In all our patients, we discarded other causes of nonfocal cerebral dysfunction of a vascular origin such as synapses, and we performed a CT scan to discard other causes of sudden focal neurological deficit. Stroke is clinically defined as the occurrence of symptoms and signs with sudden (within seconds) or at least rapid (within hours) onset, which corresponds to the afectation of vascular areas in the brain. All our patients met these conditions and displayed one-sided neurological signs, which strongly supports the clinical diagnosis of stroke.

Having proposed in our article that cerebral cysticercosis preferably affects the small vessels, it is not surprising that we do not have CT evidence of cerebral infarction in a high percentage of our patients. In other patients with lesions in small vessels, such as those with lacunar infarcts in routine CT scans, there is a positivity of between 30% and 50% of cases; even with repeat CT scans and magnetic resonance imaging, the positivity attains a rate of 89%. These facts and the angiographic evidence in one of our vasculitis patients, in whom the CT scan did not show infarct, reassert our thesis.

In two patients without risk factors and with cortical infarcts, we established that the vascular affection mechanism could be caused by its proximity to cerebrospinal fluid reflecting meningeal inflammation. We do not know with certainty whether the damage from cerebral cysticercosis is directly on the cerebral blood vessels or if it is mediated by other mechanisms. The neurocysticercosis could be an associated or independent risk factor for stroke. The risk factor contributes to the susceptibility to stroke and may or may not be the cause of stroke. There is no doubt, however, that in some of our 31 patients, the cause of stroke was not established.

Our major concern was to show that cerebral cysticercosis is a risk factor for stroke. We have studied patients with stroke in whom we have been unable to demonstrate vasculitis by angiography but have been able to do so with intracranial surgery or autopsy. On the other hand, we have studied patients with cerebral cysticercosis without stroke in whom we have found severe vasculitis by means of angiography and surgery. These patients undoubtedly have a potential risk for stroke.

We agree that there are new, demonstrable causes of stroke among young patients, especially in a subgroup with no known risk factors for stroke, and that some of the patients of our series could have one of these causes. In November 1988, we modified our standard protocol for stroke to improve research among young patients, and we have been able to establish that cerebral cysticercosis was the attributable cause of stroke in only 3% of them.

There is a probable association between cerebral cysticercosis and susceptibility to stroke. It is, therefore, a possible risk factor for stroke that should be taken into account, particularly among young and middle-aged patients; and in some cases, it is the attributable cause of stroke.

Fernando Alarcón, MD
Departamento de Neurología
Hospital Eugenio Espejo
Quito, Ecuador

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Thrombin Activity in Cerebrospinal Fluid After Subarachnoid Hemorrhage
Cerebral vasospasm is a major cause of deterioration in patients with subarachnoid hemorrhage (SAH). Several authors have reported extreme activation of the coagulation system in blood and cerebrospinal fluid (CSF) after severe SAH. Thrombin, the key enzyme of the coagulation cascade, is considered as a candidate responsible for vasospasm.

Recently, a sensitive enzyme immunoassay was developed to measure the thrombin activity of the thrombin-antithrombin (TAT) complex. These developments prompted us to examine the sequential changes in thrombin activity in the CSF and blood of 10 patients with severe SAH (computed tomographic grading of SAH by Fisher, group III). The patients were operated on within 48 hours of SAH. Samples of blood and CSF from cisternal drainage were collected from them on days 2–5, days 7–10, and days 14–16. Thrombin activity was analyzed as TAT complex. Angiography was performed in all cases on days 7–9 and revealed the presence of diffuse severe vasospasm (over 70% reduction in diameter and over 2 cm in length) in six cases. Three of these six cases showed neurological deficits as well.

The patients were divided into two groups: a spasm group (those with symptomatic and/or angiographical vasospasm; n=6) and a negative group (those without such vasospasm; n=4). For statistical comparison, we used two sample t tests with Welch’s correction.

