Intraventricular Neurocysticercosis: Comparative Analysis of Different Localizations. Clinical Course and Treatment. A Systematic Review

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Abstract

Background

Neurocysticercosis is significant due to its high prevalence and considerable morbidity and mortality. The intraventricular form of NCC is less common than parenchymal, may have a rapidly progressive course and it requires a corresponding therapeutic response. Despite the extensive literature dealing with NCC and intraventricular cystic lesions, no systematic reviews have addressed similar work related to the clinical course and treatment of the infestation. Our main objective was to analyze the clinical type of the disease and the management for each ventricle separately on the basis of case reports or series of patients with individual data on the course of the disease and its treatment. As a control group, we used data on signs & symptoms and treatment of patients from published series on intraventricular neurocysticercosis.

Method

We performed a search in the Medline database. Google Scholar was also randomly searched. We extracted the following data from the eligible case/series: age and gender, symptoms, clinical signs, diagnostic examinations and findings, localization, treatment, follow-up period, outcome, and publication year. All data are presented in the form of absolute and relative numbers. The frequency of signs and symptoms, treatment and outcomes of the observed groups were checked by the Chi-square test and Fisher's test. The hypothesis was tested with p <0.05 as statistical significance.

Results

158 cases of isolated and mixed forms of neurocysticercosis were divided into five categories. Hydrocephalus was recognized in 133 (84.2%). Patients with isolated IVNCC were younger (P=.0264) and harbored a higher percentage of vesicular cysts (p <.00001). In mixed IVNCC, the ratio was in favor of degenerative and/or multiple confluent cysts (p = 0.00068). Patients harboring fourth + third ventricle cyst (potentially obstructive form) are younger than lateral ventricle individuals (possibly less obstructive form) (p = .0083). Most patients had individual symptoms for a long period before the acute onset of the disease (p <.00001). The dominant clinical manifestations are headache 88.5%, vomiting and nausea (56.1%), altered state of consciousness (44.6%), and focal neurological deficit (37.2%). The altered level of consciousness and the focal neurologic deficit was the only clinical manifestations with statistical significance (p= .0010 and p=0.398 respectively). Endoscopy (49.1%) was an elective surgical procedure with statistical significance within the study groups (p <0.001). Forty-six (29.2%) subjects underwent standard microsurgery, the fourth ventricle was the dominant site of intervention (p <0.001).

Postoperatively, 39 (24.5%) received anti-helminthic drugs in combination with/without anti-inflammatory medication and other drugs. Endoscopy, open surgery, and postoperative antiparasitic therapy showed statistical differences (p <0.001). Favorable outcomes or regression of symptoms were observed in 79.9%; the mortality rate was 6.3%. Regarding the case series, clinical manifestations were as follows headache-64. %, nausea & vomiting 48.4%, focal neurologic deficit 33.6%, and altered level of
consciousness 25%. Open surgery was the dominant form of intervention (craniotomy (57.6% or endoscopy 31.8%); with statistical significance between them(p< .00001).

Conclusion

Ventricular neurocysticercosis is an alarming clinical condition. Hydrocephalus is the dominant diagnostic sign. Isolated IVNCC patients were recognized at a younger age than Mix.IVNCC individuals; with cysts in the fourth and third ventricles (as a potentially more occlusive type of disease), presented their symptoms at a younger age than individuals with LVNCC. The parasites in its vesicular stage are located predominantly in isolated IVNCC, while degenerative and multiple confluent cysts are the main feature of Mix.IVNCC. The majority of patients had long-term signs and symptoms before the acute onset of the disease. Headache, nausea & vomiting are the most common symptoms of infestation accompanied by altered sensorium and focal neurological deficits. Surgery is the best treatment option. A sudden increase in ICP due to cerebrospinal fluid obstruction with a successive cerebral hernia is the leading cause of fatal outcomes.

Introduction

Neurocysticercosis (NCC) is significant due to its high prevalence and considerable morbidity and mortality[21] The intraventricular form of NCC (IVNCC) is less common than parenchymal disease ranging from 3.11% to 45% [14, 25, 26, 27, ], and may have a rapidly progressive course [47]).

