Cerebral Cysticercosis and Stroke

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Background and Purpose: In 1985 we initiated a protocol for examining the relationship between cerebral cysticercosis and stroke.

Methods: In 420 stroke patients admitted to our department, our standard protocol of tests included blood tests, cardiac investigations, angiography, and immunologic cerebrospinal fluid measures. We assessed the following possible risk factors: arterial hypertension, diabetes, cardiopathy, high levels of cholesterol and triglycerides, smoking, alcohol abuse, and cerebral cysticercosis.

Results: Of the 420 patients with stroke, we found cerebral cysticercosis in 31, five of whom were >65 years of age and 26 of whom were <65 years. We determined that cerebral cysticercosis was the only possible risk factor for stroke in one of the five older patients and 15 of the 26 younger and middle-aged patients. Cortical infarctions were found in five of the 31 patients, with cerebral cysticercosis and lacunar infarctions in nine of these patients. One patient had intracystic hemorrhage. In 16 cases, neurological deficit was related to single or multiple cysts, colloids, granulomas, diffuse lesions, or pericystic edema. All patients with cerebral cysticercosis quickly recovered from their neurological deficit, except one who had a hemorrhagic cyst and died and another who remained disabled.

Conclusions: We established that, in patients with neurocysticercosis, occlusion of the small cortical or penetrating vessels at the base of the brain caused by arteriopathy was the most common mechanism of the stroke. Moreover, there is a probable association between cerebral cysticercosis and the susceptibility to stroke, particularly among young and middle-aged patients. (Stroke 1992;23:224-228)

Neurocysticercosis, a major public health problem in large areas of the world,1-2 is a parasitosis with pleomorphic clinical expression. In large series of patients with cerebral cysticercosis, the incidence of stroke varies between 4% and 12%,3-7 but in those areas where neurocysticercosis is endemic, 10% of patients with a diagnosis of stroke may have cerebral cysticercosis.8 Angiographic studies have rarely shown arterial thrombosis in cerebral cysticercosis.3,4,9-13 In those patients with cerebral cysticercosis and stroke, vascular events have been related to thrombosis of the superficial cortical vessels owing to chronic meningitis,14,15 to arteritis induced by the presence of adjacent cysts,12,15-18 to fusiform aneurysms produced by weakening of the wall of the vessels,12 and to occlusion of small perforating vessels affected by endarteritis.19-20

Some pathophysiological mechanisms of stroke in those patients with cerebral cysticercosis have been thoroughly explained, whereas others remain insufficiently understood. Parasitoses such as cerebral cysticercosis could well constitute a cause of stroke. The present study describes a group of patients with a relatively high association of stroke and cerebral cysticercosis and examines possible factors causing this association.

Subjects and Methods

In all patients admitted with a diagnosis of stroke to the Department of Neurology of the Eugenio Espejo Hospital in Quito, Ecuador, between October 1985 and November 1988, we applied a standard protocol to analyze clinical characteristics, diagnostic tests, and risk factors. Three neurologists filled out the questionnaires of 420 patients who complied with the criteria established in the protocol.

Stroke was defined as the rapid development of signs of focal or global disturbance of the cerebral function, lasting >24 hours or leading to death, without any other apparent cause.21 According to this definition, transient ischemic attacks were excluded.

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The definitions and guides for the diagnostic classification of stroke were essentially those recommended by the World Health Organization,22 with subtypes based on the schemes developed by the Pilot Stroke Data Bank.23 The diagnosis of stroke was determined by neurological examination, computed tomography (CT), angiography, cerebrospinal fluid (CSF) analysis, or autopsy. The neurological signs at onset were evaluated during the first 24 hours after admission. All patients were admitted to the hospital within the first 96 hours of onset of symptoms.

The following possible risk factors were assessed in the first 30 days: arterial hypertension (>160/90 mm Hg); diabetes mellitus (fasting blood sugar >140 mg/dl or glucose tolerance test with 2-hour postprandial value >200 mg/dl); cardiac disease, based on assessment by a cardiologist for rheumatic heart disease, nonrheumatic atrial arrhythmia, left ventricular hypertrophy, coronary insufficiency, myocardial infarction, or cardiomyopathy; hyperlipidemia (cholesterol level >200 mg/dl or triglycerides >150 mg/dl); habitual cigarette smoking; alcohol abuse; and cerebral cysticercosis diagnosed by means of CT scan, CSF, immunologic tests, or other tests performed to establish a diagnosis of this parasitosis.24 Evans's index was used to determine the severity of hydrocephalus.25

A cardiological evaluation was carried out on all patients, including Holter and two-dimensional echocardiography. Of the 420 patients with stroke, cerebral angiography was performed on those patients with no or only ill-defined risk factors, in those with subarachnoid hemorrhage, and in young patients ≤45 years of age.

