Frequency of Cerebral Arteritis in Subarachnoid Cysticercosis
An Angiographic Study

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Background and Purpose—Subarachnoid cysticercosis is a well-recognized cause of cerebral infarction. However, few patients with this infection develop cerebral infarction, and the reason for this is not known. The aim of this study was to determine the frequency of cerebral arteritis in these patients.

Methods—Using cerebral arteriography, we studied 28 patients with subarachnoid cysticercosis admitted to our hospital from July 1993 to February 1996. All patients underwent MRI to detect the presence of basal arachnoiditis. We analyzed demographic data, time to cysticercosis since the first symptom onset, mode of onset, stroke syndromes, neuroimaging features of cysticercosis and cerebral infarction, and arteriographic findings for each patient.

Results—Of the 28 patients (mean age, 37 years), 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, whereas only one patient without cerebral arteritis had cerebral infarction (P=.05). The most commonly involved vessels were the middle cerebral artery and the posterior cerebral artery.

Conclusions—The frequency of cerebral arteritis in subarachnoid cysticercosis is higher than previously reported, and middle-size vessel involvement is a common finding, even in those patients without clinical evidence of cerebral ischemia. (Stroke. 1998;29:123-125.)

Key Words: arteritis ▪ cerebral infarction ▪ cysticercosis ▪ meningitis

Cerebrovascular complications of neurocysticercosis include cerebral infarction, transient ischemic attacks, and brain hemorrhage. The most common mechanisms by which cysticercosis produces cerebrovascular disease are related to cerebral arteritis, mainly in patients with subarachnoid cysticercosis. Several clinical reports of cerebral infarction resulting from cysticercosis showed that primarily small brain vessels are compromised by the parasite. A few series studied the role of cerebral arteriography in the evaluation of patients with cysticercosis. However, the prevalence of angiographically documented cerebral arteritis associated with subarachnoid cysticercosis is unknown. Little is known about the frequency and features of cysticercotic arteritis despite the high prevalence of cysticercosis in several countries. The goals of the present study were to determine both the frequency of angiographic abnormalities in patients with subarachnoid cysticercosis and the clinical findings in this patient group.

Subjects and Methods
Among 1454 consecutive patients admitted to our Neurology Department from July 1993 to February 1996, 102 consecutive patients with cerebral cysticercosis were evaluated. Forty-nine patients with subarachnoid cysticercosis were selected to undergo four-vessel arteriography. Twenty-eight patients provided informed consent and underwent cerebral arteriography.

All patients met the following diagnostic criteria for subarachnoid cysticercosis: a positive immunologic reaction to cysticerci in cerebrospinal fluid and MRI consistent with definitive evidence of cysticerci cysts in the subarachnoid space.

In each case, demographic data, the time to cysticercosis since the first symptom of cysticercosis onset, risk factors, mode of onset, stroke syndromes, neuroimaging features of cysticercosis and cerebral infarction, and arteriographic findings were analyzed.

The mode of onset was defined as vascular when the presenting complaints resulting from cysticercosis were related to a symptomatic cerebral infarction or transient ischemic attack, and nonvascular when the presenting complaints of cysticercosis were related to seizures, intracranial hypertension, chronic meningitis, a progressive focal neurological deficit, and mental disorders including dementia and psychiatric disturbances. Acute onset was considered when symptoms appeared suddenly or developed in less than 48 hours.

The following stroke syndromes were considered: (1) symptomatic cerebral infarction, defined as rapidly developing focal loss of cerebral function with symptoms and signs lasting more than 24 hours that was associated with a corresponding area on MRI; (2) silent cerebral infarction, defined as an asymptomatic ischemic brain lesion disclosed by neuroimaging studies; and (3) transient ischemic attack, defined as an acute focal neurological deficit from ischemic origin that lasted less than 24 hours.

In all patients, an MRI scan was performed with gadolinium enhancement. The following cysticercous neuroimaging features were evaluated: (1) focal arachnoiditis when there was contrast enhancement in only one cerebral basal cistern; (2) diffuse arachnoiditis, in which contrast enhancement involved several basal cisterns; and (3)
cerebral infarction, in which the number and location of cerebral infarctions were analyzed and classified as superficial, deep nonlacunar (>20 mm), and deep lacunar. In each case with cerebral infarctions, the close relation with a neighboring subarachnoid cysticercus cyst was evaluated.

Four-vessel cerebral arteriography using the technique of Seldinger was performed in all patients. The arteriographic criteria for diagnosis of cerebral arteritis included segmental narrowing, a beaded appearance of the cerebral vessels, and abrupt or tapered areas of vascular obstruction.13,14

Demographic, clinical, and neuroimaging features were analyzed in accordance with the presence of arteriographic abnormalities (group A) or normal findings (group B). The differences between the groups were evaluated for statistical significance with the use of the \( \chi^2 \) test and Fisher’s exact test.

### Results

We studied 28 patients, 19 males (68%) and 9 females (32%), who had a mean age of 37 years (range, 16 to 58 years). Risk factors included tobacco and alcohol use in 3 each (11%), diabetes mellitus in 2 (7%), and arterial hypertension in 1 (4%).

Fifteen of the 28 patients (53%) had arteriographic evidence of cerebral arteritis; their demographic and clinical data compared with those patients without arteritis are shown in Table 1.

A stroke syndrome was found in 80% of patients with cerebral arteritis (\( P=.02 \)). None of these patients had a history of cerebrovascular disease. Vascular onset as the first manifestation of cerebral cysticercosis was more common in patients with cerebral arteritis than in those without. In 20% of patients with cerebral arteritis, this finding was asymptomatic (Figure).