Despite the extensive literature dealing with NCC and intraventricular cystic lesions, no systematic reviews have addressed similar work related to the clinical course and treatment of the infestation. We did not find a prospective, randomized, double-blind study that supports proper clinical-surgical decisions in daily medical practice. Heterogeneous signs and symptoms were reported in available IVNCC or extra parenchymal neurocysticercosis publications [(4, 7, 10, 23, 38, 41, 44, 48,)]. Ventricular NCC might be potentially severe; it requires a corresponding therapeutic response. A few clinicians presented their results with medical therapy, most of them as case reports [(19, 22, 24, 41)]. Others have published their surgical experiences applying the same treatment to all patients or did not specify which type of surgery was used [4, 14, 23, 25, 26, 41].

Our idea was to analyze the clinical type of the disease and the management for each ventricle separately based on case reports or series of patients with individual data on the course of the disease and its treatment. As a control group, we used data on signs & symptoms and treatment of patients from published series on intraventricular neurocysticercosis.

Lifecycle of the parasite

Taenia (T.) solium has an indirect life cycle that requires two different hosts for species to survive in the wild. It includes man as the final host where the tapeworm parasitizes in its adult form, and pigs and human beings as an intermediate host in which the development of the larva in the body occurs. People
become infected by eating raw and/or dried pork that contains cysts, a larval form. After swallowing the larva-form (Cysticercus), the released scolex from the cysticercus, straights, attaches to the jejunum’s mucosa, growth, and develops into a mature adult tapeworm. The adult stage secretes eggs through the genital pores in the proglottids from the intestinal lumen into the environment through feces. Poor sanitation conditions favor pig access to human feces that potentially contain T solium eggs. The embryos, released from eggs, cross intestinal mucosa, enter the bloodstream and disseminate into any organ or tissue where they develop into cysts. The life cycle is completed after a person eats such infected meat [(17]. Infection may occur when a person eats eggs that are usually in improperly cleaned food (vegetables/fruits) or are on contaminated hands and objects (fecal-oral route) or even by regurgitation of eggs [55]. Person-to-person infection is more common than contamination that comes from eating undercooked pork infected with cysts [35,40].

Material And Method

Using the keywords 'Intraventricular neurocysticercosis' 'Brain ventricle cyst' Cysticercus cyst in brain ventricles' 'Intraventricular cystic brain lesion' 'Intraventricular cysticercosis' 'Neuroendoscopy, we conducted a search of the English literature in the Medline database without restrictions on the date ranges of published articles. Google Scholar was also randomly searched.

Data collection and analysis

The document was read and reviewed by two investigators. In the first step, titles and abstracts were screened by one reviewer (SM) and by the second reviewer if the first one was uncertain about the eligibility of the case/series (ZM).

Joanna Briggs Institute Critical Appraisal Tool: Checklist for Case Reports (REF) [28] assessed case reports and case series for methodological quality. The aim of the quality assessment tool was not to eliminate articles with poor methodological quality. The objective was to stress the potential bias of selected cases/series. The searches performed are presented in Table 1 and the Supplementary tables containing the papers of the reported cases. We excluded cases-series without an individual case description from the examination (Figure 1). As a control group, we compared our findings with a case series of patients with IVNCC regardless of topics (neurocysticercosis, extraventricular neurocysticercosis, or intraventricular cysticercosis) (Fig 2).

All data are presented in the form of absolute and relative numbers. The frequency of signs and symptoms, treatment and outcomes between the examined groups were checked by the Chi-square test and the Fisher test. The hypothesis was assessed with a p<0,05. as statistical significance. Statistical data processing was performed in Open Epi Info Version 7.2.2.6 (CDC, US).

Isolated ventricular cases were divided into four categories: fourth ventricular neurocysticercosis (FV-NCC), lateral ventricular neurocysticercosis (LV-NCC), third-ventricle neurocysticercosis (TV −NCC), and migratory ventricular cysts (Mig. -IVNCC); the last group consisted of individuals with mixed
intraventricular neurocysticercosis (Mix. IVNCC), involving the brain parenchyma, subarachnoid space, and ventricles. The following data were extracted from the eligible case/series: age and gender, symptoms, clinical signs, diagnostic examinations and findings, localization, treatment (with histological confirmation of cysticercosis in the operative group of patients), follow-up period, outcome, and publication year. The lifetime of the patients was classified into six groups: under 16 years old, between 17-30 years old, 31-40 years old, 41-50 years old, 51-60 years old, and above 60 years old.

