EPIDEMIOLOGY OF NEUROCYSTICERCOSIS IN BRAZIL

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SUMMARY

A revision of literature was done with the objective of tracing an epidemiologic profile of neurocysticercosis (NCC) in Brazil. The prevalence was 0.12-9% in **autopsies**. The frequency was 0.03-7.5% in clinical series and 0.68-5.2% in **seroepidemiological** studies. The disease corresponds to 0.08-2.5% of **admissions** to general hospitals. Patient **origin** was rural in 30-63% of cases. The most involved **age** range (64-100%) was 11 to 60 years, with a predominance (22-67%) between 21 and 40 years. The male **sex** was the most affected (51-80%). In the severe forms there was a predominance of urban origin (53-62%) and of the female sex (53-75%). The period of **hospitalization** ranges from 1 to 254 days and 33 to 50% of patients suffer 1.7 ± 1.4 admissions. The **clinical picture** was variable, with a predominance of epileptic syndrome (22-92%) and intracranial hypertension (19-89%). Psychiatric manifestations were associated in 9-23% of patients. **Lethality** was 0.29% in terms of all diseases in general and 4.8-25.9% in terms of neurologic diseases. The **asymptomatic** form was detected in 6% of patients in clinical serie and in 48.5% of case from autopsies. The racemose form and ventricular localization also was observed as asymptomatic form. Among the patients with cutaneous cysticercosis 65% of them showed neurologic manifestations.

KEYWORDS: Neurocysticercosis; Epidemiology; Cysticercosis.

INTRODUCTION

Cysticercosis is the result of infection of humans and swine by the larva *Cysticercus cellulosae* of the parasite *Taenia solium*, or "solitary", producing reactions in the tissue involved locally or at a distance, in 3 different forms: swine cysticercosis, taeniasis, and human cysticercosis. In **swine cysticercosis**, when the pig is the habitual intermediate host, the larval form develops in 9 to 10 weeks. The size of parasites in swine ranges from 1.5 to 12.5 mm[71]. In **taeniasis**, the cycle is completed when man, the definitive host, ingests contaminated pork meat. The adult form develops after 62 to 72 days and the expulsion of the first proglottides, or "tapes", starts. *T. solium* may remain in the human intestine for as long as 25 years[1], and its embryos may reach any tissue but show a greater tropism for the central nervous system (CNS), which they reach through the choroid plexus and subarachnoid blood vessels. As cysticerci in the CNS, they may remain viable for one to 33 years[4, 31, 33], with a mean of five years, before triggering symptoms, or they may remain silent.

Human cysticercosis occurs through two mechanisms: self-infection and hetero-infection. The first type occurs when an individual brings his dirty hands to his mouth or through a mechanism of retroperistalsis which has not yet been confirmed[1]. The second type occurs through the ingestion of contaminated food or water as a result of precarious personal hygiene and of the use of fruits and vegetables from orchards and gardens fertilized or irrigated with material contaminated with *T. solium*. The possibility of contamination by the ingestion of pork meat containing posoncospheres in immunodepressed patients has also been reported[73].

The factors contributing to the endemic nature of cysticercosis are: **1.** rearing contaminated swine; **2.** poor

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disposal of the excrements from infected individuals; **3**. presence of vegetable gardens and/or orchards near pig pens, especially when irrigated with contaminated water or fertilized with human feces; **4**. water sources near pig pens and latrines; **5**. precarious environmental and personal hygiene habits such as ingestion of unwashed food and food handling with dirty and contaminated hands; **6**. manipulation of contaminated meat; **7**. irregular or absent inspection at slaughterhouses and butcher shops; **8**. consumption of raw or poorly cooked pork, especially in the presence of a cysticercus, popularly known in Brazil as "pipoquinha", "canjiquinha", "quireririnha", "carocinho", "pedrinha", or "bexiguinha"; **9**. deficient sanitary education. The literature has reported contamination by arthropods and birds through the wind[1, 37].

Thus, transmission of the taeniasis/cysticercosis dyad occurs from swine to man, from man to swine, and from man to man. In other words, where there is animal cysticercosis, man is always responsible for its dissemination.