Results

Of the 420 patients diagnosed with stroke and included in the standard protocol, 31 (7.3%) had cerebral cysticercosis. The mean age was 52 (range 17–86) years. Twenty-one patients were men and 10 were women; 11 were ≤45 years of age and five were >65 years of age. In the histories of the 31 patients with cerebral cysticercosis, five had neurocysticercosis, seven were epileptic, and seven had sudden neurological deficit. The incidence of cerebral cysticercosis among other hospitalized neurology patients who did not have stroke was 3.6%.

All 31 patients were hemiparetic when examined. Seven patients had hemihypesthesia; seven, a central seventh palsy; two, homonymous hemianopia; two, clouding of consciousness; two, aphasia; two, dysarthria; and two, papilledema. Other manifestations during the evaluation were headache in 15 patients, intracranial hypertension in two patients with hydrocephalus, and dementia in one case. Twelve patients (39%) without history of epilepsy and one with a history of epilepsy experienced a seizure during the acute vascular event. One year after the acute deficit, it was established that 19 patients (61%) had epilepsy. The signs and symptoms of stroke reached their maximum deficit in <24 hours in 24 cases (77%) and in <48 hours in the seven remaining cases (23%). The neurological recovery after the stroke was complete in 19 patients (61%) and partial in 11 patients (35%); of these 11 partially recovered patients, 10 returned to work within 6 weeks, whereas one patient was unable to lead an independent life. One patient died during the acute stroke, with the autopsy finding of hemorrhage into a frontal lobe cyst.

Table 1 shows the nature of the lesions detected on CT scan. Six patients (19%) had hydrocephalus; of these, two had severe intracranial hypertension and four were asymptomatic. Table 2 shows the distribution of abnormalities in these patients with and without vascular risk factors. Figures 1 and 2 show examples of findings in two hydrocephalic patients.

In nine patients with large, single-cortical cysts with or without pericystic edema (>20 mm in diameter), a cause-and-effect relationship was established with the neurological deficit (Figure 3). In five patients (16%) with cortical infarctions and in nine patients (29%) with lacunar infarctions, there was correlation with the hemiparesis. In the patient with hemorrhage, there was also a correlation with the motor deficit. In the seven remaining patients (23%), the focal neurological deficit was related to pericystic edema in deeply located cysts and the presence of colloid, granulomas, diffuse lesions, and, in one case, multiple cortical cysts. Seven of the nine lacunar infarctions were related to subarachnoid cysticercosis: five had sylvian and two had suprasellar locations;

<table>
<thead>
<tr>
<th>Type of lesion</th>
<th>No. cases</th>
<th>%</th>
<th>No. cases</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyst</td>
<td>13</td>
<td>41.9</td>
<td>14</td>
<td>45.1</td>
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<tr>
<td>Diffuse lesion</td>
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<td>...</td>
<td>1</td>
<td>3.2</td>
</tr>
<tr>
<td>Colloid</td>
<td>1</td>
<td>3.2</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Granuloma</td>
<td>1</td>
<td>3.2</td>
<td>2</td>
<td>6.4</td>
</tr>
<tr>
<td>Calcification</td>
<td>...</td>
<td>...</td>
<td>16</td>
<td>51.6</td>
</tr>
</tbody>
</table>

Table 1. Computed Tomographic Findings of Cerebral Cysticercosis

CT scan and autopsy findings

<table>
<thead>
<tr>
<th>CT scan and autopsy findings</th>
<th>Without risk factors</th>
<th>With risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. cases</td>
<td>%</td>
</tr>
<tr>
<td>Cortical infarcts</td>
<td>3</td>
<td>18.7</td>
</tr>
<tr>
<td>Lacunar infarcts</td>
<td>5</td>
<td>31.2</td>
</tr>
<tr>
<td>Intracystic hemorrhage*</td>
<td>1</td>
<td>6.2</td>
</tr>
<tr>
<td>Cases not displaying cerebral infarct or hemorrhage</td>
<td>7</td>
<td>43.7</td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
<td>100</td>
</tr>
</tbody>
</table>

CT, computed tomography. *Confirmed by autopsy.
Cerebrospinal fluid (CSF) tests were carried out on all the patients, 15 of whom (48%) showed signs of meningitis. Average CSF values for these patients were as follows: leukocytes 19.4/mm³, protein 55.4 mg/dl, and glucose 45.3 mg/dl. In 14 cases, the CSF showed indirect positive hemagglutination for cysticercosis.

