Chapter 7

Subarachnoid Cysticercosis and Ischaemic Stroke in Epileptic Patients

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Additional information is available at the end of the chapter

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Abstract

Whether subarachnoid neurocysticercosis (SNCC) induces ischaemic stroke (IS) in epileptic patients is not yet confirmed because only short-case series and anecdotal case reports have been published, and no observational studies exist in the literature to date. Our main goals are: to estimate the prevalence of ischaemic stroke in epileptic patients presenting with SNCC and stroke frequency among HIV-positive patients in three subgroups; to determine if the odds of ischaemic stroke are elevated in SNCC-epileptic patients compared to epileptic patients with intraparenchymal NCC (INCC); to determine whether the risk for stroke is elevated in HIV-seropositive patients presenting with SNCC or INCC and epilepsy; and to evaluate if and when the potential interaction varies by location of NCC in the brain (intraparenchymal or subarachnoid). Eligible epileptic patients' seropositive status was recorded, and cross-associations for the independent variables (NCC status and HIV status) and outcome variables (ischaemic stroke event) were performed. Compared to the reference group, the odds of IS in PLWNCC were 2.0 and 2.6 times greater in patients with SNCC and INCC, respectively. The frequency of IS was greater in HIV-positive patients in all three groups, but the risk was especially pronounced when seropositive epileptic patients were both NCC groups when compared with the reference group. Subarachnoid NCC increased the risk of IS three time more.

Keywords: neurocysticercosis, stroke, epilepsy, cross-sectional study, subarachnoid neurocysticercosis, HIV, imagenology

1. Introduction

1.1. Background

Neurocysticercosis (NCC) is a preventable and potentially eradicable neurological disease caused by the larva form of tapeworm *Taenia solium* which primarily affects people living in the developing world. Seizures are widely reported to be the most common symptom, occurring in 70–90% of patients [1]. Most patients respond to praziquantel, if cystic lesions are located in the parenchymal tissue, and albendazole when parasites are located in the ventricular system and subarachnoid space, including patients with an associated HIV infection [2]. A well-designed clinical trial about treatment response in subarachnoid neurocysticercosis (SNCC) has not been published; however, some authors have reported the effectiveness of these drugs in SNCC [3–5], while others have found that parasites remain alive at the subarachnoid space even after high dose of albendazole/praziquantel/prednisone was administered [6–10].

In 1977, Gubbay and Matz [11] reported two cases presenting with intracranial hypertension (ICH) and hydrocephalus in association with chronic meningitis; the authors confirmed that repeated CSF analysis may result in diagnostic confirmation of SNCC causing ICH with hemiparesis, partial seizures, and other neurological signs. Currently, it is well known that SNCC in the basal cisterns may cause inflammatory reaction; the leptomeninges become fibrotic at the base of the brain.

According to Takayanagui and Odashima [12], in approximately 60% of the cases, there is an obstruction of the CSF circulation, resulting in hydrocephalus and raised intracranial pressure. When hydrocephalus secondary to cysticercoid meningitis is present, the mortality rate is high (50%) and most patients die within 2 years after CSF shunting; therefore, ventricular and basal cisternal locations are considered to be malignant forms of NCC. In 2001, Bannur and Rajshekhar [13] reported a case as an example of difficulty in confirming diagnosis: this patient had a hypodense non-enhancing mass on CT scan in the regions of quadrigeminal cistern causing obstructive hydrocephalus. He was initially diagnosed with an epidermoid mass but subsequent MRI evaluation and surgery resulted in the diagnosis of a racemose cysticercus cyst. Authors concluded that clinical features of NCC largely depend on the number, type, size, localisation and stage of development of cysticerci, as well as on the host immune response against the parasite [13].

Typical of South Africa, high incidence and prevalence of NCC is found at the former Transkei, currently region C and D of Eastern Cape Province, which is the most disadvantaged region countrywide [14–18].

Many people in the world who suffer a fatal stroke live in developing countries where NCC is endemic. However, prevalence of several tropical diseases, including NCC, is likely to increase in Western, industrialised nations as a result of demographic changes due to migratory flows. It is very well known that stroke is the third most cause of death and the principal cause of adult disability worldwide. Cerebrovascular complications of NCC include transient ischaemic attacks, ischaemic strokes due to infective vasculitis and intraparenchymal haemorrhage [19, 20].

Several case reports about cerebral infarction related to cysticercosis have been published, including angiographic abnormalities [21–27].

In our region, there are other infectious diseases causing infective vasculitis leading to ischaemic stroke such as HIV/AIDS, tuberculosis, INCC and neurosyphilis, but Chagas disease, malaria, haemorrhagic fever, infective endocarditis and mucormycosis are also reported in the medical literature [28, 29].