In the Brazilian literature, most of the information about NCC is based on clinical data[2, 9, 11, 12, 15, 20-23, 25, 26, 28-30, 34-35, 39, 47, 48, 65, 67, 68, 72, 75, 78, 80, 85, 87, 88, 94], on complementary tests[3, 8, 13, 14, 40, 41, 44, 49-52, 54, 56-58, 61, 62, 70, 82-84, 86, 95] and on autopsy data[4-7, 32, 42, 43, 45, 46, 59, 60, 63, 64, 66, 69, 74, 90-92, 96]. Few seroepidemiologic studies are available[10, 77, 93, 96, 97].

The **objective** of this paper is to try to trace an epidemiologic profile of NCC in Brazil.

METHODOLOGY

In an extensive review up to 1995 of the Brazilian literature on NCC, we selected 94 papers which reflected epidemiologic characteristics or that could be used for this purpose when taken together with others. Forty-six of these reports involve clinical, seroepdemiological and autopsy casuistic. The others 48 are studies on specific aspects of NCC. Data were gruped in terms of frequency at autopsy,
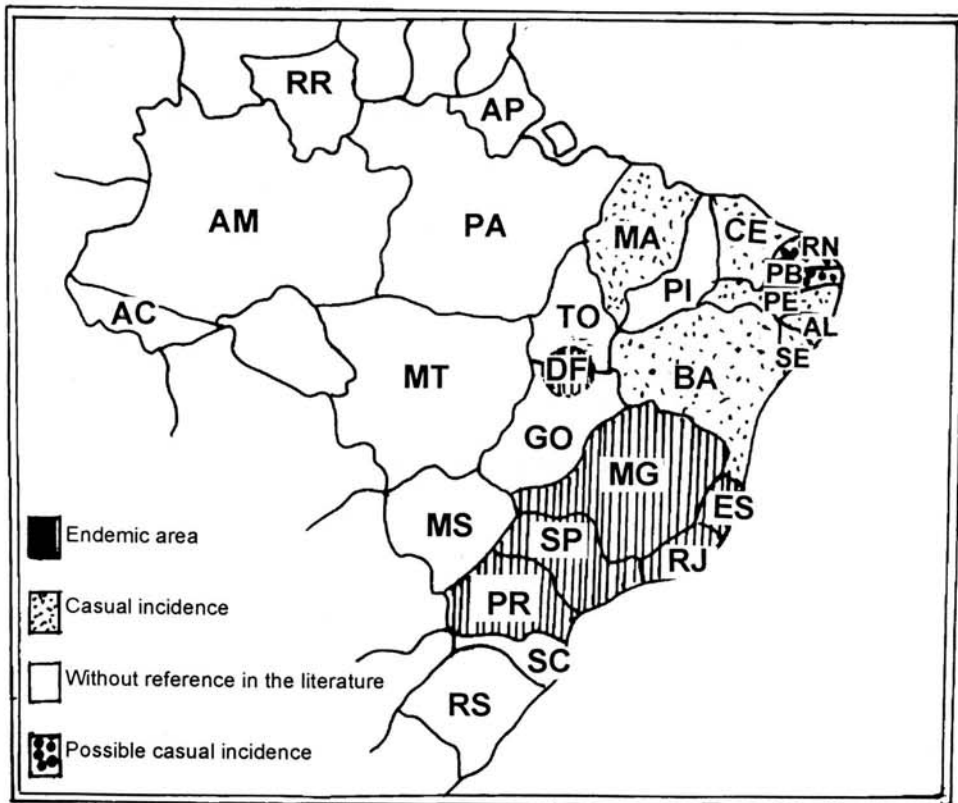

Fig. 1: Brazilian national distribution of the human neurocysticercosis.
(**PR** = Paraná; **SP** = São Paulo; **MG** = Minas Gerais; **DF** = Federal District; **RJ** = Rio de Janeiro; **ES** = Espírito Santo; **BA** = Bahia; **MA** = Maranhão; **CE** = Ceará; **PE** = Pernambuco; **PB** = Paraíba; **RN** = Rio Grande do Norte; **RS** = Rio Grande do Sul; **SC** = Santa Catarina; **MS** = Mato Grosso do Sul; **MT** = Mato Grosso; **GO** = Goiás; **TO** = Tocantins; **PA** = Pará; **AP** = Amapá; **AM** = Amazonas; **AC** = Acre; **RO** = Rondônia; **RR** = Roraima).

