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Calcific neurocysticercosis and epileptogenesis

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Abstract

Neurocysticercosis is responsible for increased rates of seizures and epilepsy in endemic regions. The most common form of the disease, chronic calcific neurocysticercosis, is the end result of the host's inflammatory response to the larval cysticercus of *Taenia solium*. There is increasing evidence indicating that calcific cysticercosis is not clinically inactive but a cause of seizures or focal symptoms in this population. Perilesional edema is at times also present around implicated calcified foci. A better understanding of the natural history, frequency, epidemiology, and pathophysiology of calcific cysticercosis and associated disease manifestations is needed to define its importance, treatment, and prevention.

Neurocysticercosis is a major cause of seizures and other neurologic problems in many less developed countries¹ and a significant health concern in developed countries as well, mostly due to migration of infected persons.² Over the last two decades the development of MRI and CT imaging, effective and safe cysticidal drugs, and specific and relatively sensitive serologic tests have given rise to a renaissance in our understanding of the disease and efficacy of treatments. Much of our increased understanding has focused on disease associated with viable or degenerating cysts, broadly referred to as “active” cysticercosis,

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and development and use of cysticidal agents.^{3,4} Disease manifestations including seizures caused by viable or degenerating cysts are well described. In contrast, few if any symptoms have been attributed to chronic calcific cysticercosis. Recently, evidence from a number of types of studies has implicated this condition as a cause of seizures or focal neurologic problems in this population. Here we review supporting studies and public health implications, define major unanswered questions, and suggest approaches to better understand calcific cysticercosis and its complications.

Life cycle

Humans harbor the tapeworm that is acquired by eating poorly cooked pork containing cysticerci of *Taenia solium*. Ova or proglottids containing ova are excreted in the feces and when ingested by free roaming pigs develop into cysts primarily in the muscles and brain. The usual life cycle is fulfilled after humans ingest undercooked pork. Ova, accidentally ingested by humans, also develop into cysts, mostly in the brain, muscle, and subcutaneous tissues, and this condition is referred to as cysticercosis.⁵

Course of infection

Although incompletely documented, a reasonable view of the natural history can be ascertained from pathologic, radiologic, and parasitologic studies. Following ingestion of ova, the released oncospheres migrate by way of the bloodstream and under favorable conditions develop into mature cysts after 2 to 3 months.^{5,6} Viable cysts in the brain are usually roundish, about 10 to 20 mm diameter, and incite little if any inflammation or symptomatology. But, after varying periods of time or following cysticidal treatment, an immune mediated inflammatory response is initiated that commonly causes seizures or other symptoms.⁷ The cystic larva continues to degenerate and eventually a granuloma forms containing host inflammatory cells and varying amounts of parasite remnants or antigens.⁸ Cysticercal granulomas have a peculiar propensity to calcify and in endemic populations one or more calcified granulomas are by far the most frequent finding seen on brain imaging.⁹⁻¹¹

Cysticercosis and epilepsy

While many patients present with single or groups of seizures at various stages of this disease, not all patients develop recurrent seizures, or epilepsy. There are three possibly different scenarios concerning the relationship between cysticercosis and epilepsy: 1) causal relationship, namely, cysticercosis as the cause of focal epilepsies; 2) non causal relationship or simple overlap of two independent and unrelated diseases; and 3) dual pathology. At present there is overwhelming evidence supporting neurocysticercosis as a cause of seizures and epilepsy. Because neurocysticercosis, particularly calcific cysticercosis, is so common in endemic regions (see below), it is likely that two pathologies known to incite seizure activity will be present in some individuals¹² and whether there are interactions or dual pathology between two conditions is speculative.¹²

Multiple causes of seizures in cysticercosis

There are multiple ways that cysticercosis can cause seizures, mostly as direct or indirect effects of inflammation. For instance, seizures occur early in the disease in the setting of intense inflammation associated with viable or degenerating cysts. They can also occur later as a result of infarcts related to vasculitis and thrombosis of penetrating vessels from sub-arachnoid cysticercosis.¹³ Encephalomalacia and gliosis, end results of prior inflammation, have also been documented in cysticercosis and are other potential causes of seizure activity. Finally, a growing body of evidence suggests the more chronic cysticercal granuloma is

associated with seizure activity either before or after calcification; the latter situation is the focus of this summary.