In 2001, Rocha et al. [30] reported three cases of stroke secondary to NCC. The first one was a 36-year-old man with bilateral middle cerebral artery occlusions presenting an acute right hemiparesis and expressive aphasia. MRI demonstrated several enhancing subarachnoid cysts surrounding the occluded vessels, a right parietal racemose cyst and a left temporal large infarction area. Angiographic study showed total occlusion of left middle cerebral artery and a subtotal occlusion of right middle cerebral artery. The second one was a 42-year-old man with vasculitis of small cortical vessels presenting headache, seizures and focal neurological deficit. CT scan demonstrated several calcifications and a left temporal infarct. Cerebral angiographic study was normal. The third case was a woman, 53 years old, with a past history of six stroke events, behaviour disturbance and seizures. MRI scan demonstrated several cortical and sub-cortical infarcts and cisternal cystic lesions, and angiographic study showed diffuse arteritis of basilar and carotid arterial system. In all three cases, CSF study showed linfomonocitic pleocytosis and positive ELISA for cysticercosis.

Aditya et al. [31] in 2004 reported two autopsied cases of chronic cysticercal basal arachnoiditis causing large arterial territory infarcts and, in the second case, a hypothalamic mass. Both patients were diagnosed and managed, clinically and by neuroimaging, as stroke and neurotuberculosis, respectively. The diagnosis was established only at autopsy, which revealed NCC causing basal arachnoiditis, major vessel vasculitis and infarcts. Histologically, one case showed degenerating racemose cysticercal cyst within the thick basal exudates. In the second case, remnants of the degenerated cysticercal cyst in the form of hooklets and calcareous corpuscles were identified within the giant cell inciting a granulomatous response to form a hypothalamic mass lesion mimicking tuberculoma. They highlighted the importance of considering the non-tuberculous aetiology of chronic basal arachnoiditis like SNCC before initiating therapy, especially in countries where both NCC and tuberculosis are endemic.

Conspicuously absent in the case reports available in the current medical literature are the following research questions: What is the prevalence of SNCC in patients living with neurocysticercosis (PLWNCC)? Is SNCC a risk factor for ischaemic stroke? Does HIV comorbidity increases the stroke frequency in epileptic patients infected with NCC? The main aim of this study is to explore these inquiries and propose new hypotheses for future study.

1.2. Our study

We performed a non-published cross-sectional study of epileptic patients diagnosed with NCC from January 1999 to December 2003 at Umtata General Hospital and from January 2004 to January 2010 at Nelson Mandela Academic Hospital from the rural areas of Mthatha, South Africa. Selected patients for a case–control study under the project: 'Neurocysticercosis' were taken for this research.

All patients were classified into one of the three respective sample groups according to presence and type of NCC or not, collected in groups A, B and C. All patients from Group A met the following selection criteria (inclusion criteria): a positive serology ELISA test for cysticercosis, CT scan of the brain with intravenous contrast enhancement consistent with definitive evidence of cystic lesion (isolate or racemose) in the subarachnoid space without hydrocephalus and suitable to evaluate: (1) focal arachnoiditis when there was contrast enhancement in only one cerebral basal cistern; (2) bilateral cystic lesions with diffuse arachnoiditis, in which contrast enhancement involved several basal cisterns; and (3) ischaemic infarction, in which the number and location of cerebral lesions were analysed and classified as superficial, deep no lacunar (>16 mm), and deep lacunar (<15 mm) at the basal ganglia, without an associated cardiac disease. Demographic and associated stroke were analysed in accordance with the presence of SNCC, and ELISA test for HIV when it was done.

From the large number of epileptic patients with NCC-HIV co-infection in our database, we selected only a few number of epileptic cases for Group B similar to Group A, regarding age and gender to assure a better statistical analysis and under an absolute diagnosis of intraparenchymal NCC (both active and calcified at the same time) with or without ischaemic stroke. The ELISA test for NCC and HIV were both positive. Patients in Group C had no NCC in any presentation and, ELISA test for NCC was negative and HIV test was positive.

Exclusion criteria: All patients with gross modifiable risk factors for stroke such as uncontrolled hypertension and diabetes mellitus, heavy drinkers or smokers, familial hyperlipidaemia, thrombophilia, bleeding disorders and other haematological disorders were excluded. We also excluded patients with heart problems, diagnosis of infective vasculitis apart from those associated with NCC/HIV, suspicion of primary or secondary arterial disease, cognitive or sensory deterioration, patients who have not had check-ups for their NCC/SNCC and stroke for more than 11 months, patients with intraventricular NCC and/or associated hydrocephalus; patients with terminal illnesses, serious psychological illnesses, active addictions to psychoactive substances; patients younger than 13 years old; pregnant patients; patients living with HIV/AIDS in stage IV, patients who have lived more than 6 months outside of our region, the 'first or worst' headache, headaches with increasing frequency or severity, progressive headache, chronic daily headache, headaches always on the same side, headache not responding to treatment, new-onset headaches in patients who have cancer or who were tested positive for HIV infection and new-onset headaches after age 45.