TABLE 1

Frequency of cysticercosis at autopsy (**AUTOPSY**), during clinical follow-up (**CLINICAL**), and in seroepidemiologic studies (**SE**) in General Hospitals and/or Neurosurgery and Neurology services reported in the literature, taking into consideration the author (**AUTHOR**), survey period (**PERIOD**), Brazilian State (**BS**) in which the study was conducted, and year of publication (**YEAR**).

AUTHOR	PERIOD	AUTOPSY		SE		CLINICAL		BS	YEAR
		%	nº	%	nº	%	nº		
ALMEIDA****[7]	1907-1915	0.71	1822	–		–		SP	1915
TRÉTIAKOFF & SILVA****[92]	1921-1924	3.6	250	–		–		SP	1924
GALVÃO****[42]	1916-1923	1.6	997	–		–		SP	1928
POVOA****[64]	1932	1.03	1073	–		–		SP	1934
SALLES[74]	1931-1934	0.12	4000	–		–		SP	1934
LANGE***[49]	1925-1940	–		–		0.31	4200	SP	1940
PINHEIRO & MELLO****[63]		0.43	465	–		–		SP	1941
PUPO et al.****[66]	1915-1944	1.5	1000	–		10 cases		SP	1945/46
MONTENEGRO[59]	1923-1927	2.5	312	–				SP	1946
BROTTO[16]	1930-1946	–		–		0.36	12361	SP	1947
SPINA-FRANÇA[81]	1947-1950	–		–		2.98	2273	SP	1956
CANELAS[19]	1945-1961	–		–		3.39	4900	SP	1962
SILVA et al.[78]		–		–		0.06	4600	PE	1965
CAMARGO LIMA[18]	(20 anos)					355 cases		SP	1966
MEGA & LISON***[58]						3.08	2500	SP	1967
BENÍCIO[76]		–		–		0.03	9077	PE	1970
REIS[76]		–		–		1.15	–	SP	1970
HELLMEISTER & FARIA[46]	1965-1970	1.78	1013	–		–		SP	1973
GUIDUGLI NETO & MATOSINHO FRANÇA[45]	1961-1974	0.86	3587					SP	1977
FACURE et al.[34]	1958-1977					11 cases		SP	1978
QUEIROZ & MARTINEZ[69]		0.3	4000	–		–		BA	1979
GOBBI et al.[43]	1960-1980	1.6	2306	–		–		MG	1980
MANREZA**[57]	1945-1980	–		–		1.76	–	SP	1982
TAKAYANAGUI & JARDIM[89]	1956-1979	–		–		7.5	500 cases	SP	1983
						As.=6%			
MOREIRA[60]	1960-1983	27 cases		–		49 cases		SP	1984
UEDA et al.[93]		–		0.87	824	–		SP	1984
VIANNA et al.[97]		–		5.2	1122	–		DF	1986
MACHADO et al.*[55]	1979-1985	–		–		0.19	126968	SP	1988
ALMEIDA & LIMA F⁰[26]	1980-1987	0.45	1773	–		–		CE	1988
TAVARES et al.[91]	1977-1988	9.0	1160	–		–		MG	1988
ALMEIDA et al.*[5]	1980-1982	1.5	200	–		–		SP	1989
GUERREIRO et al.***/**[44]	1985-1987	–		–		18 cases		SP	1989
ARRUDA et al.[10]	1987	–		0.68	1168	–		PR	1990
CHEQUER & VIEIRA[24]	1987-1989	–		–		45 cases		ES	1990
CLEMENTE & WERNECK[27]	1981-1989	–		–		100 cases		RJ	1990
VAZ et al.[96]		–		2.30	821	–		SP	1990
VIANNA et al.[99]		–		–		12.9	520	DF	1990
ANTONIUK et al.***/**[8]	1979-1988	–		–		24 cases		PR	1991
BRUCK et al.**[17]		–		–		34 cases		PR	1991
VIANNA et al.[98]	1967-1984	1.6	1520	–		–		DF	1991
SPINA-FRANÇA et al.***[83]	1929-1992	–		–		1.13	135000	SP	1993
SILVA-VERGARA[77]	1992	–		1.94	1080	–		MG	1994
AGAPEJEV[4]	1969-1990	1.85	3681	–		0.30*	132480	SP	1994
		As.=48.5%				6.1	3225		
FERREIRA et al.**[36]	1983-1990	–				10 cases		DF	1994
TAVARES****[90]	1938-1988	0.77	20741	–		12.2	188	MG	1994
COSTA-CRUZ et al.[32]	1971-1993	1.22	2862			–		MG	1995