Cause of punctate brain calcifications

Numerous conditions are known to result in cerebral calcifications and in some diseases the nature and pattern of the calcifications can suggest a diagnosis. Small, punctate, single or multiple calcifications are common in *T solium* endemic populations and there is good evidence to indicate that most are calcified cysticercal granulomas. First of all, multiple or single punctate cerebral calcifications are infrequent in other infectious diseases and commonly distinguishable. Toxoplasmosis, cytomegalovirus, and rubella infections can cause cerebral calcifications after congenital infections but only those caused by toxoplasmosis can be easily confused with calcific cysticercosis.¹⁴ However, their presence early in life and associated clinical settings are distinctive. The patterns and nature of the calcifications in cytomegalovirus and rubella are different. Untreated cerebral tuberculosis lesions are not usually calcified although they tend to calcify after resolution following therapy.¹⁵ In contrast, serial observations of treated and untreated patients with neurocysticercosis have documented the frequent evolution of cystic lesions to typical punctate calcifications.¹⁶ Biochemical and histologic studies of typical calcified lesions detected collagen characteristic of cysticerci and not host.¹⁷ Single enhancing lesions are well studied in India and these are mostly degenerating cysticerci that commonly calcify.¹⁸ Therefore the presence of characteristic cerebral calcifications in the correct clinical setting is mostly chronic cerebral cysticercosis.

Association of calcifications with seizures

Cerebral calcifications are a common finding in persons with seizures or epilepsy in endemic populations and this finding suggests a role in the pathophysiology of seizures in these groups. The proportions of patients with seizures or epilepsy and neurocysticercosis and the presence of one or more typical cerebral calcifications are shown in the table. Some of the differences among studies may be due to failure to differentiate patients experiencing acute symptomatic seizures in those with active (live or degenerating cysts) lesions from those with epilepsy. Although there is variability between studies, calcifications are common and can be the most frequent finding on brain imaging in certain populations. Prevalences range from 9% to 18% in randomized studies of endemic populations and up to 83% in selected populations with seizures.¹⁹ One of the more definitive studies¹¹ found that calcifications alone were more frequent in those with seizures compared to those without seizures in a well-defined rural population. Typical calcifications were found in 36% and 35% of persons with seizures in two different villages compared to 15% and 9% in matched controls without a history of seizures. Cysts or degenerating cysts in this study were responsible for about 25% of the seizures in patients with cysticercosis.^{11, 20} In an endemic village of Peru, 9 (31%) of 29 patients with epilepsy demonstrated lesions compatible with cysticercosis and 6 (20.7%) showed only calcifications. In contrast, only 10.5% of 38 without a history of seizures had lesions compatible with cysticercosis and all were calcifications (Cysticercosis Working Group in Peru 2003, unpublished). Similarly, in an unpublished study of a prospectively evaluated rural Honduran population with neurocysticercosis and epilepsy, Medina et al. found that 76% had only calcifications, 9% cystic lesions, and 15% mixed lesions. There are at least three pieces of evidence that suggest calcified lesions play a role in epileptogenesis: 1) high prevalences of typical cerebral calcifications in patients with seizures or epilepsy in the absence of other etiologies,^{21, 22} 2) a positive correlation between endemic populations with increased proportions of calcification and seizure activity, and 3) an increased risk of continued seizure activity due to single cysticercal granuloma that calcify.^{23, 24}

Calcifications can be foci of seizure activity

That some calcified lesions are able to initiate seizure activity comes from a correlation of the signs, symptoms, and abnormal EEGs with the neuroanatomic location of specific calcifications on imaging. Electroclinical activity correlates with the location of brain calcifications in 26% to 55% of the cases.³⁵⁻³⁷ As suggested by the authors of these studies failure to localize seizure activity to more of the calcified lesions can be due to inherent limitations and constraints of the methodology, duration, or timing of the studies, spread of electrical activity from silent regions along anatomic pathways, or other lesions or processes as the cause of seizure activity.

Perilesional edema and calcifications

Perhaps the most direct evidence implicating calcified lesions as foci of seizure activity and other focal neurologic manifestations is the episodic appearance of perilesional edema^{21,38-41} often accompanied by corresponding clinical findings. Perilesional edema appears as a bright signal using MRI FLAIR or T2 imaging. It is almost always accompanied by enhancement around the calcified focus. This phenomenon is now well documented and has been noted by multiple investigators working in various geographic regions.^{4,21,22,27,38,42} Interestingly, in any given individual, one subset of calcifications may undergo recurrent episodes of perilesional edema, while another subset remains quiescent. Because it is detectable on imaging, its occurrence, potential treatments, and prevention can easily be assessed.

There is little known about the natural history, clinical importance, and pathophysiology of perilesional edema. From anecdotal reports, collected series, and previous observations in patients with seizures, perilesional edema related to calcifications appears to be relatively frequent, ranging from 23% to about 35% in patients with calcified neurocysticercosis and a history of seizures.^{4,21,27} Associated symptoms range from frequent and disabling manifestations that usually consist of seizures or focal neurologic disease to no outward clinical manifestations.