All statistical analyses were conducted using Statistical Package for the Social Sciences (SPSS) version 16.0 for Windows (SPSS Inc., Chicago, Ill). Analyses were performed using an intention to treat bias. A descriptive analysis and an analysis of baseline comparability between the study groups were performed for all study variables. The main variables are INCC, SNCC, IS and headache. All patients were epileptic and HIV reactive. To investigate the potential associations between ischaemic stroke outcomes and the variables of NCC group type and HIV status, prevalence odds ratio and 95% confidence intervals were calculated.

Written consent forms were administered in the first contact with the eligible patients following verbal agreement for participation. For all patients, information on the study's purpose and procedures was provided in addition to ethical considerations, including the participant's

right to intimacy, anonymity, confidentiality, withdrawal and information. Due to the large proportion of low literacy among the patient population, oral consent was observed and confirmed by an impartial witness in many cases where necessary. For patients selected between 1999 and 2002 only oral consent was taken.

All investigators completed CITI training course on the protection of human research. All are sworn to the Hippocratic Oath and committed to respecting the norms of good clinical practice, as well as the requirements of the Helsinki Declaration.

Methods for patient selection and information processing were approved by clinical governance at Umtata General Hospital, and the research protocol was evaluated and approved by Mthatha Umtata General Hospital, University of Transkei and Walter Sisulu University IRB and the respective Ethical Committees (UGH:0001/99, UNITRA:0018/05 and WSU:0068/009).

Out of 459 eligible epileptic patients asked to participate, five initially refused to participate for the baseline evaluation. Four out of the five patients agreed to participate during their follow-up appointment and their data are included here.

The proportion of SNCC without hydrocephalus in PLWNCC (Groups A and B) was 4.77%.

Group A (n = 144) and B (n = 153) showed no remarkable differences between age, gender and HIV status. The control group C (n = 161) consisted of 35.1-year-old-mean age (SD 15.4), 43.5% were males (n = 70) and 56.5% were female (n = 91) and the frequency of HIV positive (stage I–II, 11.2%) almost similar to Groups A and B (**Table 1**).

In total, 243 serial CT scans with at least one scan (range = 1–2) per subject were available in Group A, 201 CT scans in Group B and 162 CT scans in Group C, over the 10-year study period.

Table 2 shows the frequencies of ischaemic stroke events in SNCC and INCC (Groups A and B) and the reference sample (Group C) without considering HIV status.

Table 2 shows the frequency and odds ratio of IS events in each NCC group (as previously discussed) now stratified by HIV seropositive status. HIV-positive patients in Groups A and B had greater odds of IS compared to the HIV-positive patients without NCC co-infection as expected. However, the increased odds were more pronounced in those HIV patients with SNCC (OR: 2.66, 95% CI). The risk of IS in HIV-negative patients followed similar trend with the greatest odds occurring in SNCC group patients comparatively. The risk of developing stroke was 2.82 times more probable in Group A compared with Group B. (**Table 2** and **Figure 1**). This suggests that although co-infection with HIV increases risk of IS, the location of NCC in the brain is a

Groups	Age	Gender (%)		HIV (%)		
	Mean (SD)	Male	Female	+	–	Unknown
A (144)	38.2 (16.9)	52.1	47.9	13.9	31.2	54.9
B (153)	36.9 (15.3)	47.7	52.3	15.7	28.8	55.6
C (161)	35.1 (15.4)	43.5	56.5	11.2	28.0	60.9

Table 1. Patient characteristics by sample group.

Groups	Stroke		Total
	Yes	No	
A	23(15.97%)	121(84.02%)	144
B	14(9.15%)	139(90.84%)	153
C	4(2.48%)	157(97.51%)	161

Table 2. Frequency of stroke by groups.

better predictor of IS risk than comorbidity status. This is also evident in a similar difference in odds ratio for HIV- compared HIV+ were exhibited in Groups A and B. These results also suggest that the interactive effect of co-infection is generalised and do not vary significantly for one type of NCC.

Taking into consideration the HIV status of patients by groups, we found that 40% of patients presented ischaemic stroke (Group A) and the risk to develop an IS among Groups A and B is almost three times more.

After comparing all three groups with similar age, gender and HIV-positive status, the risk of developing an IS increases to more than seven times in patients presenting SNCC over the control group and almost four times in patients presenting intraparenchymal NCC, as shown in **Table 4**.

