (12, 13, 57). A monoclonal-antibody-based antigen detection assay has also been developed (59). Detection of antigen in CSF was highly specific for viable or degenerating cysticerci.

The different neuroimaging modalities provide complementary information. CT scanning is considered more sensitive than MRI at detecting intracerebral calcifications (44). By contrast, MRI is much better at detecting cysticerci in the CSF (e.g. ventricular or cisternal cysticercosis) (33, 41, 44). MRI may also reveal the scolex, which is usually not visible on CT scans (41). Either method is adequate for imaging intraparenchymal cysticerci or hydrocephalus.

MANAGEMENT

Management of NCC should be based on pathogenesis and natural history, which vary with different forms of NCC. Thus, the management of different forms of disease should be considered separately.

Inactive Neurocysticercosis

Patients with inactive parenchymal NCC have no evidence of viable or degenerating parasites. Thus, antiparasitic drugs have no role in management. These patients are at high risk of recurrent seizures (3, 50). The seizures may recur years after the initial diagnosis. For this reason, most symptomatic patients require chronic anticonvulsant therapy. A variety of anticonvulsants have been used, including phenytoin, phenobarbital, and carbamazepime. Patients taking adequate doses of medications are at little risk of recurrent seizures.

Patients with inactive infection with hydrocephalus are thought to have scarring from prior subarachnoid or ventricular infection. Hydrocephalus can be relieved by ventriculoperitoneal shunting. Unlike patients with active ventricular infection, these patients are at low risk for shunt failure and do not require additional therapy.

Active Parenchymal Neurocysticercosis

Most patients with active parenchymal NCC present with seizures. The seizures usually respond to anticonvulsants and do not recur if adequate levels are maintained (60). Thus, anticonvulsants are the mainstay of therapy. Without specific antiparasitic drugs, the cysticerci will usually complete degeneration over a 1- or 2-year period (39, 60–62). In some cases, the cysticerci will calcify, leading to inactive NCC, which requires continued anticonvulsants. In most cases, the neuroimaging studies will normalize. If the patients are seizure free and neuroimaging studies have normalized, the anticonvulsants may be tapered.

Praziquantel was the first antiparasitic drug for NCC and has been widely used since the early 1980s. It is well absorbed after oral administration, but there is extensive first-pass metabolism. Metabolism is induced by anticonvulsants (carbamazepime, phenytoin, and probably phenobarbital) as well as by corticosteroids (63, 64). Metabolism can be inhibited by cimetidine (e.g. 400 mg taken orally three times a day), which should generally be used when praziquantel is coadministered with anticonvulsants or corticosteroids (65). Dose ranging studies showed greater effects at doses (≥ 50 mg/kg/day in 3 daily doses for 14 days). Subsequent studies demonstrated that doses as high as 100 mg/kg/day could be given safely (66). Recent studies with praziquantel given in 3 doses of 25 mg/kg separated by only 2 h suggest similar efficacy with longer courses of therapy (67).

The main adverse effect is worsening neurologic function (e.g. headaches, dizziness, seizures, and increased intracranial pressure) thought to be caused by the host inflammatory response to the dying parasite. Based on these observations, some observers recommend corticosteroids along with antiparasitic drugs (68). Others have argued that routine use of corticosteroids may decrease efficacy.

Albendazole is a benzimidazole anthelmintic agent with broad-spectrum activity against most nematodes and cestodes. It is well absorbed orally. Adverse effects include minor gastrointestinal side effects and neurologic symptoms similar to those described above for praziquantel. Studies at doses of 15 mg/kg/day in two divided doses demonstrated reduction in number and size of parenchymal cysticerci (69). Controlled trials in parenchymal NCC showed no difference in neuroradiologic resolution with treatment for 7 days versus longer courses (70, 71). Comparative studies suggested responses as good as or better than those seen with praziquantel (2, 3, 71).

Although studies of praziquantel and albendazole attributed the responses to the antiparasitic drugs, others noted similar responses with symptomatic therapy alone (39, 60). The first randomized, controlled trials of antiparasitic drugs for parenchymal NCC were reported in the mid-1990s (61, 72, 73). Some showed more rapid radiographic resolution, but the long-term results were similar (61, 62). No well-controlled study has convincingly demonstrated a clinical benefit of antiparasitic drugs over symptomatic therapy alone. A systematic review by the Cochrane Library concluded that there is currently no clear evidence that use of antiparasitic drugs in parenchymal NCC does more good than harm (74). The

current practice at Ben Taub General Hospital is to treat parenchymal NCC symptomatically (i.e. anticonvulsant therapy alone).

Extraparenchymal Disease

Although most cases of NCC are caused by parenchymal disease, most of the morbidity results from extraparenchymal disease (38). Extraparenchymal disease is often the result of hydrocephalus (38). Initial studies suggested that extraparenchymal disease responds poorly to antiparasitic drugs, but subsequent studies demonstrated that many patients who do not have hydrocephalus may be treated medically.

Ventricular Neurocysticercosis Symptoms from cysticerci in the ventricles are often the result of obstructive hydrocephalus. Until recently, surgical removal of the cysticercus was recommended for all patients with intraventricular NCC (75). Cysticerci in the lateral ventricles can be removed via a transcortical/transventricular approach. Cysticerci in the third ventricle can be removed via either a transcallosal or a transventricular approach. Fourth-ventricular cysts are usually approached via a suboccipital craniotomy. Resection is highly effective, but surgery is associated with a significant risk of postoperative sequelae (infection or neurologic damage including seizures) as well as high costs. Furthermore, if neuroimaging studies reveal significant epididymitis, patients may be left with residual hydrocephalus even after resection (33). Therefore, the role of an aggressive surgical approach is being reevaluated (76). Some patients may benefit from postoperative treatment with albendazole for cysticerci not recognized or adequately removed at surgery. Neuroendoscopic approaches to removal of the cysticerci have been described in a few cases and may represent a less invasive approach to removal of the cysticercus (77).