As. = asymptomatic; **nº** = number of cases studied, from which those with cerebral cysticercosis were selected; * = Studies in general hospitals; ** = Studies limited to pediatric cases; *** = Study based on the results of complementary tests; **** = Studies conducted on psychiatric patients; **BA** = Bahia; **CE** = Ceará; **DF** = Federal District; **ES** = Espírito Santo; **MG** = Minas Gerais; **PE** = Pernambuco; **PR** = Paraná; **RJ** = Rio de Janeiro; **SP** = São Paulo.

(**Note**: Except for the data obtained in seroepidemiologic studies, all other data refer to the frequency of cerebral cysticercosis and not to cysticercosis in general.)

seroepidemiologic studies and hospital admissions. The frequency of onset of NCC was evaluated in terms of patient origin, age range, sex, predominant clinical picture, lethality and appearance of the asymptomatic form. The statistical median of these frequencies was obtained whenever possible.

RESULTS

Table 1 lists the studies that are of fundamental importance for the characterization of the Brazilian epidemiology of NCC.

The States of São Paulo (SP), Rio de Janeiro (RJ), Paraná (PR), Minas Gerais (MG), Espírito Santo (ES) and the Federal District (DF) are considered to be endemic areas (**Figure 1**). The occasional presence of NCC has been reported in the States of Bahia (BA)[61], Maranhão (MA)[23], Pernambuco (PE)[12, 40] and Ceará (CE)[6] and among patients proceeding from the States of Paraíba (PB) and Rio Grande do Norte (RN)[12, 40].

A total of 52,789 cases of NCC were recorded during the period from 1907 to 1993 in autopsies carried out in MG, PR and SP, corresponding to a mean of 600 cases/year, or 1.65 cases/day. The clinical series investigated in Neurology Services correspond to 38,276 cases of NCC from 1915 to 1993, with a mean of 491 patients/year or 1.35 patients/day. Among a total of 259,448 patients admitted to general hospitals over a period of 24 years, 623 were due to NCC, corresponding to 26 admissions/year, or 2.17 admissions/month.

The prevalence of NCC detected at autopsy (**Table 1**) ranged from 0.77 to 9% (median = 1.4%) in MG, from 0.12 to 3.6% (median = 1.5%) in SP, and from 0.3 to 0.45% in the Northeast Region (States of BA and CE). Among clinical series seen at Neurology and Neurosurgery services in SP, the frequency ranges from 0.31 to 7.5% (median = 2.98%). In the Northeast Region (State of PE), this frequency was 0.03 to 0.06%. TAVARES Jr.[90] detected the presence of NCC in 12.2% of the patients in a psychiatric hospital in MG. The frequency observed at four general hospitals in SP, two in the capital city[55, 57] and two in the interior of the state[4, 89], was 0.08 to 0.19% and 0.30 to 2.5%, respectively. In seroepidemiologic studies, the incidence of cysticercosis including NCC was 0.68 to 5.2%[10, 96, 97]. In one of these studies, VAZ et al.[96] detected a frequency of cysticercosis of 0.82% among children, of 2.3% among adults, and of 5% among patients in a psychiatric hospital.

In a previous study by the author[4], it was observed that, up to the 1980 decade, a diagnosis during the life of the patient was made in 3.6% of patients with NCC through CSF

examination. Within 10 years, with the advent of computerized tomography, the percentage of diagnosis during the patient's life increased to about 50 to 63%[4, 5].