The diagnosis of NCC was established by a combination of Computed Tomography (CT) Scan and/or Magnetic resonance imaging (MRI) (except in one case) with new acquisition techniques (CISS, FIAR, SWAN, ESTA), serological test, and in most cases by histologic confirmation of cysticerci. The imaging findings of NCC cysts were defined according to the neuroimaging definition [11]. We also adopted a set of revised criteria and degrees of diagnostic certainty to confirm the definite and probable diagnosis of IVNCC[18].

**Results**

Out of 121 articles that dealt with various problems related to intraventricular cysticercosis, we singled out 158 cases that met the inclusion criteria. The most common localization of the cysticerci was FV-NCC-44 (28.3%), followed by Mix. IVNCC-35 (22%) and LV-NCC-35 (22%).

Immunodiagnostic tests were performed in 44(27.8%) individuals; only thirty persons (19.%) had the enzyme-linked immunosorbent assay (ELISA) and enzyme-linked immune electrotransfer blot test (EITB) in serum and cerebrospinal fluid (CSF) positive.

The gender ratio was practically equal: 78(49.7%) males and 79(50.3%) females (one data was missing); noticeable in favor of women between isolated IVNCC vs Mix.IVNCC(65:14) but without statistical significance (p = .1660) (Table 2a). Age ranged from 6 to 77 (30.42 ±2.16); the average age ratio between isolated IVNCC and Mix IVNCC was 28.73 ±2.28 (median 28) vs 36.83 ± 4.99 (median 35). (Fig 3). In Mix. IVNCC the predominant cases fell between 31 and 40 years old (25.75%); in the isolated form of IVNCC, the most common prevalence was identified in the group of 21 to 30 years old (46.7%), with statistical significance within the group (P=.02646). Similarly, patients who had cysts in the fourth and third ventricles (potentially obstructive form) were younger than persons with LV NCC (possibly less obstructive form) (median 45:14; p = .0083) (Table 2b and Fig 3 a, b).

Of the 35 patients with Mix.IVNCC, 22 (62.8%) possessed nodular NCC or multiple parenchymal calcifications; 11 (31.4%) had only subarachnoid cysts situated in basal cisterns of the brain, a Sylvian fissure, an interhemispheric region, or a subarachnoid space of the spinal cord; in 3 (8.6%) cases there was a combination of parenchymal and subarachnoid lesions (p <0.001). Intraventricular distribution was as follows: FV-NCC-8 (22.8%), LV-NCC-10 (28.6%); multiple IVNCC-7 (20.%), Mig. IVNCC-7 (20%) and TV-NCC-3 (8.6%).
Hydrocephalus was recognized in 133 (84.2%) patients with a prevalence in Mix. IVNCC (94.3%), FV-NCC (88.4%), less in LV-NCC 72.3%, but without statistical significance (p= .137949) (Table 2a). Eleven patients (8.3%) had unilateral ventricular dilatation as a result of Monroe's foramen occlusion.

In relation to the histopathological/radiological stage of neurocysticercosis, a higher percentage of vesicular cysts was observed in isolated IVNCC 70 (76.1%) versus 22 (23.9%) mixed forms of IVNCC; the proportion was the opposite: Mx. IVNCC 20 (80%) vs. 5 (20%) in favor of degenerative and multiple confluent cysts (p <0.001). No relevant data for 50 cases.

Symptoms in most patients lasted from several years to more than seven days 73(71.6%), and only 28(27.7%) had an acute onset at admission (p < .00001). Data were missing for 59 people.

The prevailing clinical manifestation was headache (88%), followed by vomiting and nausea (60%), altered sensorium (44.%), and focal neurological deficit (35.3%) (Table 3a). The altered level of consciousness (ranging from 20.9% to 76.2%) and focal neurologic deficit (ranging from 51.2% to 14.3%) were the only clinical manifestations with statistical significance (p= .0010 and p=0398 ).