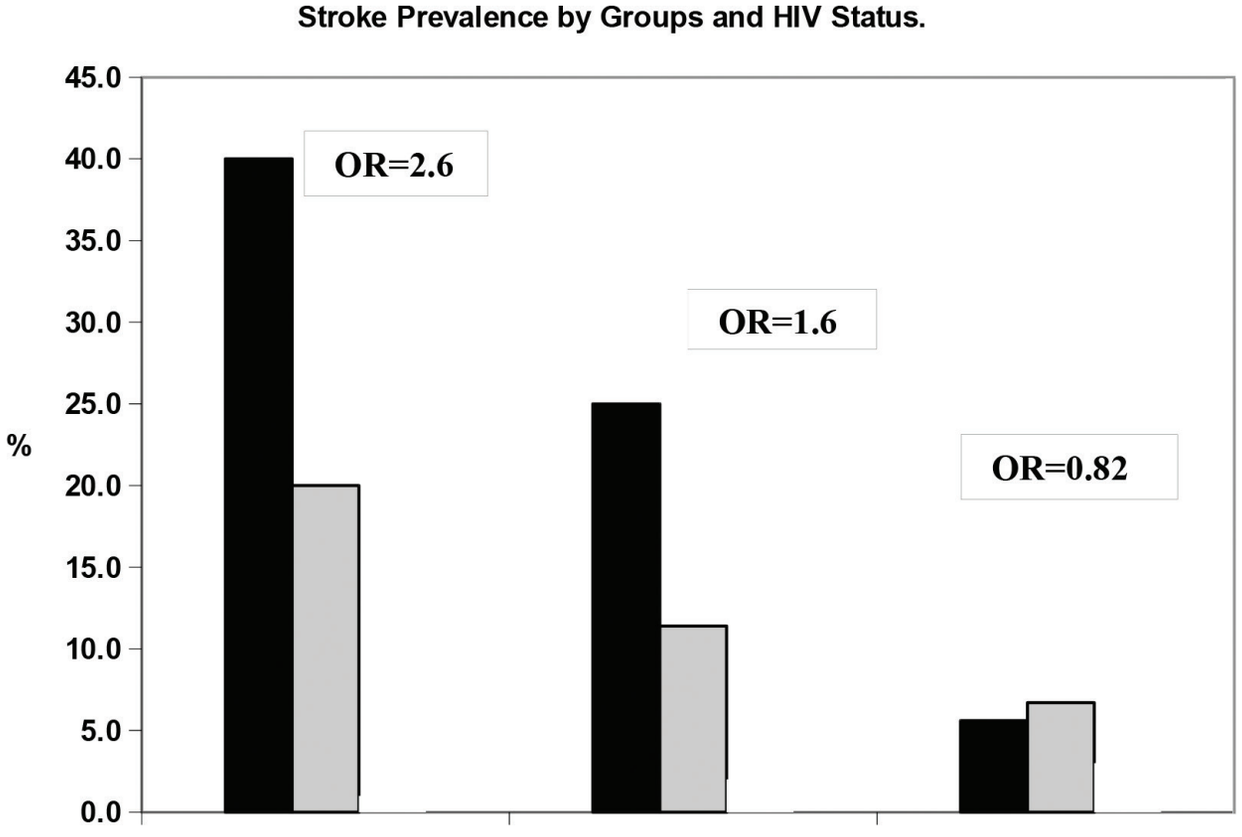

Figure 1. Shows prevalence of stroke by groups and HIV status. *Source*: **Table 3**.

HIV status	Groups	Stroke (%)	OR (95% CI)	A vs. B
+	A	40.0 (n = 20)	2.66 (n = 23)	2.82
	B	25.0 (n = 24)	2.60 (n = 14)	
	C	5.6 (n = 18)	0.82 (n = 4)	
−	A	20.0 (n = 121)		
	B	11.4 (n = 139)		
	C	6.7 (n = 157)		

Table 3. Proportion of patients with IS events by groups and HIV status.

Groups	OR	IC 95%	
A vs. C	7.46	2.51	22.1
A vs. B	1.88	1.33	3.82
B vs. C	3.95	1.27	12.2

Table 4. Odds ratio after comparing different groups.

Concurrent infection with *T. solium* and HIV was expected to occur more frequently because of the increasing frequency of HIV infection in endemic areas of cysticercosis like our region. However, little is known about the influence of HIV infection on the frequency and the clinical course of cysticercosis. Delobel et al. [32] established that giant cysts and racemose forms of neurocysticercosis seem to be more frequent in HIV-infected patients and may be secondary to an uncontrolled parasitic growth because of an impaired cell-mediated immune response.