The pathophysiology of perilesional edema is unknown. Calcified granulomas are a result of the host's inflammatory response to viable or degenerating cystic larva, so one hypothesis is that there are episodic host inflammatory responses to residual antigen that is intermittently released or recognized by the host similar to the responses provoked by anticysticidal drugs. One perplexing aspect of the phenomenon is that only certain calcified foci are capable of developing perilesional edema, so only lesions that undergo perilesional edema would be predicted to contain or expose antigen while others would not. In support of this idea a recent report correlated the presence of a recognizable scolex in specific calcified lesions with the presence of perilesional edema.⁴² The authors contend that the presence of the scolex is indicative of the presence of recognizable parasite remnants and therefore the likely presence of parasite antigen in these particular calcified lesions. However, parasite antigen has not been directly detected in these lesions, and whether the edema is associated with inflammation and the nature of the inflammation have not been established. It is not known whether the presence of calcium in a lesion is solely a visual marker of past or present pathology or plays a direct or indirect role in the induction of seizures. Direct calcium toxicity has been suggested and there are some data indicating that lesions that calcify are associated with increased seizure activity compared to those that fail to calcify.⁴³ Calcium may also form an insoluble matrix that could release incorporated antigens at certain times. Direct injury to brain tissues associated with single calcified or non calcified cysticercal granulomas is another possible reason for continued or recurrent seizure activity. This is suggested by the presence of gliosis around foci associated with seizure activity

compared to clinically silent lesions as noted by the use of specific MRI magnetization transfer sequences.^{44,45} Another hypothesis is that edema can be caused by seizure activity itself. Focal edema has been documented in lesions in a few cases with partial status epilepticus from other causes.⁴⁶⁻⁴⁸ However, this finding is rare and the MRI pattern of edema associated with perilesional edema in cysticercosis is most consistent with vasogenic edema resulting from blood–brain barrier breakdown of the lesion. In contrast the edema does not have the radioimaging appearance of cytotoxic edema resulting from cell swelling associated with prolonged seizures.²¹

Conclusions

Neurocysticercosis is an important and preventable cause of seizures and epilepsy in endemic populations. The authors, including persons with expertise in various aspects of parasitology, *T. solium* infections in humans and animals, epidemiology, epilepsy, and public health, agree that there is a body of consistent, reproducible, and believable data implicating chronic calcific cysticercosis as a cause of seizure activity and other focal neurologic manifestations. Because brain calcifications due to cysticercosis occur in over 10% of heavily endemic populations, even a modest increase in seizure rates would affect large numbers of individuals. Because what is presently known is based mostly on anecdotal reports and serial observations, prospective and controlled studies are required to confirm and quantify these initial observations, to determine the natural history and importance, and to understand the pathophysiology. The latter would likely be difficult to understand solely from human studies; therefore, it would be useful to develop an appropriate porcine model for cysticercosis and seizures.

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Table
Brain calcifications in patients with seizures or neurocysticercosis (NCC)

Location	Country	Population	Characteristics	Calcifications only	% Active lesion without calcifications	Time	Reference
Urban	Ecuador	NCC	Epileptics	26	74	Retrospective	4
Urban	Ecuador	NCC	First seizure	11	89	Prospective	25
Urban	Portugal	NCC	Epileptics	83	17	Retrospective	26
Urban	India	Seizures	Uncontrolled partial epilepsy	40	31	Retrospective	27
Urban	USA	NCC	Pediatric, mostly seizures	30	70	Retrospective	28
Urban	Mexico	Epilepsy	Adults	26	24	Retrospective	29
Urban	India	Seizures	Pediatric, partial seizures	12	10	Retrospective	30
Urban	Brazil	Seizures	Any type	13	10	Retrospective	31
Urban	Colombia	Seizures	>10 y	15	7	Prospective	32
Urban	Honduras	Seizures	Suspected NCC	68	16	Prospective	33
Rural	Ecuador	Seizures	Population-based (field)	35	19	Prospective	10
Rural	Mexico	Seizures	Tapeworm carriers and seropositive	40	30	Prospective	34
Rural	Guatemala	No seizures	Tapeworm carriers and seropositive	5	8		
Rural	Guatemala	Seizures	Population-based (field)	36	12	Prospective	11, 20
			Population-based (field)	35	12		
		No seizures	Population-based (field)	15	8		
			Population-based (field)	9	0		
Rural	Honduras	Seropositive	Population-based	19	4	Prospective	9
		Seronegative	Population-based	16	3		
Rural	Mexico	Seropositive	Asymptomatic, population-based	25	0	Prospective	19
		Seronegative	Asymptomatic, population-based	8	0		