No symptomatology data were available in 9(5.7%) patients; two individuals had asymptomatic cysts. Four cases had signs and symptoms of elevated intracranial pressure (Table 3a, b). Endoscopy was the elective surgical procedure in 78(49.4%) patients, and the proportion was significantly different among the examining group (p<0.001) (Table 3a). No statistical significance between isolated IVNCC vs Mix. IVNCC ( p=.55509).Four patients experienced partial resection of the cyst; the rest had complete excision of the parasite( 97.5%).( Table 3a,b).

Forty-two(26.7%) and probably another four patients(2.5%) underwent microsurgical intervention. Three had a combination of craniotomy and endoscopy. The fourth ventricle was the dominant site of open surgery 21(47.7%) with statistical significance in comparison with other reviewed groups (p<0.001).No statistical difference between isolated IVNCC and Mix.IVNCC (p=.753548) (Table 4).

All surgically treated patients experienced favorable outcomes

Twenty-one (14%) patients underwent cerebrospinal fluid diversion such as , ventriculoperitoneal( VP),or ventriculoatrial(/VA)- shunt, endoscopic third ventriculostomy(ETV ),or extraventricular drainage(EVD); the percentage within groups ranged between 4.5% -25.7% with statistical significance(.019365 ).Fifteen of them received anti-helminthic and steroids,2 only steroids, and four died not receiving any other therapy(Table4).

As the only treatment, anti-helminthics were used in only 7 (4.4%) patients.

Postoperatively, 39 (24.5%) subjects received either antiparasite drugs alone or in combination with steroids and antiepileptics. Statistical differences were observed among endoscopy, open surgery, and postoperative anti-helminthics (p <0.001)
Fortuitously found cystic lesions encountered in two (1.2%) individuals. Lack of information about therapy in five patients (Table 4).

All treated cases met favorable outcomes or regression of symptomatology; **not a report on the failure of the VP shunt (Table 5).**

Twelve persons (7.6%) had a fatal outcome. Eight patients died due to obstruction of cerebrospinal fluid flow and consequent cerebral hernia and brain stem compression; two cases suffered a severe chest infection and postoperative cardiac arrest. One patient, a 77-year-old man, had a decrease in the "immune response and resistance to applicable therapy." The last patient to experience a rupture of a giant basilar aneurysm harbored an asymptomatic cyst confirmed at autopsy. The follow-up (FU) was recorded in 65 (41.1%) patients, ranging from 17 days to 5 years (average days 360 ± 243.54); no mention of FU in 93 (58.9%) subjects (Table 5).

The published case-control series on IVNCC had 16 publications with 286 individuals with the clinical course and treatment method. Clinical manifestations were heterogeneous: headache - 64.4%, nausea and vomiting 48.4%, focal neurological deficit 33.6%, and altered level of consciousness 25%. Hydrocephalus was present in 68.3%. In several cases, symptomatology was described as a syndrome of increased intracranial pressure (1.6%). The findings were statistically significant with Yates correction only in the frequency of headaches and altered level of consciousness (p < 0.00001 and p = 0.00 respectively) as well as hydrocephalus (p < 0.00001) (Table 6).

Surgical excision of cysts was the dominant form of treatment (craniotomy (57.6%) or endoscopy (31.8%); a smaller number of patients had different types of CSF diversion (17.8%), or only medical treatment (2.8%). The ratio of craniotomy and endoscopy was statistically significant (p < .00001). In most individuals, the outcome was satisfactory (Table 5). The statistical significance was affirmative in relation to endoscopy and the craniotomy between "our clinical materials" and patients with case series (P-value = .000266 and P-value = .00001). This was not the case with CSF diversion (P-value = .2139) or with only drug therapy (P-value = .3768). The majority of patients had involvement of the fourth ventricle with NCC (44.7%), and a smaller number of individuals had parasites in the lateral ventricle, the third ventricle, and mixed IVNCC in total (30.4%); for fifty patients there were no precise descriptions of localization (Table 7).