Thirty to 63% of the patients are from the rural zone[4, 19], but urban **origin** becomes more frequent (53 to 62%) when the selection factor was the severe form of NCC[4, 34, 39, 60], and in pediatric studies[57, 94]. **Skin color** does not seem to be a selection factor since its frequency was proportional to the population studied[4]. In most studies, the most involved **age** range (64 to 100%, median = 80%) was that from 11 to 60 years[4, 7, 19, 27, 31, 34, 43, 46, 55, 66, 81-83], with a frequency of 22 to 67% (median = 54%) between 21 and 40 years[2, 4, 7, 19, 27, 31, 34, 46, 55, 66, 81, 82]. In general, NCC was more frequent (51 to 80%, median = 55%) among males[2, 4, 7, 19, 24, 27, 32, 43, 46, 47, 55, 66, 81]. A predominance of females, however, was observed in some cases[89, 99] (53 to 75%), especially among children[57] or when the manifestations were more severe[4, 31, 34, 39, 60].

The most common **symptoms** reported by the patients were headache, convulsions, vomiting and behavioral alterations. In 25 to 52% (median = 40%) of patients[4, 24, 57, 89] neurologic examination was normal. The severe forms of NCC manifested through multiple symptoms, with a predominance of complaints of headache, vomiting, convulsions, dizziness and clouded vision, as well as changes in behavior, nausea and weakness of the legs, always with an altered neurologic examination[4, 18].

Among patients admitted to Neurology wards with a diagnostic suspicion of NCC there was an almost absolute predominance of **epileptic syndrome** (22 to 92%, median = 59%)[4, 16-19, 24, 27, 44, 57, 58, 66, 81, 89, 99], which was more common in outpatient series, and of **intracranial hypertension** (30 to 89%, median = 36%)[2, 4, 19, 24, 31, 34, 57, 81, 87, 88, 99], which was more common in hospitalyzed patient series. The picture of intracranial hypertension was more common among children[17, 57], although it frequently manifests also among adults[4, 24, 31, 34, 81]. **Psychiatric manifestations** occurred in association with neurologic manifestations in 9 to 23% (median = 16%) of the patients with NCC[18, 19, 24, 58].

The **period of hospitalization** for patients with NCC ranged from 1 to 254 days (median = 9 to 28 days)[4, 55], with approximately 30 to 35% of patients being hospitalized for up to one week. The shortest periods of hospitalization usually involved female patients who die and whose basic cause of death was NCC. About 33 to 50% (median = 47%)[4, 24, 55] of the patients need 1.7 ± 1.4 readmissions to the hospital, and some of them need as many as 9 readmissions[4].

Lethality caused by NCC was 0.29%[4] compared to all diseases in general, with a predominance of males in the 31 to 60 year age range. According to some investigators[4, 19, 55, 81, 89, 99], the lethality rate of NCC at Neurosurgery and Neurology services ranged from 4.8 to 25.9% (median = 14.7%). In a neurosurgical study[28] this rate was 0 to 60%. Compared to lethality due to neurologic pathologies, the lethality rate of NCC was 0.58 to 3.6% (median = 2.2%)[4, 81, 99]. Autopsy studies have shown that NCC was considered to be the basic cause of death in 16 to 25% (median = 18%) of cases[4, 32, 45]. In addition, when the terminal causes of death of these patients were analyzed it was observed that the pathologic state which directly contributed to death due to cerebral edema and hypertensive hydrocephalus was intracranial hypertension in 47% of cases[4].

In approximately 67% of cases[4, 32, 46], the most frequent **localization** of the parasites was in the cerebral hemispheres, with the parietal and frontal lobes being most often involved, predominantly on the right (59%)[4]. The cyst was single in 45 to 55% of cases[4] and multiple (2 or more) in 30 to 67% (median = 46%) of cases[4, 32, 46]. The single racemose form was detected in 6 to 25% (median = 17%) of cases[4, 32, 46], the association with *C. cellulosae* was detected in 7 to 22% of cases[4] and the presence of *C. cellulosae* only, in 53 to 76% of cases[4]. The cysts were viable in 26 to 67%[4, 12]. The **size** of the cysts detected in pathology studies[4, 69] ranged from 2 to 20 mm (median = 5 to 10 mm) for *C. cellulosae* and from 10 to 75 mm (median = 40 mm) for *C. racemosus*[4, 31].

