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Asymptomatic Cerebral Infarction in Patients With Chronic Atrial Fibrillation

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A retrospective analysis of 54 patients with atrial fibrillation presenting with symptoms of cerebral ischemia between 1980 and 1985 was performed. Seven patients (13%) had computed tomographic evidence of previous, clinically silent cerebral infarction. In a control group of 168 persons (studied prospectively) in sinus rhythm presenting with symptoms of cerebral ischemia, seven (4%) had computed tomographic evidence of previous, clinically silent cerebral infarction ($p < 0.05$). In those patients with atrial fibrillation all infarcts were peripheral and consistent with embolism, while in three of the seven patients in sinus rhythm the asymptomatic infarcts were lacunes. (*Stroke* 1988;19:955-957)

Chronic atrial fibrillation is a recognized risk factor for cerebral infarction. The Framingham Study documented the risk of an initial cerebral infarct in patients with atrial fibrillation to be 5.6 times greater than that in patients in sinus rhythm when age, blood pressure, and cardiac disease are taken into account.¹ The risk of first stroke is 4.1%/yr in patients with nonrheumatic atrial fibrillation and 4.5%/yr in patients with rheumatic valve disease etiology and atrial fibrillation.¹ As this rate of stroke occurrence approximates that of serious hemorrhagic complications with conventional anticoagulation therapy,² most clinicians do not use anticoagulants to prevent primary stroke in patients with atrial fibrillation. The risk for recurrent stroke following an episode of cerebral embolism is considerably higher, with recurrence rates of approximately 20% for the first year³⁻⁵ and 37% for 3 years.⁴ The problem with secondary stroke prophylaxis using anticoagulation in patients with atrial fibrillation is that many initial strokes in this situation are extensive, with severe residual neurologic disability.^{3,6} However, the first clinical presentation with an ischemic stroke syndrome may not be the first episode of cardiogenic embolic brain infarction; cerebral emboli causing infarction may be asymptomatic. Furthermore, recurrent multifocal cerebral infarction due to cardiogenic embolism may lead to progressive cognitive disturbance and multi-infarct dementia without clinically distinct

stroke events. The purpose of our study was to assess the prevalence of previous asymptomatic cerebral infarction in patients with atrial fibrillation at the time of their initial symptomatic cerebral ischemic event.

Subjects and Methods

Admissions to a public teaching hospital between 1980 and 1985 were retrospectively reviewed. The medical records of all patients with coding on discharge for both atrial fibrillation and cerebral ischemia (ischemic stroke and transient cerebral ischemia) were examined. When a patient had been admitted to the hospital more than once during the study period, only the first admission with symptomatic cerebral ischemia was considered. Only patients with confirmed atrial fibrillation on electrocardiography (ECG) who had a cranial computed tomogram (CT scan) performed during the coded admission were included. Details regarding diagnosis and clinical localization of the presenting cerebrovascular syndrome, previous cerebrovascular events, cause and duration of atrial fibrillation, and coexistent cardiac disease were obtained from the medical records.

The medical records of 160 patients coded for cerebral infarction and atrial fibrillation were reviewed, and from these 63 were identified in whom cranial CT scan results were available and the diagnosis of atrial fibrillation was supported by ECG. Nine of these 63 patients had been incorrectly coded as having had cerebral infarction, and in fact they had suffered intracranial hemorrhage and were thus excluded. It is interesting to note that one of these nine patients, who presented with a right temporoparietal intracerebral hemorrhage, in fact

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had two asymptomatic infarcts present on the CT scan.

A control group of persons admitted to the hospital with cerebral ischemia (transient ischemic attacks or stroke) without atrial fibrillation was obtained by reexamining a series of persons seen prospectively during 1985 in a study of cardiac disease associated with cerebral ischemia. That study had evaluated all persons admitted to the same hospital with cerebral ischemia over a 6-month period.⁷ All were assessed neurologically after admission; details regarding previous and presenting cerebrovascular events were obtained, and cranial CT scan was performed in 93%. ECG documentation of cardiac rhythm and details of cardiac history and examination were also available in all controls.

Asymptomatic infarction was defined as a cerebral infarct present on CT scan for which no corresponding symptoms were documented.