Prevalence on SNCC in patients LWNCC is not as higher as we expected but can represent the frequency of this problem, while the prevalence of HIV is increasing in countries where NCC is also endemic. Therefore, sooner or later SNCC will be highest, if adequate measures are not taken to eradicate NCC at due time. Other co-infection rates are expected to raise it but unfortunately no systematic reviews of the subject are available in the medical literature.

In spite of the variety of ways used by the parasite to modify host immune response, their mechanism of excretory/secretory product fail and its anti-immune properties are weaker, paradoxically these pathologic changes on the parasite membrane and the surrounding tissue (without remarkable oedema) can be seen in HIV patients with CD4 count around 350 cells/mm^3 and viral load around 55,000 copies/ml or even in window period. Therefore, we have had hypothesised that at the colloid stage, there is an increased microglial activation, associated oligodendrocyte, astroglial changes and subsequent damage of the axonal functions and blood–brain barrier, which can explain the well-known mechanism of *pathological concentration of macrophage histocompatibility complex, interleukin-1 and -6 and tumour necrosis factor-alpha*

among other neurotoxins causing neurovascular lesions, accompanied by increased concentration of pro-inflammatory molecules from meningeal macrophages, choroids plexus macrophages, perivascular macrophages, multinucleated giant cells, according to the number and location of the cysticercus as previously described [33]. *At the present moment, we believe that toxins released by the T. solium cysticercus cause inflammatory changes on the perforating arteries (toxic vasculitis?) at the subarachnoid space rather than a direct effect on the cluster of parasites (mechanical compression). However, the differential of infective vasculitis should be preserved because pathological source is the presence of intracranial cysticercosis.*

In our series, we did not identify any case presenting immune reconstitution inflammatory syndrome, probably because we selected patients from stage I to III of AIDS not on HAART. It is interesting to know that in the brain of patients with ischaemic stroke-associated deaths, there are abundant CD3+, CD8+ and CD68+ cells in the postmortem autopsy [34]. In our study, only one patient died from Group A. We believe that low mortality rate may be related to the exclusion of patients with subarachnoid cysticercosis growing to giant size causing mass effect and obstructive hydrocephalus with mechanical compression, as already discussed [33]. The disease course in SNCC is often long in duration and cysticerci continue to grow and proliferate through tissue. This increase in volume and mechanical resistance from the brain parenchymal may cause osmotic exchange with the CSF and this one factor that may lead to a poor prognosis [35]. Obviously, selecting patients before this stage can help to investigate the effect of the SNCC on the blood vessels without an effect of associated mechanical compression.

Evidence of carotid occlusion in cysticercosis [23, 36, 37], transient ischaemic attack of the vertebrobasilar territory [38] even in children [39] has been reported as anecdotic cases.

The most common affected vascular territory in our series was middle cerebral artery followed by posterior cerebral artery. We did not confirm any patients with SNCC and ischaemic infarct on the anterior cerebral artery territory and only one case has been reported in the medical literature [40].

Haemorrhagic stroke (intracerebral or subarachnoid) associated with NCC and HIV was not selected in our series and some cases reported in the medical literature are not certain [41, 42]. We will investigate the association of haemorrhagic stroke and SNCC in a forthcoming research. Strengths of our study include the large sample size, geographically distinct locations of the participating clinics from the former Transkei in rural South Africa, and potential feasibility of its replication in similar regions worldwide.

Weaknesses of this study include the exclusion of a number of variables that may have contributed to the analysis, such as patient's response to anti-parasitic treatment and the degree of immune compromise. Better reporting of HIV status is also necessary, as over half of the patients selected were HIV-status unknown. In our study, the prevalence of SNCC without hydrocephalus in patients living with NCC is 4.77%.

Risk for ischaemic stroke in patients with subarachnoid NCC is almost three times more for patients with intraparenchymal NCC. Comorbidity of subarachnoid NCC in HIV-positive patients increases up to 7.6-fold.

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2. Racemose neurocysticercosis

