Neurocysticercosis

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ABSTRACT

Cysticercosis is a helmenthic infection involving pigs and man. Most cases of cysticercosis occur in developing countries. The disease is very rare in Islamic countries, as moslems are supposed to abstain from eating pork meat. In the last 10 years, reports of cysticercosis among moslems were published. Imigrants from endemic countries, who work as housemaids and food handlers played a role in transmitting the disease. Man becomes a definitive host if he ingests insufficiently cooked pork meat, which contains viable Iarvae of Taenia Solium or cysticerci. Neurocysticercosis denotes presence of a Taenia Solium larva cysts (Cysticercus cellulosae) in the brain parenchyma, meninges, or ventricular spaces. Neuroimaging by computerized tomography and magnetic resonance imaging are the best procedures to diagnose neurocysticercosis. Serological tests (EITB) or ELISA) are not sensitive as more than 50% of patients with neurocysticercosis have negative serology. Alpendazole and praziquentel are the most effective antihelmenthic drugs. Prevention of the disease and its complications as epilepsy is the management corner stone. A single dose of praziquentel for every emigrant from endemic areas will eradicate the adult tapeworm and reduce the incidence of neurocysticercosis. Physicians in moslem countries should be aware about the disease not only among immigrants but among moslems. We reviewed the available information about the disease epidemiologically, clinically, radiologically, laboratory tests, and methods of prevention.

Keywords: Neurocysticercosis, cysticercosis, epilepsy, helmenthic infection.

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Cysticercosis remains a major health problem in underdeveloped countries. Deficient sanitary disposal structures, poverty, ignorance and eating undercooked pork meat are the main causes of failure to control the disease. Forbidding pork protects moslems countries. Immigration to and from endemic areas has largely increased disease transmission in the last two decades.[1-3] Fortunately, the advances of radiological work improved our abilities to diagnose the disease. Despite the serious hazards of the disease, the preventive measures against it are definitely lacking.

Biological background. People become a definitive host when they become infected by eating raw or undercooked pork that contains viable larvae of taenia solium (TS) or cysticerci. Following ingestion of a living cysticercus, it develops in the small intestine into a tapeworm of 1 to 8 meters in length. The tapeworm releases terminal proglottids in stools bearing up to 50,000 eggs per proglottid.[4] When the intermediate host, pig and rarely man ingests the ova the gastric juices dissolve the thick outer shell of the ova to release the oncosphere. The oncosphere or immature larva which penetrates the gastrointestinal mucosa to bloodstream to be lodged in multiple body organs, especially muscles. The life cycle continues when humans eat undercooked pork meat that contains the viable cysticercus (Figure 1). In man 95% of infections occur by ingestion of ova through contaminated food or water. Autoinfection occurs in 5% of patients via the fecal-oral route.[5-6] The oncospheres commonly lodge in the small cerebral gray matter blood vessels. From there, the parasites migrate to chorodial plexus, and may end in ventricle or subarachoniod space.[7] Cysts involving the spinal cord are somewhat unusual.[8]

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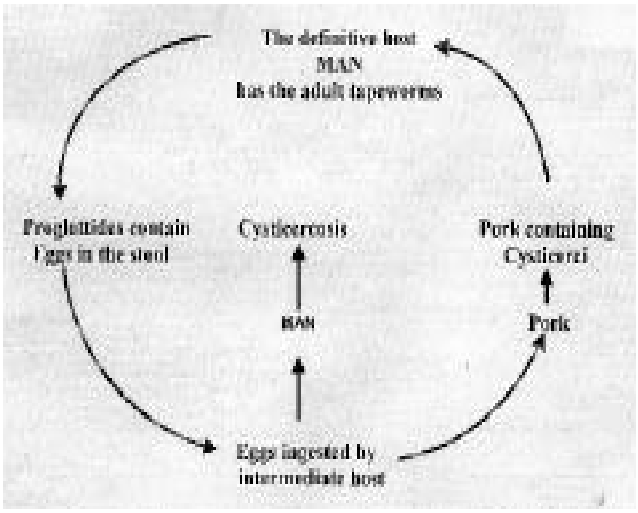

Figure 1 - Life Cycle of Taenia Solium.

