CEREBRAL ISCHEMIC EVENTS of cardiac origin, secondary to embolism or to hemodynamic changes, have long been recognized. The association between embolism and bacterial endocarditis, myocardial infarction, rheumatic valve disease, cardiomypathies, atrial myxoma, atrial fibrillation and valve prostheses has been documented by extensive experience. M-mode and 2-dimensional echocardiography have aided the identification of possible sources of emboli previously difficult to diagnose: e.g. intracardiac thrombi, mitral valve prolapse (MVP). Echocardiographic findings appear to be of limited usefulness in the study of unselected stroke patients.

The relationship between MVP and cerebral ischemic events, first suspected on the basis of anecdotal observations, has been validated by a growing body of evidence. Bicuspid aortic valve, cardiomypathies, miscellaneous cardiac diseases, and atrial myxoma have been described in patients with cerebral ischemic events and have been suspected as sources of emboli. The likelihood of a causal relationship between cardiac abnormalities and cerebral ischemic events is presumably greater in selected cases without intra- and extra-cranial vascular lesions at angiographic examination and without risk factors of vascular involvement. The aim of this study was to analyze a series of patients submitted to angiographic examination in order to identify the possible cause of cerebral ischemia, in particular potential cardiac causes.

Patients and Methods

Eighty-eight consecutive patients with cerebral ischemic events in the carotid territory were studied. Between September 1981 to April 1982 there were 63 males and 25 females with a mean age of 43 years (range 14-68 years) admitted within 1 month of the cerebral event to the Division of Neurosurgery of Florence.

All patients were submitted to ipsilateral carotid angiography of the extra- and intra-cranial carotid territories and all but three of the patients underwent contralateral carotid angiography. The patients were assessed for risk factors such as hypertension, diabetes, coagulopathy and hyperlipemia.

On the basis of the angiographic findings the patients were allocated into two groups: Group A — patients with atherosclerotic cerebrovascular disease — and Group B — patients without atherosclerotic lesions or with vascular lesions other than atherosclerotic changes in the major intracranial branches interpreted as being due to emboli. Group A consisted of 27 patients (22 males and 5 females). Fifteen of the patients presented with TIA, 5 with RIND and 7 with mild stroke. None of them had a clinical history that might suggest cardiac disease as a possible source of cerebral embolism. Group B consisted of 61 patients (41 males and 20 females): 39 of these patients presented with TIA, 11 with RIND, 3 with a mild stroke and 8 (all of whom went to a reasonable recovery) with a major stroke. Every patient was subjected to standard M-mode and 2-dimensional echocardiography using a mechanical sector scanner (ATL Mark III). The recordings were carried out by two observers who had no knowledge of the angiographic diagnosis. The examination was done according to current methods, 2-dimensional echocardiograms being taken in the parasternal and apical views. MVP was diagnosed according to the criteria of Morganroth et al. Bicuspid aortic valve was diagnosed when the commissures were well delineated and only two cusps were clearly recorded.

Intraventricular thrombi were diagnosed according to the criteria of Asinger et al.
TABLE 1  Results of Echocardiography in Group A Patients

<table>
<thead>
<tr>
<th>Echocardiographic results</th>
<th>Males</th>
<th>Females</th>
<th>Total No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>11</td>
<td>—</td>
<td>11</td>
</tr>
<tr>
<td>Left ventricular wall asynergy</td>
<td>6</td>
<td>—</td>
<td>6</td>
</tr>
<tr>
<td>Aortic valve diffuse thickening</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>—</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Bicuspid aortic valve</td>
<td>1</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>—</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Technically unsatisfactory</td>
<td>1</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
<td>5</td>
<td>27</td>
</tr>
</tbody>
</table>

Results

In the 27 patients of Group A the echocardiographic examination was normal in 11 (41%), pathological in 15 (56%) and technically unsatisfactory in the remaining 1 (3%). Mitral valve prolapse was not found in any of these patients (table 1). The results of carotid angiography in this group are shown in table 2. All the angiographic lesions found were consistent with the side of the ischemic event. In 44% of cases contralateral stenosing or occlusive lesions were found in varying associations. Group B: In this group of 61 patients, the echocardiogram was normal in 25 (41%), pathological in 33 (54%) and technically unsatisfactory in the remaining 3 (5%). MVP was seen in 21 (34%) cases while in 12 (20%) other abnormalities were present (table 3). The 8 patients who recovered quickly from the acute stage of a major stroke at angiographic examination had an "embolic block." In the remaining 53 patients, angiographic studies revealed no lesion potentially responsible for the ischemic event although in 2 cases an angiographic pattern suggesting an embolization of distal branches of the intracranial cerebral circulation was found. On comparing the frequencies of the various echocardiographic abnormalities we found a significant prevalence of left ventricular wall motion abnormalities (asynergy) in Group A and Mitral Valve Prolapse in Group B (p < 0.01 in the chi square test). The patients with MVP were neither suffering from nor had a history of hypertension or diabetes. None had a history of cardiovascular trouble and only three had a lipid profile abnormality.

Discussion

It has been estimated that between 5% and 14% of the normal population have a MVP.1,7,13,21,24

Table 2  Angiographic Findings in Group A Patients (27 cases)

<table>
<thead>
<tr>
<th>Angiographic findings</th>
<th>No. of cases</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICA stenosis (neck)</td>
<td>13</td>
<td>48</td>
</tr>
<tr>
<td>ICA occlusion (neck)</td>
<td>9</td>
<td>33</td>
</tr>
<tr>
<td>ICA stenosis (siphon)</td>
<td>2</td>
<td>7.5</td>
</tr>
<tr>
<td>MCA stem occlusion</td>
<td>2</td>
<td>7.5</td>
</tr>
<tr>
<td>Multiple atheroma</td>
<td>1</td>
<td>4</td>
</tr>
</tbody>
</table>

12 patients (44%) presented contralateral stenotic or occlusive lesions.

ICA = internal carotid artery; MCA = middle cerebral artery.

The 27 patients of Group A had a recognizable atherosclerotic lesion demonstrated on angiography. MVP was not seen in this group. This could be due both to a lower incidence of MVP in the older age group and/or to a lower thromboembolic potential of MVP in older patients. The Framingham Study recently reported an incidence of MVP of only 3%-4% in two groups of men with the mean age of 40 ± 10 and 70 ± 10 years25 and in another study cerebrovascular events were not identified as occurring in the older patients with MVP.27

In group B, as mentioned earlier, 21 out of 61 patients had MVP. The possible mechanism whereby MVP can become a source of embolism depend upon the anatomical conformation of the recess that is created by the prolapse. Pathological studies have shown the presence of fibrin and platelets adhering to the myxomatous valve and there is a report of small thrombi adhering to the valve27 an associated coagulation defect had also been incriminated;26 both hypertrophic cardiomyopathy and bicuspid aortic valve have been suspected of being possible sources of emboli.3,14,15,21 In our study these pathological conditions were identified in both groups with similar frequency.

On our evidence echocardiographic examination would appear to be mandatory in young cerebral ischemic patients, without atherosclerotic lesions in the carotid territory at angiography, in order to identify a potential cardiac cause of the cerebral ischemic disturbance and establish a correct therapeutic approach.

References

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