Involvement of **other organs**, simultaneous or not, ranged from 0.4 to 27%[4, 32, 46, 98], depending on the analysed study. Ocular cysticercosis has been seldom reported in the Brazilian literature[43, 55, 98]. Among the patients with a surgical diagnosis of cutaneous cysticercosis, approximately 70% were males, with a predominance of the 21 to 60 year age range and showing associaton with neurologic manifestations in 65% of them[4].

Asymptomatic cases, i.e., cases with no neurologic manifestations, make up 6% of all clinical cases[89] and 48.5% of all autopsies[4]. When neurologic manifestations were present and NCC was only a secondary diagnosis, approximately 26% of these autopsies correspond to the asymptomatic form[4].

Autopsy studies[4] have shown that, among infecto-parasitic diseases, pulmonary tuberculosis may be associated with NCC in 1.7% of cases, with paracoccidioidomycosis in 0.5% and with Chagas' disease also in 0.5% of cases.

COMMENTS

The diagnosis of NCC, regardless of the clinical manifestations, may be presumptive or confirmatory. The diagnostic hypothesis is obtained by clinical history, epidemiology, and complementary tests, parameters utilized at outpatient clinics and on hospital wards. It means that clinical-laboratory evaluation depends on the subjectivity of the physician and on technology, but permits the manipulation of information with the objective of reducing morbidity and mortality by the use of therapeutic conducts and especially of prophylaxis.

On the other hand, diagnostic confirmation is obtained from the anatomopathologic examination of biopsies and autopsies. In other words, the anatomopathologic study permits a definite diagnosis, the definition of the severity of a disease and of its natural course, and therefore becomes an important element for clinical and laboratory studies.

In view of the wide discrepancies in terms of laboratory resources and the need to diagnose a picture of NCC, knowledge of the epidemiology and natural evolution of the disease is often the only element available to most neurologists. In addition, NCC should be considered at least as a differential diagnosis in endemic areas.

Autopsy represents an important means of confirming the clinical diagnosis, in addition to showing in an objective manner the natural course of a disease. Even so, autopsies tend to be performed less frequently both in Brazil and in other parts of the world[5] due to various reasons such as excessive trust in laboratory diagnoses, difficulty in obtaining authorization by the family, the lack of systematic requirement of obligatory autopsy especially at university hospitals[5], and the high frequency of deaths at home. In this same study[5], the authors noted that NCC occupied 19th position among the pathologies diagnosed at autopsy in a general hospital in the capital city of the State of SP.

Another factor to be considered is that the doctor is socially acquainted with patients living in the urban zone and frequently considers autopsy to be unnecessary, also in view of the difficulty in performing the procedure in many places, thus permitting many cases of disease to go unnoticed.

The difference in prevalence seems to be due to the lack of a uniform routine of organ examination at Autopsy services. Indeed, in autopsy series the reported number of parasites never corresponds to the real number present because the thickness of the sections is usually more than 1cm.

The analyzed series reflect the frequency of the disease in selected samples seen at Neurology and Neurosurgery services and rarely at general hospitals. The approach to these series is diverse and the method used is not uniform. The Brazilian studies suggest that the frequency of NCC with

respect to sex, age range and patient origin varies according to the severity of clinical manifestations[4, 31, 34, 39, 57].

The higher frequency of intracranial hypertension among children and women with the severe form of NCC does not seem to depend on age range but rather on the severity of the picture which requires hospitalization in a specialized service, representing one of the fundamental elements for the selection of patients to be admitted to the hospital. Factors of a social, cultural or genetic nature and still undefined factors may affect these frequencies.

The high percentage of NCC in psychiatric hospitals[90, 96] is possibly due to the lack of hygiene in the life of these patients and to the coprophagy habit common to various neuropsychiatric pictures.

In a previous study by the author[4] one case is described which did not show neurologic manifestations but had viable intraventricular parasites which were only detected at autopsy. This observation suggests that ventricular cysticercosis may exist in the asymptomatic form. The presence of the racemose form of cysticercosis was detected in another patient of the same study, also without clinical neurologic manifestations, indicating that the racemose form can also evolve in an asymptomatic manner. In other terms, NCC may have a natural evolution without symptoms, regardless of the form of presentation of the cysticercus, its location or its stage of development and its quantity in the brain.