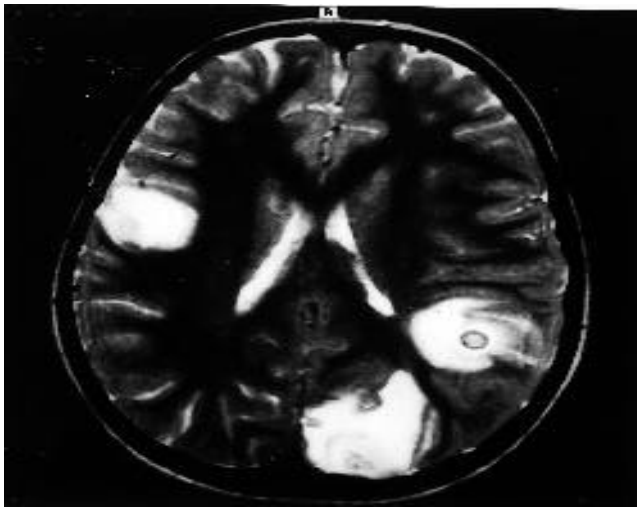

Figure 2 - MRI T2 image showing three cysticerci lesions with extensive surrounding edema.

Uncommonly cysts are found in the retina, heart, skeletal muscles and subcutaneous tissue.

Infection with TS infection leads to formation of specific IgG antibodies. The immune response is both humoral and cell-mediated.[9] Antigen-antibody reaction is unpredictable, ranging from a complete tolerance to an intense inflammation.[10,11] Different immunologic reactions may be found in the same patient.[10] The viable cyst (vesicular cyst stage), 3 to 18 mm in diameter contains clear fluid and scolex.[12] It evokes a minimal surrounding inflammation and remains viable from 2 to 10 years. If the osmotic barrier of the cyst wall breaks down, the clear cyst fluid thickens and becomes opaque, the cyst wall thickens, and hyaline degeneration and mineralization begin. The cyst wall begins to leak cyticercus cellulosae antigens, eliciting an intense inflammatory reaction in the adjacent brain, that intense inflammatory reaction causes the clinical symptoms. Calcification occurs over months to years.[12]

Epidemiology. Cysticercosis is the most common parasitic infection of the human central nervous system. Most cases of cysticercosis occur in developing countries. However, its prevalence is difficult to be accurately estimated, since a high percentage of patients remains asymptomatic.[10,13] The disease is endemic in Mexico,[14] Central and South America,[15] India,[16] and China[17]. Cases have been reported from Eastern Europe, Portugal, Africa, and Asia.[18-21] Recently, the increased immigration from Mexico and Latin America has resulted in an increased prevalence of neurocysticercosis (NC) in the United States, particularly in the Southwest.[22] In Los Angeles, neurocysticercosis accounted for more than 2% of all neurology and neurosurgery admissions.[8] Few cases of neurocysticercosis have been reported in American tourists visiting Mexico or Latin America.[8] Still NC was reported in US citizens who have never traveled to an endemic country.[16,2] A report from New York about about two Jewish families; 2 who had NC 1.3% of the Jewish community was seropositive for EITB (Jews are not supposed to eat pork). The seropositivity was associated with the presence of employees from endemic countries. Five Qatari children with NC were reported, none of them traveled out of the country.[23] Presumably, infection occurred in both occasions through ingestion of ova from housemaids and food handelers emigrated from endemic areas.