Regarding the frequency of cerebral infarction demonstrated by MRI, 8 of 15 patients (53%) with cerebral arteritis had evidence of cerebral infarction, whereas only one patient without cerebral arteritis (7%) had evidence of cerebral infarction (\( P=.05 \)). The characteristics of cerebral infarctions are shown in Table 2.

Cerebral arteritis was angiographically documented in 30 vessels in 15 patients. The most commonly affected vessels were the middle cerebral artery and the posterior cerebral artery, which were involved in more than half of the patients. The numbers of affected vessels were as follows: one vessel in 8 patients (53%), two vessels in 4 (26%), and three, five, and six vessels in 1 patient each (6%). Among 7 patients with multiple-vessel involvement, 5 of them (71%) had diffuse arachnoiditis; only 3 of 8 patients (37%) with one-vessel involvement had diffuse arachnoiditis.

### Table 1. Demographic Data and Clinical Features of Patients With (Group A) and Without (Group B) Cerebral Arteritis

<table>
<thead>
<tr>
<th>Finding</th>
<th>Group A (n=15)</th>
<th>Group B (n=13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, M:F</td>
<td>12:3</td>
<td>7:6</td>
</tr>
<tr>
<td>Mean age, y (range)</td>
<td>40 (22-58)</td>
<td>35 (16-58)</td>
</tr>
<tr>
<td>One risk factor</td>
<td>2 (13)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Two risk factors</td>
<td>1 (7)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Mean time to cysticercosis</td>
<td>5 mo</td>
<td>31 mo</td>
</tr>
<tr>
<td>Acute onset</td>
<td>6 (40)</td>
<td>2 (15)</td>
</tr>
<tr>
<td>Vascular onset</td>
<td>6 (40)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Nonvascular onset</td>
<td>9 (60)</td>
<td>12 (92)</td>
</tr>
<tr>
<td>Stroke syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transient ischemic attack</td>
<td>3 (20)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Silent cerebral infarction</td>
<td>1 (7)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Symptomatic cerebral infarction</td>
<td>7 (46)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Brain hemorrhage</td>
<td>1 (7)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Non–stroke syndrome</td>
<td>3 (20)</td>
<td>11 (84)</td>
</tr>
</tbody>
</table>

Values are number (percentage), unless otherwise indicated.

* \( P=.05 \)

\( \dagger \) \( P=.02 \)

\( \ddagger \) \( P=.0006 \)

A, T1-weighted MRI shows a basal cysticercus (large arrow) in peduncular cistern. B, Cerebral arteriography, vertebrobasilar territory, anteroposterior view discloses a long, narrow segment of the left posterior cerebral artery (small artery).
TABLE 2. Neuroimaging Features in Nine Patients With Cerebral Infarction

<table>
<thead>
<tr>
<th>Findings</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic</td>
<td>8/9</td>
<td>89</td>
</tr>
<tr>
<td>Location</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superficial</td>
<td>2</td>
<td>22</td>
</tr>
<tr>
<td>Deep lacunar</td>
<td>3</td>
<td>33</td>
</tr>
<tr>
<td>Deep nonlacunar</td>
<td>4</td>
<td>44</td>
</tr>
<tr>
<td>Arterial distribution</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carotid territory</td>
<td>7</td>
<td>78</td>
</tr>
<tr>
<td>Vertebrobasilar territory</td>
<td>2</td>
<td>22</td>
</tr>
</tbody>
</table>

Discussion

Angiographically documented cerebral arteritis has been poorly demonstrated. The first angiographic study of cysticercosis was performed by Moniz et al., who reported two patients with arteritis at the level of the internal carotid artery. Lombardo and Mateos described the angiographic findings in seven patients with cysticercosis and reported only elongation of the pericallosal artery resulting from hydrocephalus, with no mention of cerebral arteritis. After studying eight patients, Santín and Vargas concluded that cerebral arteriography is not useful in cerebral cysticercosis. Rocca and Monteagudo reported angiographic abnormalities in 23 of 46 patients (50%) with cysticercosis. Cysticercosis was not described in detail in any of those studies. In the last few years, several case reports of cerebral infarction related to cysticercosis have been published, few of them with angiographic abnormalities.

The frequency of symptomatic cerebral arteritis in patients with cysticercosis is not well known. In pathological studies, the presence of endarteritis in the vessels located in the vicinity of the parasite is a well-recognized phenomenon. The frequency of cerebral infarction related to cysticercotic arteritis varies in published series between 2% and 12%. In the present series, 53% of all patients with subarachnoid cysticercosis had angiographically documented cerebral arteritis, most of them symptomatic (80%). Interestingly, one in five patients with subarachnoid cysticercosis could have asymptomatic cerebral arteritis. The natural history of this form of arteritis is not known. Otherwise, 15% of those patients without evidence of cerebral arteritis after cerebral arteriography had a stroke syndrome, probably the result of small-vessel involvement, as has been previously recognized.

In the present study the major involved vessels corresponded with the major intracranial arteries, mainly the middle cerebral artery, as has been reported in seven of 13 cases.

In conclusion, our study demonstrates a high frequency of cerebral arteritis in patients with subarachnoid cysticercosis and a close relation between arteritis and the presence of cerebral ischemia (definite or transient). The distribution of cerebral ischemic lesions is mainly in the deep portion of the brain and results from the involvement of medium-size arteries. The recognition of this complication could modify conventional therapy to prevent cerebrovascular complications, which increase the morbidity and mortality in this disease.

References

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