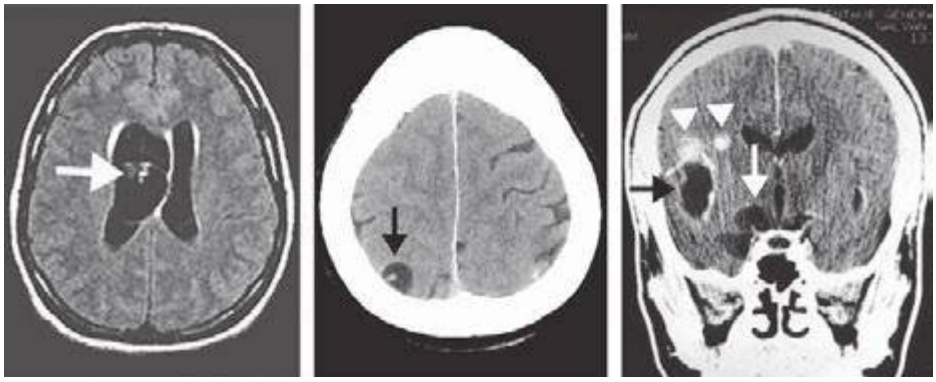

FIGURE 260-1 Neurocysticercosis is caused by *Taenia solium*. Neurologic infection can be classified on the basis of the location and viability of the parasites. When the parasites are in the ventricles, they often cause obstructive hydrocephalus. ***Left:*** Magnetic resonance imaging showing a cysticercus in the lateral ventricle, with resultant hydrocephalus. The *arrow* points to the scolex within the cystic parasite. ***Center:*** CT showing a parenchymal cysticercus, with enhancement of the cyst wall and an internal scolex (*arrow*). ***Right:*** Multiple cysticerci, including calcified lesions from prior infection (*arrowheads*), viable cysticerci in the basilar cisterns (*white arrow*), and a large degenerating cysticercus in the Sylvian fissure (*black arrow*). *(Modified with permission from JC Bandres et al: Clin Infect Dis 15:799, 1992. © The University of Chicago Press.)*

vision, dizziness, ataxia, or confusion, are often evident. Patients with hydrocephalus may develop papilledema or display altered mental status. When cysticerci develop at the base of the brain or in the subarachnoid space, they may cause chronic meningitis or arachnoiditis, communicating hydrocephalus, hemorrhages, or strokes.

Diagnosis The diagnosis of intestinal *T. solium* infection is made by the detection of eggs or proglottids, as described for *T. saginata*. More sensitive methods, including antigen-capture enzyme-linked immunosorbent assay (ELISA), polymerase chain reaction (PCR), and serology for tapeworm stage-specific antigens, are currently available only as research techniques. In cysticercosis, diagnosis can be difficult. A consensus conference has delineated absolute, major, minor, and epidemiologic criteria for diagnosis (Table 260-1). Diagnostic certainty is possible only with definite demonstration of the parasite (absolute criteria). This task can be accomplished by histologic observation of the parasite in excised tissue, by funduscopic visualization of the parasite in the eye (in the anterior chamber, vitreous, or subretinal spaces), or by neuroimaging studies demonstrating cystic lesions containing a characteristic scolex (Fig. 260-1). With improving resolution of neuroimaging studies, the scolex can now be identified in many cases. In other instances, a clinical diagnosis is based on a combination of clinical presentation, radiographic studies, serologic tests, and exposure history.

Neuroimaging findings suggestive of neurocysticercosis constitute the primary major diagnostic criterion (Fig. 260-1). These findings include cystic lesions with or without enhancement (e.g., ring enhancement), one or more nodular calcifications (which may also have associated enhancement), or focal enhancing lesions. Cysticerci in the brain parenchyma are usually 5–20 mm in diameter and rounded. Cystic lesions in the subarachnoid space or fissures may enlarge up to 6 cm in diameter and may be lobulated. For cysticerci within the subarachnoid space or ventricles, the walls may be very thin and the cyst fluid is often isodense with CSF. Thus, obstructive hydrocephalus or enhancement of the basilar meninges may be the only finding on CT in extraparenchymal neurocysticercosis. Cysticerci in the ventricles or subarachnoid space are usually visible to an experienced neuroradiologist on MRI or on CT with intraventricular contrast injection. CT is more sensitive than MRI in identifying calcified lesions, whereas MRI is better for identifying cystic lesions, scolices, and enhancement.

The second major diagnostic criterion is detection of specific antibodies to cysticerci. Although most tests using unfractionated antigen have high rates of false-positive and false-negative results, this problem can be overcome by using the more specific immunoblot assay. An immunoblot assay using lentil lectin purified glycoproteins is >99% specific and highly sensitive. However, patients with single intracranial lesions or with calcifications may be seronegative. With this assay, serum samples provide greater diagnostic sensitivity than CSF. All of the diagnostic antigens have been cloned, and assays using recombinant antigens are being developed. Antigen detection assays using monoclonal antibodies to detect parasite antigen in the blood or CSF may also facilitate diagnosis and patient follow-up. These assays are only now becoming available for patient care.

Studies have demonstrated that clinical criteria can aid in diagnosis in selected cases. In patients from endemic areas who had single enhancing lesions presenting with seizures, a normal physical examination, and no evidence of systemic disease (e.g., no fever, adenopathy, or chest radiographic abnormalities), the constellation of rounded CT lesions 5–20 mm in diameter with no midline shift was almost always caused by neurocysticercosis. Finally, spontaneous resolution or resolution after therapy with albendazole alone is consistent with neurocysticercosis.

Minor diagnostic criteria include neuroimaging findings consistent with but less characteristic of cysticercosis, clinical manifestations suggestive of neurocysticercosis (e.g., seizures, hydrocephalus, or altered mental status), evidence of cysticercosis outside the central nervous system (CNS) (e.g., cigar-shaped soft-tissue calcifications), or detection of antibody in CSF by ELISA. Epidemiologic criteria include

TABLE 260-1 DIAGNOSTIC CRITERIA FOR HUMAN CYSTICERCOSIS[a]

1. Absolute criteria
   a. Demonstration of cysticerci by histologic or microscopic examination of biopsy material
   b. Visualization of the parasite in the eye by funduscopy
   c. Neuroradiologic demonstration of cystic lesions containing a characteristic scolex
2. Major criteria
   a. Neuroradiologic lesions suggestive of neurocysticercosis
   b. Demonstration of antibodies to cysticerci in serum by enzyme-linked immunoelectrotransfer blot
   c. Resolution of intracranial cystic lesions spontaneously or after therapy with albendazole or praziquantel alone
3. Minor criteria
   a. Lesions compatible with neurocysticercosis detected by neuroimaging studies
   b. Clinical manifestations suggestive of neurocysticercosis
   c. Demonstration of antibodies to cysticerci or cysticercal antigen in cerebrospinal fluid by enzyme-linked immunosorbent assay
   d. Evidence of cysticercosis outside the central nervous system (e.g., cigar-shaped soft-tissue calcifications)
4. Epidemiologic criteria
   a. Residence in a cysticercosis-endemic area
   b. Frequent travel to a cysticercosis-endemic area
   c. Household contact with an individual infected with *Taenia solium*

[a]Diagnosis is confirmed by either one absolute criterion or a combination of two major criteria, one minor criterion, and one epidemiologic criterion. A probable diagnosis is supported by the fulfillment of (1) one major criterion plus two minor criteria; (2) one major criterion plus one minor criterion and one epidemiologic criterion; or (3) three minor criteria plus one epidemiologic criterion.

Source: Modified from OH Del Brutto et al: Neurology 57:177, 2001.