Clinical manifestation. Clinical presentations vary widely, range from no symptoms to life threatening disease. The most common manifestations are seizures (60%), symptoms and signs of increased intracranial pressure (ICP) (15%), mental changes (15%) and focal neurological deficits (10%). Brainstem dysfunction, cerebellar ataxia, sensory deficits, involuntary movements, dementia and hydrocephalus were infrequently seen.[24-32] The clinical signs vary according to the number of cysts, their central nervous system (CNS) location and the cyst's state of health.[30] In general, cysticerci are asymptomatic until they begin to degenerate. Degeneration induces inflammatory reaction that produces clinical manifestation. Carpio et al proposed a classification based on the viability and location of cyst; active when the parasite is alive, transitional if it is in degenerative state and inactive if it is dead. Each phase is subdivided into parenchymal and extra parenchymal.[30] The aim of this classification is to correlate between the clinical manifestation and each category.[31] Seizures occur in

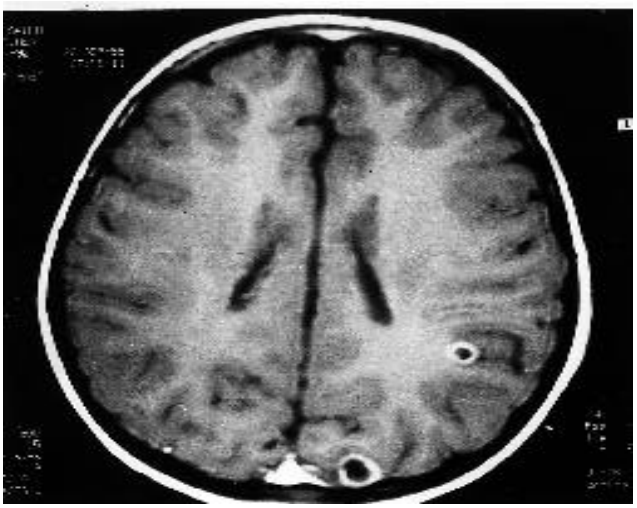

Figure 3 - MRI T1 enhanced with gadolinium showing two cysticerci without surrounding edema.

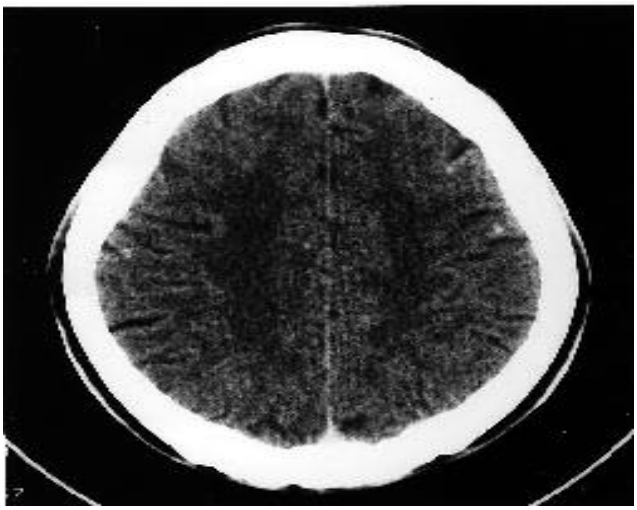

Figure 4 - Non enhancing CT scan showing multiple calcified cysts in a patient who presented with seizures.

82% of patients with active parenchymal cyst, 88% of patients with transitional parenchymal cyst and 75% of patients with inactive parenchymal cyst. Headaches, stiff neck, papilloedema and ICP occur in 86% of patients with active extraparenchymal and 100% of patients with transitional extraparenchymal. Patients with meningeal or ventricular cysticeri develop obstructive hydrocephalus leading to severe ICP with impending transtentorial herniation. A rare form of NC called "cysticercotic encephalitis" occurs in children and young women.[33-35] These patients infested with hundreds of brain cysts will develop early clinical symptomatology, beginning weeks to months after the original infestation. Such patients often develop focal neurologic deficits and signs of increased ICP. Individuals with dead calcified cysts seldom develop new neurologic symptoms and signs.

Seizures and neurocysticercosis. Seizures due to NC can be classified into two groups: 1- unprovoked remote symptomatic seizures in-patients who have alive cysticerci without inflammatory reactions or calcified lesions. 2- symptomatic seizures provoked by the inflammatory reaction around the degenerated cysts. This classification is important in management, as will be discussed later.