![Figure 1. Sequential changes in the thrombin–antithrombin (TAT) III complex in cerebrospinal fluid (CSF) (upper panel) and blood (lower panel) of patients with subarachnoid hemorrhage. Angio+Sympt, patients with angiographic and/or symptomatic vasospasm (n=6); Neg, patients without such vasospasm (n=4). *p<0.05 by two sample t tests with Welch’s correction.](http://stroke.ahajournals.org/content/41/11/1181.full)
tion. Levels of TAT in CSF of the spasm group (938.7±619.2, mean±SD) were significantly higher than those in CSF of the negative group (182.5±108.5, p<0.05) on days 2–5, after which they decreased sequentially and reached the same level as the negative group on days 14–16. The negative group value remained unchanged throughout these periods. Levels of TAT in blood, however, were higher than the normal control levels (below 3.0 ng/ml) and decreased gradually, but there was no significant difference between the two groups (Figure 1).

During this decade, thrombin has been proven to have many biological roles other than coagulation. It facilitates the induction of endothelin-1 (ET-1) gene expression, the release of serotonin and platelet-derived growth factor (PDGF) from platelets, and the recruitment of inflammatory cells. ET-1, serotonin, and PDGF are potent vasoconstrictors, and PDGF is also a candidate for causing proliferative vasculopathy after subarachnoid hemorrhage. Inflammation has also been speculated to be a factor in the etiology of vasospasm. In this context, thrombin activation in CSF might be the initial cause of vasospasm; therefore, measurement of thrombin activity may be important in predicting vasospasm.

Michiyasu Suzuki, MD
Akina Ogawa, MD
Yoshinori Sakurai, MD
Akiko Nishino, MD
Koji Ueno, MD
Kazuo Mio, MD
Takashi Yoshimoto, MD
Division of Neurosurgery
Institute of Brain Diseases
Tohoku University of Medicine
Sendai, Japan

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E. Clarke Haley Jr., MD
Neal F. Kassell, MD
Department of Neurology
University of Virginia
Health Sciences Center
Charlottesville, Va.

Sneddon’s Syndrome and Antiphospholipid Antibodies: Clarification of a Controversy by Skin Biopsy?

The possible role of antiphospholipid (aPL) antibodies in the pathogenesis of Sneddon’s syndrome (SS) was emphasized in several reports, but the significance is still controversial. Recently, we investigated 17 patients with clinical manifestations of cerebral infarcts associated with livedo racemosa. The diagnosis of SS was confirmed by skin biopsy and magnetic resonance imaging of the brain. In addition to standard laboratory tests, we determined aPL antibodies (including anticardiolipin antibodies and lupus anticoagulant factor) and performed Venereal Disease Research Laboratory (VDRL) and partial thromboplastin time (PTT) tests in all patients. None of the 17 patients had evidence of an aPL antibodies, VDRL was nonreactive, and PTT was within the normal range. These results agree with previous studies.

Considering our analysis, the following aspects may help to distinguish SS from other phenomenologically similar disorders. First, it is important to make a clear distinction between livedo racemosa and livedo reticularis. In our study, the European nomenclature was used. Livedo racemosa is a violaceous, netlike pattern of the skin. Irregular, broken circular segments are seen on the trunk and extremities and persist after warming of the skin. The regular, netlike appearance of livedo reticularis is caused by functional disturbances (i.e., vasocostriction). In contrast, livedo racemosa results from focal and persistent impairment of peripheral blood flow caused by occlusions of small arteries in cases of vasculitides or SS.

Second, a special strategy for skin biopsy is necessary to demonstrate the histopathologic features of SS. According to the recommendations of Copeman and Zelger and colleagues, biopsy cones must be obtained from the normal-appearing center of the skin lesions. The biopsy must include subcutis and serial sections, as they are necessary to identify arteriopathy in SS. Based on this clinical distinction and biopsy procedures, we found characteristic histopathologic alterations in all patients. The findings consisted of inflammatory changes of the endothelium (endothelitis) in the early stage, which was later followed by subendothelial cell proliferation with partial and complete occlusion of the involved arteries. Insufficient biopsy techniques may explain negative histological findings in previous reports.

The absence of aPL antibodies in our patients supports the view that aPL antibody arteriopathies represent an entity different from SS. However, histopathologic studies according to our criteria are necessary in aPL antibody-positive patients and SS to prove their value for differential diagnosis, which could be carried out in an international SS cooperative study group, as recommended by Levine.
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