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Atrial Fibrillation, TIAs and Completed Strokes

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SUMMARY A retrospective survey of 1076 patients with completed strokes and 789 with transient ischaemic attacks (TIAs) revealed that 5.6% of those with completed strokes but only 1.6% of those with TIAs were in atrial fibrillation. TIAs in the presence of atrial fibrillation tended to last longer than 60 minutes except in individuals who had coexistent carotid disease that might have been the source of their attacks. It is suggested that emboli from the fibrillating atrium rarely cause brief TIAs, and more usually cause 'long' TIAs or completed strokes.

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ALTHOUGH THE ROLE OF ATRIAL FIBRILLATION as a risk factor for stroke has been well established by epidemiological studies,^{1,2} few details are available about the clinical features of ischaemic events occurring in patients with fibrillation. The present retrospective study was designed to assess the relative prevalence of atrial fibrillation in patients referred with transient ischaemic attacks or completed strokes, and to analyse the clinical features that might be particular to cardiac embolism from the fibrillating atrium.

Materials and Methods

Since 1967 all patients referred to JM with transient ischaemic attacks (TIA) or strokes have been recorded on special index cards. From these, all noted to be in atrial fibrillation at the time of referral were selected and their hospital case records reviewed for details. The ECG was reviewed to confirm the presence of atrial fibrillation and the notes were searched for demographic data and details of the clinical presentation. Since atrial fibrillation might be coincidental rather than causal in patients with cerebrovascular disease, the evidence of other causes obtained clinically or angiographically was also noted.

Results

Between 1967 and April 1983, 789 patients were referred with TIAs (recovering within 24 hours) 1,076 with completed strokes and 369 patients gave a history of both TIAs and more persistent attacks. Thirteen TIA patients were in atrial fibrillation (1.6%), as were 10 who had both TIAs and strokes (2.7%). Sixty stroke victims had atrial fibrillation (5.6%) (table 1). The difference between the TIA and stroke groups is highly significant ($p < .001$ chi square).

The small group with TIAs were considered separately. There were 11 males and 2 females aged between 57 and 77 years (mean age 62.7 yrs). Only 3 were digitalised at the time of occurrence of their TIAs. The mean pulse rate was 96 (72 to 160) and 2 had paroxysmal atrial fibrillation. One patient described amaurosis fugax with rather long attacks (15

minutes). Eight more also had symptoms in the carotid territory, 3 were vertebrobasilar and one had attacks in both territories. Interestingly only 4 had brief episodes lasting less than 60 minutes. Of the 9 with carotid territory events only 2 had brief attacks and these both had carotid atheroma from which their emboli might have come. This contrasts with our previously published experience that 56% of patients with TIAs in the carotid territory have attacks lasting less than 60 minutes.³ The aetiology of the atrial fibrillation where known was varied (2 thyrotoxicosis, one rheumatic, one hypertensive, one cor pulmonale).

Of the 10 patients with histories of both TIAs and completed strokes 5 were male and 5 female aged from 53 to 72 years (mean age 63.2 yrs). None were digitalised and the mean rate was 98. One patient had amaurosis fugax and a small retinal infarct with persistent visual field defect. Six more had hemisphere symptoms and 3 had both vertebrobasilar and carotid territory events. The fibrillation was due to ischaemic heart disease in 2. In one instance each it could be attributed to diphtheria, thyrotoxicosis or rheumatic carditis. Atheromatous stenosis of the appropriate carotid artery represented an alternative explanation for episodes in 3 cases.

Sixty patients had completed strokes associated with atrial fibrillation. Twenty-five female and 35 male subjects were aged between 32 and 76 years (mean age 59.9 years). Seventeen were already digitalised with an average rate in the whole group of 80. Twenty had rheumatic heart disease, 14 ischaemic heart disease. Six were thyrotoxic and one was thought to have atrial fibrillation due to hypertension. In 3 patients there was clinical and/or angiographic evidence of neck vessel disease.

The site of infarction judged by clinical and or CT scan evidence was determined. Thus most were in the middle cerebral territory but a striking number (15) had a posterior parietal or occipital lesion with for example a macular sparing hemianopia or an inferior quadrantanopia with crossed hemisensory inattention. Five had temporal lobe lesions. Only one had an anterior cerebral artery territory syndrome. Two developed multi-infarct dementia and six had brain stem strokes.

Comment

Clinical surveys have suggested that atrial fibrillation is present in some 10–20% of patients with completed strokes but is rare in accounts of large numbers of subjects with TIAs. Though clinical detection of

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TABLE 1 Prevalence of Atrial Fibrillation in Patients Referred with TIAs, Strokes or a History of Both Kinds of Event

	n	Atrial fibrillation	Percentage
Strokes	1076	60	5.6
TIAs	789	13	1.6
TIAs and strokes	369	10	2.7

possible cardiac sources of embolism underestimates their rate of discovery at autopsy,⁴ the identification of atrial fibrillation is simple and it is unlikely that many cases are missed.

Atrial fibrillation was found in 5.6% of cases of completed stroke and in 1.6% of patients with TIAs and 2.7% of those with both TIAs and completed events. Whilst selection factors clearly play a role it seems likely that there is a real difference in the prevalence of atrial fibrillation amongst individuals with TIAs and completed strokes. The cause of the atrial fibrillation was often unknown. As echocardiography was not carried out some valvular heart lesions were probably missed but it nevertheless seems likely that many were examples of lone fibrillation also found by Gautier and Morelot⁵ to be the commonest aetiology in current practice. As noted by others atrial fibrillation of any cause can be associated with presumed embolism.² Nine patients were thyrotoxic, 22 had rheumatic heart disease, and 16 ischaemic heart disease. One had diphtheric heart damage, another cor pulmonale. Only 20 of the whole group were digitalised (24%) and the pulse rate was at least 90 in 46% suggesting that an uncontrolled rate may be a factor in the genesis of embolism.

As well as the rarity of atrial fibrillation amongst TIA patients, it was noteworthy that brief attacks were less usual than in our survey of TIAs associated with carotid stenosis.³ When brief attacks in the carotid territory occurred in patients with atrial fibrillation there was commonly an alternative possible embolic

source in the neck vessels. It appears that the size of emboli emanating from the atrium are more likely to cause strokes than TIAs, and if they do cause TIAs these are likely to last hours not minutes.

The present evidence suggests that TIAs should not be too easily attributed to atrial fibrillation, especially if brief. Non-invasive investigation of the carotid vessels or digital subtraction angiography would seem indicated in these cases to detect alternative sources of embolism. In the case of completed strokes only 3 had evidence of a possible atheromatous source for their deficit (i.e. 5% compared with 30% of the TIA patients and 30% of those with both TIAs and strokes).

The sites of cerebral infarcts in association with atrial fibrillation were also of interest. The rarity of embolism into the anterior cerebral artery was confirmed. A striking number had signs of infarction in the posterior parietal and occipital areas.

The present findings suggest the concept that the nature of emboli affects their sequelae. Whilst the emboli from carotid stenosis commonly causes TIAs, but less frequently strokes,⁶ those from atrial fibrillation appear to cause longer TIAs, and more usually completed strokes.

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