Radiological findings in neurocysticercosis. Computed tomography (CT) or magnetic resonance imaging (MRI) of the brain is extremely sensitive for neurocysticercosis diagnosis.[12,36] Computed tomography scan is superior to MRI in detecting calcified lesions, while MRI is more sensitive for earlier stages.[6,30,37-40] In the vesicular stage CT scan usually show small homogeneous contrast enhancing lesions, which are somewhat ill defined. As the cyst matures (3 to 18mm) non-contrast enhancing cysts will be seen without significant adjacent edema. Magnetic resonance imaging in early stage will show cystic lesion isointense to CSF without surrounding edema in T2 weighted image (Figure 2). Apathognomonic scolex of larva 2-4 mm is sometimes seen within the cyst as a high intensity dot.[41] As the cyst begins to degenerate; two stages are seen; the colloid stage and granular nodular stage.[12,30] In the colloid stage: the fluid changes to gelatinous fluid and the capsule thickens. Computed tomography scan demonstrates a ring-contrast-enhancing cyst surrounded with edema. While gadolinium enhancing MRI demonstrates ring on the capsule on T1-weighted images, with higher signal than adjacent brain branchyma (Figure 3). The colloid cystic fluid has higher signal in T1 than CSF sometimes isodense to adjacent branchyma.[30,38,41] In the granular nodular stage: the cystic fluid is replaced by granulation tissue and as a result shrinkage of the cyst occurs. This can be seen in MRI T2 image as isodense as adjacent parenchyma.[39,42] Over months to years, some cysts will calcify and be identified as calcified nodules 2 to 6mm in diameter.[12,30] If the parasite is living in CSF rich space, it tends to be larger in size (up to 100 mm), and is called hydropic racemose cyst. Calcification is rarely reported in these cases.[31] Ventricular cyst causes inflammatory reaction in ependymal lining which can be visualized by CT or MRI as hyperdense ependymal layer.[43,44] Identification of cysts within the ventricles or meninges is often difficult.[45] The cyst fluid is usually isodense with CSF and may not enhance with contrast on CT (Figure 4). The cyst may be detected on the basis of distortion or disproportionate enlargement of the third or fourth ventricles.

Serological test. Serological tests use crude, non-specific antigens and lack both specificity and

sensitivity due to cross-reaction with other helminthes infections, Serum ELISA test has sensitivity 70% with a specificity of about 50%. In the CSF it has a sensitivity of more than 80% and specificity of 90%.[46,47] Serological tests for cysticercosis have recently improved using specific protein antigen (EITB), an enzyme-linked immunoelectrotransfer blot assay which has 98% sensitivity and 100% specificity.[48,49] Unfortunately its sensitivity drops to 28% in patients who had single enhancing cyst. Patients with only calcified cysts appear to have a varying prevalence of antibody ranging from 10% if there is only one calcified cyst to 88% in patients with multiple calcified cysts.[49] In endemic countries individuals frequently have positive serology in the absence of active cysts on CT scan.[50] While only 30-50% of cases with NC diagnosed by CT\MRI have positive antibodies against cysticercosis.[50,51] This discrepancy may be due to the poor selection of patients; some patients may have both systemic cysticercosis and neurocysticercosis while others may have active calcified stage.

CSF findings in neurocysticercosis. 50% of patients will have normal CSF, increased intracranial pressure (40%), pleocytosis (45%), elevated protein (40%), low glucose 25%.[36]

Management. Management of NC included 1. management of cysticerci, if indicated, and management of the treatment complication. 2. management of NC complications. 3. methods of NC prevention.

