

Host response in childhood neurocysticercosis

Some pathological aspects

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Abstract. The autopsy findings in 18 cases of childhood neurocysticercosis are described. Twelve of the children had died from other causes. Asymptomatic disease correlated with immunodeficiency and with negative serum anticysticercosis antibody test.

Key words: Cysticercosis – Brain – Autopsy – Immunodeficiency.

The clinical manifestations of neurocysticercosis are protean. This diversity is sometimes reflected in the anatomopathological findings. It is recognized that the prognosis is influenced by such factors as: the degree of inflammatory reaction, the localization of the cysticerci in the brain and impaired CSF circulation. However, the basic mechanisms determining the outcome are still unknown, and for this reason it is important to study this peculiar host-parasite relationship in different populations. The postmortem findings of 18 cases of neurocysticercosis in children are presented; the data were taken from the autopsy files of the Department of Pathology at the National Institute of Pediatrics in Mexico City.

The general features of this disease appear in Table 1. The incidence is 0.5% of all pediatric autopsies. This figure is significantly lower than that found in adults, probably because babies have less opportunity to ingest contaminated food. In adolescents, the incidence increases dramatically 3 to 4 times. Ninety percent of the cases are between 7 and 15 years old. The age average is 9 and our youngest patient was an 11-month-old baby boy. In 8 patients neurological symptoms were present with increased intracranial pressure and/or epilepsy. Nine cases were non-symptomatic and the parasites were found in children dying from other diseases. One child died on arrival and his past clinical history is not known. In two cases the infection was systemic involving the heart, lungs, liver and lymph nodes in addition to the brain. One of these cases had leukemia and teniasis. The coexistence of cysticercosis and tenia is unexpectedly very low. The racemosus form of cysticerci is characterized by a large-size, lobulated bladder and absence of scolex; this form is located usually in

the cisterns and ventricles, and in such cases the infection is always severe and in adults. In children, this form is very uncommon and in this series was found in only one case.

In Table 2, the associated diseases are shown. Cysticercosis was the cause of death in six patients (one-third of the total.) It was noteworthy that seven cases had some kind of immunodeficiency. The other four cases had a variety of infectious diseases associated with malnutrition. The association with depressed immunity perhaps explains some of the clinical features of this disease. As shown in Table 3, it would appear that most of the immunodeficient

Table 1. Cerebral cysticercosis in children: general features in 18 cases

Total autopsies	3424
Cerebral cysticercosis	18 (0.5%)
Age average	9 years
Range	11 months–15 years
Girls	11
Boys	7
Symptomatic	8 (40%)
Asymptomatic	9
Unknown	1
Systemic cysticercosis	2
Taeniasis	1
Cisticercus racemosus	1

Table 2. Cerebral cysticercosis in children: basic disease in 18 autopsy cases

Cysticercosis	6 (33%)
Leukemia	3
Lupus erythematosus	2 (39%)
Ataxia-telangiectasia	1
IgA deficiency/lymphoma	1
Gastroenteritis	1
Hepatitis	1
Pneumonia	1 (26%)
Cervical cellulitis	1
Poliomyelitis	1

Table 3. Cerebral cysticercosis in children: basic disease and clinical presentation

	Total	Symptomatic
Cysticercosis	6	6
Immunodeficiency	7	2
Others	4	0
Total	17	8

Table 4. Cerebral cysticercosis in children. (Numbers in parentheses indicate cases with immunodeficiency)

Characteristics	Symptomatic	Asymptomatic	Total
Multiples parasites	5	3 (2)	8
Basal meningitis	5 (1)	0	5
Severe inflammation	4 (1)	0	4
Viable parasites	2 (1)	7 (5)	9
Intraventricular	3	1	4
Total	8	9	

Table 5. Cerebral cysticercosis in children: serum anticysticercus antibodies (immunoelectrophoresis). (Numbers in parentheses indicate cases with immunodeficiency)

Antibodies	Total	Symptomatic	Asymptomatic
Positive	4	4	0
Negative	6	3 (1)	3 (2)
Total	10	7	3

patients had no symptoms; only 2 of 11 cases were symptomatic.

With regard to the tissue response, some of the cases had intact parasites surrounded by compressed tissue with astrocytic proliferation and scant inflammatory exudate. Others had hyalinized or calcified structures surrounded by inflammatory cells and a capsule of connective tissue. In addition to the local reaction around the parasite, we found two cases with an "encephalitic" picture with cerebral edema and perivascular mononuclear infiltration. Basal meningitis with obstruction of the cisterna was present in five patients. In Table 4, the relationship between the tissue response and clinical status is presented. Basal meningitis and severe inflammation were seen only in the symptomatic patients; the intraventricular location also occurred more frequently in this same group. In contrast, living parasites without inflammation occurred more frequently in asymptomatic and immunodeficient patients.

We tried to correlate the immunological response with the clinical picture (Table 5). We studied ten cases looking for antibodies in their sera obtained at autopsy. All four cases with positive serology were symptomatic with a very active inflammatory reaction. In six patients, the serological test was negative: three of them had no symptoms; in the symptomatic group one was immunodeficient and the remaining two were treated with corticosteroids.

It can be concluded that cysticercosis in children is a very polymorphic disease with a variability related to the location of the parasite and perhaps to the immunological response. The nonsymptomatic patients are of much interest. There is probably a mechanism that permits some parasites to evade host recognition, which enables them to survive in the human nervous system.