**Discussion**

In "our study group" the gender ratio is slightly in favor of women, and the finding does not differ much from other available clinical materials [26., 38., 23] . Patients with isolated IVNCC are younger than those with Mic.IVNCC. Symptomatology of patients with cysts in the fourth and third ventricles occurs at a younger age than those with LVNCC. It is postulated that the parasites in the subarachnoid compartment have more room to increase their dimensions before they come into contact with the cerebral tissue [38]. A similar explanation can be for cysts in the lateral ventricles.
Intracranial hypertension and focal neurological deficit were the dominant clinical features in the present collection of patients. A focal neurological deficit occurs due to compression of local brain tissue (corticospinal tract, visual pathways, periaqueductal gray matter, fourth ventricle) by the parasite [16]; while direct pressure on the brain cysticerci on cerebellar structures due to the increased dimension of the fourth ventricle can cause neurological loss, gait ataxia, dysmetria, and diplopia [13, 15] On the other hand, migratory cysticerci due to their small size move freely through the ventricular system and do not exert local pressure on the tissue.

In the extensive literature on neurocysticercosis, a limited number of clinicians have reported the clinical features of IVN CC [1, 4, 6, 8, 12, 23, 30, 34, 36, 37, 39, 42, 44, 45, 49, 50]. Predominant symptoms were headache (acute, intermittent, chronic, severe, throbbing, etc.) and nausea/vomiting accompanied by focal neurological deficit and altered level of consciousness. Additional manifestations varied considerably depending on the clinical material, such as visual impairment, papilledema, seizures, visual disturbances, psychiatric syndrome, or chronically elevated intracranial pressure.

Symptoms lasted from several years to more than seven days; a small percentage had an acute onset of the disease. The duration of clinical signs before the diagnosis can vary significantly, depending on the cyst's location or its evolution. A growing cyst that interferes with CSF flow manifests its symptomatology more quickly; inflammation is a slow-developing process, and signs and symptoms can appear several years after infection [2]

A higher percentage of vesicular cysts was observed in isolated IVNCC than in the mixed form of NCC; the proportion of degenerative and multiple confluent cysts was more common in Mix.IVNCC than in the isolated form of IVNCC. By invading the ventricle, viable parasites initiate evasion of the immune response through negligible inflammation, allowing them to persist in the host for long periods, sometimes years [54]. Active, floating cysts are asymptomatic and can freely migrate within the ventricular system. Clinical manifestations can occur if the cyst blocks the ventricular openings, causing disruption of cerebrospinal fluid flow with consequent acute hydrocephalus [5, 24]. Gravity and changes in CSF pressure are provoking mechanisms of cyst migration. In the degenerative stage, the cyst releases antigenic substances that cause an inflammatory reaction; the capsule becomes attached to the ventricular wall with fibrous adhesions, which may give rise to ependymitis, ventriculitis, scars, and obstruction of CSF flow. The outcome may be lethal [43]. If inflammation involves meninges, the symptoms of meningeal irritation become manifest (fever, change of consciousness, nuchal rigidity [29]. Post-inflammatory sequelae cause mental deterioration, blindness, quadriplegia, and ataxia [22, 24, 29].

Surgery is the best treatment option for IVNCC. The main goal is to eliminate the symptoms of elevated ICP and to resect the cyst in the same setting. Sudden obstruction of the cerebrospinal fluid flow requires immediate operative intervention. In patients with a cyst in the involutional or granular phase, surgical resection of the cyst may be difficult, can initiate damage to the surrounding structures, and cause ventricular bleeding. If the lesion is not amenable to complete excision, the primary goal is to treat hydrocephalus with or without partial resection of the cyst [14, 26, 30, 52]. Partial cyst removal does not
appear to cause recurrence [23,26, 47]. Cyst rupture during surgery has been reported harmless and without complications [26, 30]. Cyst removal sometimes requires a permanent shunt due to chronic inflammation caused by cysticerci [9 ]. Unfortunately, VP shunt failure has proven to be a frequent complication with its tendency to infection, occlusion of the system (by cyst-associated gelatinous material or high CSF protein), and cyst migration [3, 14, 32]. External ventricular drainage is still warranted in cases with severe infection and ependymitis [27]. Surgical removal of a single cyst is considered the end of treatment. Any suspicion of the existence of multiple cysts requires antihelminthic therapy [53]. It is not recommended to install antiparasitic drugs before surgery because they lead to parasite disruption, which may be accompanied by an inflammatory response of the adjacent tissue and compromise of surgical removal of the cyst [52]). This therapy is also not recommended in patients with increased ICP [51].