1. Management of cysticerci. Since many asymptomatic patients have a benign natural history, questions have been raised as to whether all patients with neurocysticercosis require treatment.[52] The argument in favor of treatment stems from the observations that anticysticercus drugs rapidly kill cysticerci with collapse of the cysts within weeks instead of months and possibly avoid calcification.[53] Since drugs act by killing active cysticercus, the drugs are not useful in treatment of patients with dead calcified cysts. In children with active and ring enhancing cyst, it is recommended to start treatment rather than waiting for natural destruction because it is usually followed with calcification. The rapid cyst death is thought to reduce the incidence of neurologic sequellae. In a study from Mexico, patients with seizures from neurocysticercosis who were treated with praziquantel or albendazole had significantly improved seizure control compared to the nontreated control group.[54] Today, most symptomatic patients with neurocysticercosis are treated with praziquantel or albendazole. Both drugs are capable of killing cysticerci and TS tapeworm by mechanisms that are poorly understood.[55] Praziquantel appears to kill the scolex while albendazole appears to interfere with cyst wall metabolism. Praziquantel is well tolerated orally and has minimal side effects. The adverse effects usually include gastrointestinal upset, dizziness, fever, headaches, and occasionally a diminished sense of well-being.[56] Praziquantel is bound to serum albumin, but free praziquantel readily crosses the blood brain barrier with CSF/serum ratio 24%.[57] Concomitant administration of cimetidine often increases praziquantel blood levels, because of that some clinician recommend its use with praziquantel. While corticosteroids, phenytoin, or carbamazepine may lower praziquantel blood levels.[58-60] The usual dosage of praziquantel is 50mg/kg per day in three divided doses for 15 days. This will decrease the number of cysts by 50-80% in follow-up neuroimages over 3 to 6 months.[61] Albendazole crosses blood brain barrier with CSF\serum ratio 43% which is considerably higher than that of praziquantel (24%). This is why some authors suggested that albendazole might be better for neurocysticercosis treatment.[62,63] Albendazole oral dose is 15 mg/kg/day divided into 2-3 doses for 14 to 30 days. Side effects occur in 1% of patients and consist of pancytopenia, elevated serum transaminases, dizziness, headache, vertigo, abdominal pain, nausea and vomiting.

2. Management of complications. 1. The rapid death of the cysticerci with sudden release of its antigen into the surrounding brain leads to an intense reactive inflammation which sometimes causes increased clinical symptomatology.[56] Dexamethasone (12-24 mg/day) is often added to lessen the intensity of the inflammation.[64,65] 2. Management of seizures. Seizures are easily controlled with antiepileptic drugs (AEDS), but duration of therapy still needs further studies. In retrospective studies the risk of seizure recurrence after 2 years of treatment ranges from 12%-67%.[66-68] While others recommended treating seizures with inflamed NC as acute symptomatic seizures for several months.[31] Once the lesion has resolved on neuroimaging and the electroencephalogram is normal, treatment may be tapered off. While in patients having calcified lesion, treatment with AEDS for 1-2 years is recommended.[31,66-68] 3. Management of hydrocephalus. Patients who develop obstructive hydrocephalus from a chronic arachnoiditis blocking of intraventricular CSF pathways require placement of a ventriculoperitoneal shunt. Unfortunately, if often is difficult to maintain shunt patency as inflammatory debris or cyst debris may occlude the shunt. Death may occur from shunt malfunction or from brainstem vasculitis.[70] Intraventricular cysts usually require surgical removal.[71]

Prognosis. Most patients with neurocysticercosis have an excellent prognosis. Many remain asymptomatic throughout the entire course of infection. However, those with intraparenchymal cysts often develop transient acute symptoms during cyst degeneration, which usually resolve within months to 2 years. Some patients develop either focal or generalized epilepsy, which responds well to

AEDs. Rarely patient with large numbers of CNS cysts may die from the overwhelming CNS infection. Patients with hydrocephalus if untreated, may herniate and die.[72] Patients with intraocular cysts may lose vision.

3. Prevention. Prevention of the disease in non-endemic countries should be done by screening employees from endemic areas for tapeworm infection. A single dose of praziquentel 5-10 mg/kg results in 99% eradication of adult worm.[73-74] There will be a drop of NC new cases for a few years. One tablet of 600mg per person (0.27 dollar/tab) is cost effective. As a method of eradication, we suggest this type of treatment in new comers from endemic areas to Gulf countries without screening.

Cysticercosis is a serious disease which can threaten a patient's life. Employees from endemic area should be screened for tapeworm infection and a single dose of praziaquentel should be given to eradicate the adult tapeworm. The disease should be looked for even in-patients who have never been to endemic areas, not only expatriates but also muslims.

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