Ventriculoperitoneal shunting has now become the most common approach to the management of hydrocephalus in NCC (76). In older series, shunts were successful at initially relieving hydrocephalus. Shunt failure requiring surgical revision occurred in up to three quarters of cases and resulted from obstruction of the shunt by portions of the cysticercus or by proteinaceous debris that develops after cyst degradation. We have noted fewer shunt failures since we began treating patients with antiparasitic drugs (2, 5). Sotelo and colleagues (78) noted lower shunt failure rates in patients who were treated with prednisone (50 mg orally 3 days a week). A third approach has been the use of a valveless drainage systems with flow constricted by the diameter of the peritoneal catheter (79, 80). This method may decrease the rate of shunt malfunction. However, it is associated with higher rates of inadequate drainage and normopressure hydrocephalus.

The role of antiparasitic drugs in ventricular disease is evolving. Early studies suggested that treatment with praziquantel alone was associated with a poor outcome. Others have noted a clinical response to antiparasitic drugs after hydrocephalus is corrected with shunting (5, 32, 81). Treatment appears to be associated

with a lower risk of shunt failures. Resolution of ventricular NCC in patients without hydrocephalus treated with antiparasitic drugs alone has been reported (82, 83). These are the only prospective studies of antiparasitic drugs alone for ventricular NCC. Thus, there are few data available to judge whether this approach is safe or efficacious.

Subarachnoid Neurocysticercosis The clinical manifestations and natural history of cysticerci in the subarachnoid space are variable. When few cysticerci are isolated to the gyri over the cerebral convexities, the presentation and pathogenesis are similar to those noted for parenchymal cysticerci (37). These cysticerci are more likely to cause CSF pleocytosis. They may not resolve as quickly and have been associated with treatment failure when antiparasitic drugs are used (84).

Cysticerci in the Sylvian fissure often enlarge to a diameter of several centimeters (37). These large cysticerci occasionally cause mass effect. If the mass effect cannot be decreased with corticosteroids, the cysticerci may require surgical decompression (43). This can usually be accomplished by aspiration of the cyst fluid. In most cases, cysticerci in the Sylvian fissure are accompanied by others in the subarachnoid space of the basilar cisterns or spine, in the ventricles, or in the brain parenchyma. Uncontrolled case series noted good clinical responses when giant cysticerci were treated with corticosteroids and antiparasitic drugs (42, 85). However, antiparasitic drug therapy may have to continue longer for giant cysticerci than for isolated parenchymal disease.

Cysticercosis involving the basilar cisterns is associated with a prominent inflammatory response and arachnoiditis (3, 4, 37). Patients may present with meningeal signs, though usually without fever (4). They often are infected with large numbers of cysticerci and may present with symptoms of coexisting parenchymal or ventricular disease. The basilar arachnoiditis can also lead to vasculitis that can cause lacunar infarctions (45). In some cases, cysticerci will erode into larger vessels causing large-vessel strokes. The arachnoiditis may also result in fibrosis, which can cause communicating hydrocephalus (31).

Anti-inflammatory drugs, especially corticosteroids, are the key to managing NCC of the basilar cisterns (3). Symptomatic relief can usually be accomplished with corticosteroids alone, but symptoms will usually recur if no other therapy is used. Hydrocephalus usually requires shunting. However, patients treated with shunting alone have a high mortality rate (86).

Although there are numerous case reports of cisternal cysticerci resolving with antiparasitic drugs, especially albendazole, no controlled trials of antiparasitic drugs in this form of disease have been reported (87). The response to antiparasitic drugs is less pronounced than in parenchymal disease, perhaps because of the prominent role of the host in parenchymal disease. Patients with cisternal cysticerci frequently relapse after treatment regimens that would have been effective in parenchymal disease (84). Case series suggest that treatment with corticosteroids, prolonged courses of antiparasitic drugs, and shunting for hydrocephalus

are associated with a better clinical prognosis than was noted in earlier series (32, 87). This has become the standard approach to these patients, but the evidence that it is superior to symptomatic therapy is minimal.

Spinal and Ocular Cysticercosis Surgical removal of individual lesions is the accepted therapy for cysticerci of the eye or spine (47, 88). For spinal subarachnoid disease, there is now considerable experience with chemotherapy (32). In cases with inflamed degenerating intramedullary cysticerci, corticosteroids may result in symptomatic relief, allowing resolution as the cysticercus degenerates (89). There are anecdotal reports of clinical cures of spinal intramedullary cysticercosis with antiparasitic drugs (90).

SUMMARY

NCC is now recognized as a major public health problem worldwide. Different forms of disease vary in their clinical presentation, underlying pathogenesis, and response to therapy. Management should be guided by knowledge of the natural history and underlying pathogenesis. Current data suggest that parenchymal disease can be treated symptomatically. Extraparenchymal disease is more severe and usually requires therapy with a combination of corticosteroids, antiparasitic drugs, and/or surgical procedures such as ventriculoperitoneal shunting. Carefully controlled studies guided by understanding of the pathogenesis and natural history are needed to better define the optimal management.

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