The endoscopic transventricular-transforaminal approach seems appropriate to solve problems for cysts in the lateral and third ventricles. Endoscopic management of the fourth ventricle may be challenging. However, endoscopy is not without risks. Intraventricular bleeding, memory loss, hemiparesis, mutism, ventricular entrapment, and aphasia have been reported [39, 41]. The common approaches in open surgery are transcortical (lateral ventricular cyst); transcallosal or transcortical (lateral or third ventricle) and suboccipital midbrain (fourth ventricle) [4, 31, 33]. Open surgery has drawbacks: craniotomy, blood loss, possibly delayed hydrocephalus, withdrawal of brain invasion with the potential risk of damage to the vital structure, periventricular edema, and ventricular entrapment [23,26, 46]. Controversy exists between ‘our clinical material’ and the collective case-series regarding the type of surgical treatment. While the percentage of endoscopically treated patients in ‘our series’ is higher than patients with open surgery, in the case-series patient the situation is the opposite. The explanation may lie in the fact that the number of NCC cases involving LV, TV, and mixed forms of IVNCC were more numerous than the number of patients with parasites in the fourth ventricles; in the case series, the sum of individuals with the fourth ventricle’s infestation was higher than in the other three compartments. According to Clinical Practice Guidelines, neuroendoscopy is recommended as the first option in the treatment of cysts in LV-NCC or TV-NCC, and surgical resection of fourth ventricular cysts by suboccipital approach [52].

Hydrocephalus, a common clinical sign, was treated surgically in most cases with endoscopy, ETV, EVD, and VP/VA shunt [10,14, 23, 26]. Unilateral hydrocephalus occurs as a result of unilateral obstruction of the openings of Monro; clinical manifestations are usually slowly progressive and less dramatic [20]. Anti-helminthic treatment, as the only therapy, was applied to seven individuals(4.4%) with favorable outcomes. Several authors witnessed satisfying results with the absence of cysts on imaging outcomes after the single use of the medication [19,24].

The limitation of the study is the modest number of IVNCCs, because the survey includes only case reports and case series with individual reports published in English. In general, publication bias is a limitation of systematic reviews, especially for case/case reports, as each has its subject of interest that
might neglect another area of research. Searching a single database, such as Medline, brings about the possibility that the analysis does not cover all individual cases. Language bias is another limitation since a number of IVNCC patients were published in other languages.

In conclusion: Ventricular neurocysticercosis is an alarming clinical condition. Hydrocephalus is the dominant diagnostic sign. Isolated IVNCC patients were recognized at a younger age than Mix.IVNCC individuals; with cysts in the fourth and third ventricles (as a potentially more occlusive type of disease), presented their symptoms at a younger age than individuals with LVNCC. The parasites in its vesicular stage are located predominantly in isolated IVNCC, while degenerative and multiple confluent cysts are the main feature of Mix.IVNCC. Most patients had long-term signs and symptoms before the acute onset of the disease. Headache, nausea & vomiting are the most common symptoms of infestation accompanied by altered sensorium and focal neurological deficits. Surgery is the best treatment option. A sudden increase in ICP due to cerebrospinal fluid obstruction with a successive cerebral hernia is the leading cause of fatal outcomes.

Declarations

Conflict of Interest

All authors declare no conflict of interest

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Ethic declaration

The authors declare that ethics approval was not required for this case report

Consent to participate

Not applicable

References


Tables

Tables 1 to 7 are available in the Supplementary Files section

Supplementary Tables

The Supplementary Tables are not available with this version

Figures
Figure 1

Flow Diagram of IVNCC

Database search (N= 121 papers )

excluded
clinical manifestation and treatment in case-series on intraventricular neurocysticercosis.
(N=36 papers)

papers with individual description of cases with intraventricular neurocysticercosis (N= 85)

reported intraventricular cases excluded
809 cases

reported patients
N=158
Figure 2

Flow diagram of case-series patients with IVNCC addressing clinical status and treatment
In Mix. IVNCC the predominant cases fell between 31 and 40 years old (25.75%, median 28); in the isolated form of IVNCC, the most common prevalence was identified in the group of 21 to 30 years old (46.7%, median 35), with statistical significance within the group (P=.02646).
Patients who had cysts in the fourth and third ventricles were younger than persons with LV NCC (median 45:14; p = .0083)

**Supplementary Files**

This is a list of supplementary files associated with this preprint. Click to download.

- TABLES.docx