Racemose neurocysticercosis (RC) also known as SNCC refers to a very uncommon form of NCC, with the cyst localised mainly in the subarachnoid space and basal cisterns. Usually, the scolex is absent and multiple complex small cysts may form (cluster of grapes), filling the basal cisterns, determining mass effect and distortion of adjacent structures, namely sulci, brainstem and cranial nerves [43–45], these authors consider that in racemose NCC, there is a presence of abnormally large growths of many cystic membranes without a scolex, normally without enhancement, in subarachnoid space and basal cisterns and they found on imagenology that the cysts have a thin wall without a scolex; their signal is isointense or slightly different from CSF, hypointense on T1-weighted images (T1-WIs) and fluid-attenuated inversion recovery (FLAIR), hyperintense on T2-WI, without diffusion restriction, and after contrast there is no wall enhancement. A three-dimensional balanced steady-state free precession sequence (constructive interference in steady state (CISS)), driven equilibrium (DRIVE) or contrast-enhanced MR cisternography helps to detect the underlying cysts [46]. Pamplona et al. [46] reported a case of a *43-year-old woman from Cabo Verde, with an eight-month history of right frontotemporal headaches (without releasing or aggravating factors), ataxia and loss of vision, without significant past medical history of note and no history of head trauma. Home hospital computed tomography (CT) disclosed a large intraventricular cyst, without enhancement after ionic contrast administration, distorting lateral and third ventricles, with obliterations of Monro foramina, determining non-communicating hydrocephalus, with enlargement of occipital and temporal horns of lateral ventricles and ependimary transudation.* They established that *racemose neurocysticercosis (INCC) refers to cysts in the subarachnoid space and is characterised by proliferative lobulated cysts without a scolex.* We also agree with such definition if the presence of scolex is not excluded from the definition, as discussed later. These cysts may vary in size, from 2 to 3 mm in subarachnoid space and basal cisterns, to a few centimetres when intraventricular. Intracranial hypertension and hydrocephalus are two complications that happen when there is a flow obstruction due to intraventricular cysts, arachnoiditis, ependymitis secondary to inflammatory response or mass effect in cases of very large cysts [44, 45].

It is known that in the next several months to years, there is degeneration of the cyst, with associated inflammatory response and peripheral oedema, leading to clinical symptoms and manifestations that may vary according to localisation and mass effect [43–45]. The final stage (calcified) with or without associated oedema is seen in the intraparenchymal presentation and is the main cause of epilepsy in this series; obviously, associated oedema never happens at the subarachnoid space.

The differential diagnosis depends on where the cysts are localised; if in the subarachnoid space and basal cysterns, the differential diagnoses are arachnoid cysts, neuroglial cysts, gliomas, cavernous malformations and echinococcal cysts.

Detection of cysticercal antigens by monoclonal antibody-based enzyme-linked immunosorbent assay (ELISA) in the CSF of clinically suspected patients supports the diagnosis of active SNCC.

3. Stroke

Almost all clinical presentations of stroke can be seen in epileptic patients having SNCC [27, 41, 46–65]. Despite the increasing number of reports on haemorrhagic stroke to the medical literature [19, 20, 23, 24, 26, 27, 29–31, 37–41, 47–62], the prevalence of stroke secondary to NCC remains higher in IS secondary to infectious vasculitis. The most common clinical manifestations of NCC are seizure and headaches. In addition, cysticerci may cause mass effect, obstructive hydrocephalus, intracranial hypertension, cerebral infarction, vasculitis and meningitis [66, 67]. Seizures are more common in intraparenchymal NCC, resulting from cystic perilesional inflammation, new infarcts and vasculitis. Acute encephalitis-like presentation is rare, but can be the first symptom in the paediatric population [66]. Cerebrovascular complications of NCC include lacunar infarction, large-vessel disease, transient ischaemic attacks, progressive midbrain syndrome and brain haemorrhage, resulting from a multiplicity of mechanisms, including luminal narrowing due to sub-intimal cushions, vasospasm due to arteritis in midsized and small perforating vessels of the brain, and fresh thrombi [44, 45].

In our region, the incidence and prevalence of ischaemic stroke due to infectious vasculitis is higher in HIV patients compared with other causes of infectious vasculitis because HIV/AIDS is more common than other mentioned infectious diseases, at the present moment. Tuberculosis (TB) of the CNS and neurosyphilis are not uncommon problems. Unfortunately, the worse prognosis is reserved for HIV/AIDS followed by TB, neurosyphilis and others.

Stroke as a complication of NCC occurs in a very small percentage of cases, mostly involving small perforating vessels while major intracranial vessel involvement is extremely rare. Coleman et al. [47] also reported two autopsied cases of chronic cysticercal basal arachnoiditis causing large arterial territory infarcts and, in the second case, a hypothalamic mass. In their patient, *the diagnosis was established only at autopsy, which revealed SNCC causing basal arachnoiditis, major vessel vasculitis and infarcts. Histologically, case 1 showed degenerating racemose cysticercal cyst within the thick basal exudate. In the second case, remnants of the degenerated cysticercal cyst in the form of hooklets and calcareous corpuscles were identified within the giant cell inciting a granulomatous response to form a hypothalamic mass lesion mimicking tuberculoma*. These authors highlighted the importance of considering the non-tuberculous aetiologies of chronic basal arachnoiditis like SNCC before initiating therapy especially in countries endemic to both NCC and tuberculosis.