Carotid angiography was performed in 20 patients and vertebral angiography in one. Vasculitis, found in two patients, was related to a subarachnoid cyst and lacunar infarction in one case and to a huge cortical cyst in the other (Figure 3). An avascular mass with displacement of the midline structures was found in 10 patients.

Since we established a protocol in 1984 for standardized collection of the clinical data and complementary tests in patients with symptomatic neurocysticercosis, five patients have been autopsied. Two of the five patients had a history of stroke. In the blood vessels adjacent to the parasite, there was endothelial hyperplasia as well as fibrosis of the media. Adventitial hyperplasia contributed to reduction of the vessel or even occlusion of the lumen. In the cases of basal meningeal cysticercosis, the blood vessels around the circle of Willis and in the penetrating vessels also showed these changes.

In one of the 11 patients ≤45 years of age, hyperlipidemia was found; in another, smoking and alcohol abuse; and in five, CSF changes reflecting meningeal inflammation. In nine cases (81.8%), cere-
cerebral cysticercosis was established as the only possible risk factor for stroke.

We found cardiac disease in four of the 15 patients who were >45 years but ≤65 years old. Of these four, three had hypertrophy of the left ventricle, and one showed rheumatic heart disease with atrial fibrillation. Three of these middle-aged patients had arterial hypertension, one had diabetes, two had hyperlipidemia, two had smoked cigarettes, and two had abused alcohol.

In three of the five patients >65 years of age, a diagnosis of cardiac disease was established; of these three, one showed hypertrophy of the left ventricle, one showed coronary insufficiency, and the remaining patient showed myocardial infarction. Two of these older patients had arterial hypertension, one had diabetes, two had hyperlipidemia, and one had meningitis. In one patient, cerebral cysticercosis was the only possible risk factor that could be established for stroke.

Discussion

The association of cerebral cysticercosis and stroke has been widely accepted. Various mechanisms are involved in the genesis of vascular events in patients with cerebral cysticercosis. The average age of our patients was lower than the age reported in other series with stroke, which is related to the fact that cerebral cysticercosis predominates in young adults. The high percentage of epilepsy found in our cases is similar to that found in other studies with cerebral cysticercosis, but higher than that found in patients with stroke. A high percentage of patients experienced a seizure during the acute vascular event, which could be related to the cortical location of the parasitic lesions, the edema or infarction surrounding the cerebral cysticercosis lesions, and the probable participation of meningeal cysticercosis in the genesis of stroke in these patients.

We could not demonstrate occlusion of a large vessel in any of our cases, although the CT scan showed cortical infarctions and large single or multiple cysts related to the neurological deficit of the patient. The endothelial proliferation adjacent to the cortical cysts found in our autopsied cases, and previously reported by other authors, especially affected the small vessels, as was demonstrated by angiography in one of our cases (Figure 3).

A cause-and-effect relationship between the two main risk factors of stroke, that is, cardiac disease and arterial hypertension, could be established in only three patients. We found that the only possible risk factor for stroke in 57.6% of patients ≤65 years of age was cerebral cysticercosis.

It is a well-documented fact that subarachnoid cysticercosis or subacute, chronic, or recurrent meningitis produces abnormal thickening of leptomeninges at the base of the skull and endarteritis of blood vessels around the circle of Willis. This vascular compromise can lead in some cases to the complete occlusion of a blood vessel, with subsequent infarction (Figure 2).
These mechanisms would explain the presence of lacunar infarctions related to subarachnoid cisticercosis or meningitis in our patients. In two patients with deeply located cysts, the neurological deficit was related to arteriopathy adjacent to the cyst found in the autopsy. In two patients who displayed cortical infarctions in their CT scans, without any evidence of a subarachnoid or adjacent cortical cyst, and who also had meningitis (Figure 1), the vascular affection could be explained by its proximity to a CSF reflecting meningeal inflammation. Our findings, together with the rapid and notable improvement of the neurological signs in those patients with stroke and cerebral cisticercosis, suggest that cerebral cisticercosis preferably affects the small arteries. We are in agreement with other authors in our findings that cerebral cisticercosis must be included in the list of causes of stroke, especially in young and middle-aged people, in those countries where this parasitosis is endemic and in developed countries with a high immigrant population coming from developing countries where neurocysticercosis is endemic. Epidemiological studies are needed to confirm the potential association between cerebral cisticercosis and stroke suggested by our study. In addition, a greater knowledge of the pathophysiology of patients with cerebral cisticercosis would contribute to a better understanding of the mechanisms related to the production of cerebrovascular diseases.

References

Key Words • cerebrovascular disorders • cisticercosis • risk factors • vasculitis
Cerebral cysticercosis and stroke.
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