Some data suggest a tendency to earlier manifestations when the nervous system is involved, since in patients with cutaneous cysticercosis only, the age range involved was 41 to 60 years. When NCC is analyzed from the viewpoint of presentation of symptoms, it can be seen that, after 40 years of age, it will probably cause more benign manifestations[4].

QUEIROZ & MARTINEZ[69], in a necroscopic study carried out in a region not considered endemic, observed that 67% of the parasites examined were viable and that cerebral cysticercosis was only an autopsy finding. In a study carried out in an endemic region, although using a different methodology, only 26.5% of the patients were found to have viable cysticerci[4]. The literature provides no information to explain this possible discrepancy. Perhaps genetic and regional factors act on the host, on the parasite and on their relation.

The encouragement of surveys in Legal Medicine institutes, as well as population surveys may provide a more real evaluation of the incidence of this neuroparasitosis in the general population, also considering that in necroscopic studies not only all brains are analyzed, but also the ocular globes, skeletal muscles and spinal cord with the nerve roots.

Since egg resistance in the external medium is high and since there are indications that humidity is one of the most important factors in the regulation of *T. solium* egg viability[53], sanitary hygiene acquires fundamental importance in the control of the taeniasis/cysticercosis complex. For this reason, while general prophylaxis continues to be an utopic goal, individual preventive measures should be instituted.

The population in which cysticercosis is endemic could be instructed to store pork meat in small pieces for 1 to 4 days at temperatures of -20°C to 5°C[79], or, if the portion is 3 to 4 cm thick, to cook it for 1-2 hours or freeze it at -20°C for 3 1/2 to 4 hours[38]. The cysticerci are resistant to detergent and to oxidants. However, exposure of the food to 5 M NaCl, concentrated detergent or 6% $NaClO_4$ for more than 4 hours seems to be sufficient for sterilization[38].

Support from government organs showing greater interest in, and concern for, the health of the population by investing in Public Health Programs would certainly be the best measure for the treatment of NCC. At the basis of these suggestions are the serious consequences caused by NCC to a potentially productive population.

An attempt to start the eradication of the taeniasis/cysticercosis would be to require compulsory notification of the disease. Basic notions about the biological cycle of the taeniasis/cysticercosis complex and of endemic factors taught in schools or even presented as subliminal advertising, may also represent another long-term attempt at the eradication of the taeniasis/cysticercosis complex.

However, as long as poor disposal of the feces and lack of hygiene in swine rearing exist, together with the lack of inspection of slaughterhouses and butcher shops, deficient basic sanitation and insufficient sanitary education, cysticercosis will continue to be endemic.

RESUMO

Epidemiologia da neurocisticercose no Brasil.

Realizou-se revisão da literatura com o objetivo de tentar delinear um perfil epidemiológico da neurocisticercose no Brasil. A prevalência em **necrópsias** variou de 0,12-9%. A freqüência, nas casuísticas **clínicas** foi de 0,03-7,5% e, nos estudos **soroepidemiológicos**, de 0,68-5,2%. Compreendeu 0,08-2,5% das internações em hospitais **gerais**. A **procedência** foi rural em 30-63% dos doentes. Comprometeu mais (64-100%) na **faixa etária** dos 11 aos 60 anos, predominantemente (22-67%) entre 21 e 40 anos. O **sexo** masculino foi mais atingido (51-80%). Nas formas graves, houve predomínio da origem urbana (53-62%) e do

sexo feminino (53-75%). O **período de internação** variou de 1-254 dias, com 33 a 50% dos doentes necessitando 1.7 ± 1.4 admissões. Houve variabilidade no **quadro clínico**, predominando síndrome epiléptica (22-92%) e hipertensão intracraniana (19-89%). A presença de manifestações psiquiátricas foi observada em 9-23% dos doentes. A **letalidade**, frente as doenças em geral, foi de 0,29% e, entre as doenças neurológicas, de 4,8-25,9%. A forma **assintomática** foi detectada em 6% dos doentes de casuística clínica e em 48,5% dos casos de necrópsia. A forma racemosa e a localização ventricular também se apresentaram de maneira assintomática. Entre os doentes com cisticercose cutânea, 65% apresentavam manifestações neurológicas.

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