> Other authors [48, 49] reported stroke *as a recognised complication of NCC, occurring in 2–12% of cases, mostly in the form of small lacunar infarcts and informed about a 51-year-old Hispanic woman, which was secondary to complete occlusion of the left internal carotid and bilateral anterior cerebral arteries. Their report represents the third reported case of internal carotid artery occlusion and the first reported case of anterior cerebral artery occlusion secondary to neurocysticercosis at that time*. Another author [50] considered that ischaemic stroke *is a relatively common but underrecognised complication of NCC and it is usually caused by inflammatory occlusion of the arteries at the base of the brain secondary to cysticercotic arachnoiditis. In most cases, the involved vessels are of small diameter and the neurological picture is limited to a lacunar syndrome secondary to a small cerebral infarct. However, large infarcts related to the occlusion of the middle cerebral artery or even the internal carotid artery have also been reported* [50]. Viola et al. [57] reported a patient

who *presented with relapsed subarachnoid haemorrhage possibly linked to NCC*. In addition, they performed a *literature review of all of the reported cases of aneurysmal and non-aneurysmal haemorrhagic cerebrovascular events associated with NCC until year 2011 and identified 11 such cases. The majority of the individuals were young males (mean: 38 years; 70% males). Four cases (36%) had aneurysms. Four (36%) others had negative cerebral angiograms and therefore classified as non-aneurysmal, while the remaining three (28%) did not report sufficient information to classify them. All cases with aneurysmal haemorrhage underwent successful surgical repair of the aneurysms. Seven patients received albendazole (including three who have had surgery). Three patients died; all three presented in the pre-albendazole era.* In summary, NCC should be considered in the differential diagnosis of haemorrhagic cerebrovascular events in young patients without classical vascular risk factors who have lived or visited NCC endemic areas [57].

In 1998, using cerebral arteriography Barinagarrementeria and Cantú [27] studied 28 patients (mean age, 37 years) with subarachnoid cysticercosis admitted to their hospital from July 1993 to February 1996 and found that *15 patients had angiographic evidence of cerebral arteritis (53%); 12 of the 15 had a stroke syndrome (P = .02). Eight of the 15 patients (53%) with cerebral arteritis had evidence of cerebral infarction on MRI*. In that series, *the most commonly involved vessels were the middle cerebral artery and the posterior cerebral artery*, as we already discussed [33]. They concluded that *the frequency of cerebral arteritis in subarachnoid cysticercosis was higher than previously reported, and middle size vessel involvement is a common finding, even in those patients without clinical evidence of cerebral ischaemia.*

Some authors [58, 62] consider that NCC *is a disease with protean manifestations, which depends upon the number of parasites, their location and the degree of host inflammatory response. Most common clinical manifestations include late onset epilepsy or symptoms of intracranial hypertension and cerebrovascular complications have been reported to occur in 4–12% of patients* [58, 62]. In the majority of their cases, the diagnoses were ischaemic cerebrovascular events associated with vasculitis and/or thrombosis from surrounding cysts in both small- and large-diameter vessels. *Subarachnoid haemorrhages have been noted in SNCC and have been associated with cerebral aneurysms in some, but not all, cases* [58, 61]. Inflammatory aneurysms are usually located at distal intracranial arteries, not at bifurcations like congenital aneurysms, and are more commonly fusiform in shape. The wall of inflammatory aneurysms and parent vessels are extremely friable, and the possibility of intraoperative rupture is higher; in addition, inflammatory aneurysms are fusiform in shape so clipping of the aneurysm neck while preserving the parent artery is technically challenging [61]. As a result, *they are generally secured by wrapping* [59], *clipping of the proximal artery* [64] *or trapping* [61]. Obviously, SNCC should be considered in the differential diagnosis of aneurysmal and non-aneurysmal haemorrhagic stroke events in all patients living in regions where NCC is endemic.

4. Imagenology

Based on our experience, imagenology is the investigation of choice for confirmation of SNCC. MRI scan is better than CT scan to identify intraventricular NCC and CT is better to confirm calcified intraparenchymal NCC, and both can be used for confirmation of SNCC but

CT scan costs less; for establishing the stage of NCC, one of them are mandatory, and both are necessary before and after the treatment [65–69].

Recently Xiao et al. [70], investigated the imaging features of NCC to provide clinicians with valuable information in the diagnosis and treatment and then investigate the imagenological features of 71 consecutive cases of NCC diagnosed by CT and MRI in 5 years time; finding parenchymal cysticerci in 53 cases (92.9%), subarachnoid cysticerci in 39 cases (68.4%), ventricular cysticerci in 13 cases (22.8%) and spinal cysticerci in 1 case; 35 cases were associated with leptomeningitis, 10 cases were with hydrocephalus and they concluded that the imaging findings of the cysticerci, including their location, numbers, cystic sizes, capsular thickness, densities and signals of the scolices, as well as the peripheral oedema, have distinct value in timely making possible diagnosis of neurocysticercosis for clinicians. Similar characteristics are found in our series [1, 7, 14–18, 33, 71–89]. Combination of SNCC and INCC in the same patient as described by Hauptman [69] was also found in our series.