Results

Fifty-four patients with chronic (not paroxysmal) atrial fibrillation presented with cerebral ischemic symptoms. Of these, seven patients (13%) had cranial CT evidence of previous cerebral infarcts that had been asymptomatic. The seven included five men and two women ranging in age from 57 to 82 (mean 69) years. The cardiac disease associated with atrial fibrillation was ischemic heart disease (without evidence of congestive cardiac failure or a dyskinetic segment) in three and nonrheumatic mitral regurgitation in one; three of the seven had no identifiable coexistent cardiac disorder. Six of these seven patients had chest x-ray evidence of cardiac enlargement (cardiothoracic ratio of >0.5).

Four of the seven patients presented with severe stroke, two with mild stroke, and one with dementia. In two patients stroke syndromes were predated only by progressive cognitive disturbance, and CT scan suggested a diagnosis of multi-infarct dementia: both had four asymptomatic infarcts; one had bilateral frontal and bilateral large external capsular infarcts and the other had bilateral cerebellar, right frontal, and right parietal infarcts. Four of the seven patients had only one asymptomatic infarct, one a left frontal, one a right frontal, one a left cerebellar, and the fourth a large left external capsular infarct. In one of the seven patients there were two asymptomatic infarcts, right parietal and left occipital. All asymptomatic infarcts were peripheral and consistent with an embolic etiology.

The control group contained 168 persons, 96 men and 72 women. The mean age of this group was 69 years. Seven controls (4%) had CT evidence of cerebral infarction that had been clinically silent. Three of these seven controls had only small deep asymptomatic lacunar infarcts (<15 mm diameter): pontine and internal capsular in one, in another a left caudate nuclear infarct, and in the third a brainstem infarct and an infarct adjacent to the

anterior horn of the lateral ventricle. Three of these seven controls had both cortical and lacunar infarcts: bilateral frontal and left caudate nuclear in one, right posterior parietal and right thalamic in a second, and bilateral occipital infarcts and multiple periventricular lacunar infarcts in the third. One of these seven controls had an asymptomatic right parietal infarct. Five of these seven were hypertensive. If the controls with only lacunar infarcts are excluded (as the mechanism is almost certainly not embolism), then the prevalence of asymptomatic infarction in the control group is 2.4%.

The prevalence of asymptomatic cerebral infarction in the atrial fibrillation group was significantly greater than for the control group ($p < 0.05$, χ^2 analysis).

Discussion

Our results show that some patients with atrial fibrillation who present with symptomatic cerebral ischemia have had previous asymptomatic cerebral infarction, significantly more frequently than persons in sinus rhythm. This finding is in keeping with previous studies that demonstrate an increased rate of stroke in patients with atrial fibrillation and reflects the fact that a proportion of all cerebral embolic events will be clinically silent. Many patients with atrial fibrillation who have cerebral embolism sustain major neurologic damage, but neurologically intact patients with atrial fibrillation with asymptomatic cerebral infarcts, if identified, may represent a therapeutically attractive subgroup for stroke prophylaxis with anticoagulants. However, the possible benefits of identifying and treating patients with atrial fibrillation and silent cerebral infarction depend on the predictive value of CT demonstration of asymptomatic cerebral infarction for occurrence of subsequent clinical stroke. As the cerebral infarct is asymptomatic, it cannot be stated with certainty whether the cerebral infarct occurred while the patient had atrial fibrillation, and in a number of our patients the duration of atrial fibrillation could not be clearly established.

Admittedly, there were 97 patients in the retrospective group with atrial fibrillation who were excluded because no CT scan was performed; this may have biased our sample. It should be noted that, at the time of our study, CT was not regarded by the general medical staff as a routine investigation in the management of patients with cerebral vascular disease.

Although hypertension (58% vs. 32%) and diabetes (9% vs. 5%) were significantly more common in the control group, this may reflect the fact that one study is retrospective and thus the details were not recorded rather than representing a true difference between the groups that would support the role of atrial fibrillation in the causation of the cerebral ischemia.

Our study is based on data obtained from a retrospective analysis and contains several meth-

odologic problems related to patient selection. However, it does suggest that asymptomatic cerebral infarction is more common in patients with atrial fibrillation than in a similar group of controls. A preliminary report from Petersen et al⁸ examining asymptomatic patients with atrial fibrillation records a more striking incidence of silent cerebral infarction. Further prospective evaluation of the frequency and natural history of asymptomatic cerebral infarction associated with atrial fibrillation is required to define the best policy of investigation and treatment of this group with respect to stroke prevention.

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KEY WORDS • atrial fibrillation • cerebral infarction • embolism