Apart from CT/MRI, angiographic studies sometimes are necessary to determine the imagenological features of vascular malformations, vasculitis and occlusion of blood vessels of the brain associated with NCC. In a previous study, 15 (53%) out of 28 patients with subarachnoid cysticercosis who underwent cerebral angiography had evidence of cerebral arteritis in middle size arteries (middle cerebral artery and posterior cerebral artery) [27].

> In one of the patient reported by Rocha et al. [48], MRI *demonstrated several enhancing subarachnoid cysts surrounding the occluded vessels, a right parietal racemose cyst and a left temporal large infarction area. Angiographic study showed total occlusion of left middle cerebral artery and a sub-total occlusion of right middle cerebral artery.* In the second case, CT scan *demonstrated several calcifications and a left temporal infarction area. Angiographic study showed diffuse arteritis of basilar and carotid arterial system.* In the patient reported by Levy et al. [49], the MRI demonstrated the presence of enhancing subarachnoid material surrounding these occluded cerebral arteries, providing antemortem, non-invasive documentation of the inflammatory meningeal cysticercoid reaction that was presumably responsible for the occlusive arteritis causing the cerebral infarction. In this setting, CT scan and CSF examination usually support the cause-and-effect relationship between neurocysticercosis and the cerebral infarct by showing abnormalities compatible with cysticercotic arachnoiditis. Pamplona et al. [46] *reported a case of a 43-year-old woman with an eight-month history of headaches, ataxia and loss of vision. CT and MRI showed an intraventricular cyst, causing entrapment of Monro foramina and hydrocephalus, smaller cysts at subarachnoid space in temporal lobes, Sylvian fissures, suprasellar and peri-mesencephalic cisterns, and an intra-orbital cyst. Additionally, there were acute ischaemic vascular lesions on the left thalamus and corpus callosum splenium and sub-acute ischaemic lesions of both occipital lobes.* Gilman et al. [90] reported a patient who presented with a relapsed non-aneurysmal subarachnoid haemorrhage possibly associated with subarachnoid cysticercosis and the *MRI of her brain revealed a new left subarachnoid haemorrhage involving the left suprasellar cistern, inter-peduncular cistern, left ambient cistern, and again the left Sylvian fissure. Additionally, the images showed dilatation of the lateral and third ventricles, and the aqueduct of Sylvius, with obstruction caused by cysts associated with leptomeningeal enhancement of the supracerebellar cistern.* Other authors also reported similar findings [43, 44].

Without doubt, MRI is an ideal test for investigating SNCC. However, imagenological diagnosis of SNCC is usually difficult when classical MRI sequences are used [91]. Therefore, Carrillo et al. [91] evaluated the advantages of 3D MRI sequences [fast imaging employing steady-state acquisition (FIESTA) and spoiled gradient recalled echo (SPGR)] with respect to classical sequences [fluid attenuation inversion recovery (FLAIR) and T1] in visualising *T. solium* cyst in

these locations and they found that 47 *T. solium cysts located in the basal cisterns of the subarachnoid space were diagnosed in 18 Mexican patients. A pre-treatment MRI was performed on all patients, and all four sequences (FIESTA, FLAIR, T1 SPGR, and T2) were evaluated independently by two neuroradiologists*. The mentioned authors found *FIESTA sequences allowed the visualisation of cyst membrane in 87.2% of the parasites evaluated, FLAIR in 38.3%, SPGR in 23.4% and T2 in 17.0%. Therefore, the superiority of FIESTA sequences over the other three imaging methods was statistically significant ($P < 0.001$). Scolices were detected by FIESTA twice as much as the other sequences did, although this difference was not significant ($P > 0.05$). Differences in signal intensity between CSF and parasite cysts were significant in FIESTA ($P < 0.0001$), SPGR ($P < 0.0001$) and FLAIR ($P = 0.005$) sequences*, and they [91] concluded that, *for the first time, the usefulness of 3D MRI sequences to diagnose T. solium cysts located in the basal cisterns of the subarachnoid space was demonstrated. The routine use of these sequences could favour an earlier diagnosis and greatly improve the prognosis of patients affected by this severe form of the disease.* An accurate diagnosis of this condition is important since early treatment with steroids is advised to ameliorate the subarachnoid inflammatory reaction which may cause recurrent cerebral infarcts [50].

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