

Clinical Reports

Internal carotid artery occlusion in a young female with mitral valve prolapse.

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Summary: A healthy 28 year old housewife presented with sudden right hemiplegia and aphasia. No predisposing factors could be ascertained apart from posterior cusp mitral valve prolapse on two dimensional echocardiography. Extensive investigations confirmed the presence of cerebral infarction and persistent occlusion of the left internal carotid artery near its origin.

Introduction

Mitral valve prolapse (MVP) is commonly noted in asymptomatic subjects, where it may be seen in 6% of females and a lesser percentage of males (Procacci *et al.*, 1976; Markiewicz *et al.*, 1976). It is also generally regarded as a benign condition, although several major complications are recognized. These include transient cerebral ischaemic episodes, strokes and visual disturbances in addition to various cardiac complications (Barnett *et al.*, 1976; Barnett, 1982; Sandok & Giuliani, 1982; Beton *et al.*, 1983; Kouvaras & Bacoulas, 1984). The association with embolic cerebrovascular disease was first reported by Barnett *et al.* (1976). In a further report the association was confirmed and it was suggested that this was more true in the younger patient with a stroke, where MVP was noted in 40% of a population of patients with stroke compared to 6.8% of an age-matched control group (Barnett *et al.*, 1980).

Occlusion of the internal carotid artery in a patient with MVP and a stroke has been reported previously in two patients. The first was in a 21 year old female, where the occlusion was at the origin of the vessel in the neck (Barnett *et al.*, 1980). The second was in a 55 year old man in whom the occlusion was at the siphon (Watson, 1979).

We report a case where the occlusion was near the origin of the internal carotid artery in a 28 year old female with MVP.

Case report

A 28 year old housewife suddenly fell to the ground with right-sided hemiplegia and aphasia. She had

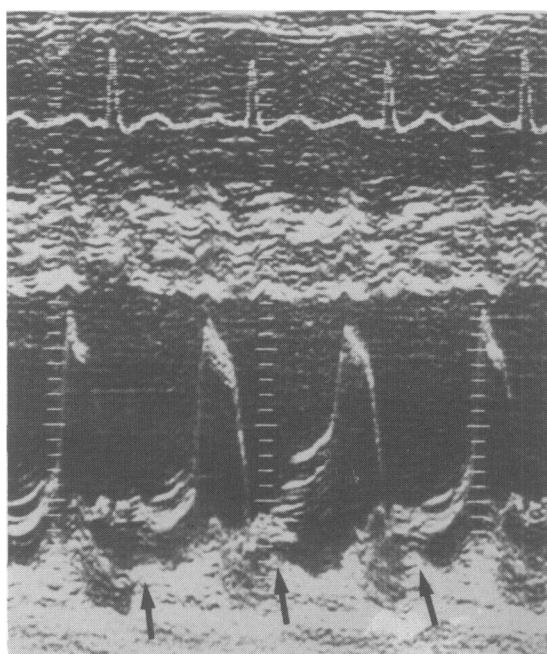


Figure 1 Two dimensional echocardiography showing pansystolic hammocking of the mitral valve.

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enjoyed good health previously with no previous spontaneous abortions or thrombotic episodes and had no known risk factor to date.

On examination she was found to have a global aphasia with a dense right hemiplegia. Cardiovascular examination was completely normal.

Investigations included normal coagulation and haematological, renal, mineral, hepatic, metabolic and lipid profiles. Urine examination, autoimmune antibody profile and anti-syphilis serology in both blood and cerebrospinal fluid were normal.

Head computed tomographic scanning showed a well defined non-enhancing low density area in the left fronto-temporoparietal region without shift consistent with infarction. Chest radiology, electrocardiography as well as 48 hours Holter monitoring were completely normal. Two dimensional echocardiography showed pansystolic hammocking of the mitral valve (Figure 1).

Bilateral carotid intra-arterial digital subtraction angiograms showed complete occlusion of the left internal carotid artery near its origin with good contrast filling on the right side (Figure 2). Intravenous digital subtraction angiography was done six months later and continued to show total occlusion

with no evidence of recanalization.

The patient showed little improvement in her clinical condition and remained severely disabled one year after her stroke.

Discussion

Cardiac embolism is an important source of stroke. It accounts for 14–16% of all cases of cerebral infarctions (Mohr *et al.*, 1978; Martin & Biller, 1984). MVP has been reported to be a possible cause particularly in the young stroke patient (Barnett *et al.*, 1976; Barnett *et al.*, 1980). MVP can be associated with supraventricular arrhythmias particularly atrial fibrillation. The latter even in the absence of other heart disease is associated with a high incidence of cerebral infarction (Sherman *et al.*, 1984). There was no evidence of a significant arrhythmia in our patient on prolonged monitoring with no evidence of other cardiac disease on chest radiography and two-dimensional echocardiography. This, however, does not exclude the possibility of a sudden dysrhythmia which may provoke embolization. The mechanism of the formation of a thrombus on the surface of a prolapsed valve in the absence of endocarditis is unclear. However, pathological changes at the margins of the prolapsed valve have been described (Pomerance, 1979), leading to platelet fibrin thrombus over the redundant leaflets. This, in association with paroxysmal atrial fibrillation, may be responsible for embolization.

Two dimensional echocardiography is highly sensitive in detecting MVP but is less sensitive in detecting a left atrial thrombus (Nichol & Kertesz, 1980; Knopman *et al.*, 1982). No evidence of thrombus was noted in our patient.

It has been reported that very few patients with MVP with primarily cardiac symptoms presenting to the cardiologist develop strokes compared to young patients with MVP and stroke presenting to the neurologist (Jones *et al.*, 1982). Our patient was asymptomatic prior to her ictus and her cardiac monitoring failed to show any significant abnormality.

Total internal carotid artery occlusion at its origin in a patient with MVP has been reported in one patient (Barnett *et al.*, 1980; Barnett, 1982). The sudden onset of the condition makes the diagnosis of an embolic phenomenon likely.

In another series of strokes in patients with MVP a case of internal carotid artery occlusion at the siphon was reported (Watson, 1979). The patient was over 50 years of age which makes the possibility of an atherosclerotic lesion more likely.

Occlusion of the internal carotid artery in young patients with stroke without MVP has also been reported (Barnett, 1982). The angiograms when re-



Figure 2 Digital subtraction angiograms showing complete occlusion of the left internal carotid artery near its origin with good contrast filling on the right side.

peated showed recanalization of the obstructive lesion. This latter occurrence was not the case in our patient where a repeat angiogram six months after the ictus continued to show total occlusion. This suggests that the thrombus is firm and organized. Intracranial branch rather than major vessel occlusion is the recognized feature in patients with MVP and a stroke (Hanson *et al.*, 1980). Although certain authors have reported recurrent cerebral ischaemic events in their

patients with MVP (Barnett *et al.*, 1980) other workers denied the existence of second embolic events (Kouvaras & Bacoulas, 1984). The evidence thus is not conclusive. We therefore feel that antiplatelet drug prophylaxis is justified for patients who have had a stroke associated with MVP. In conclusion, this case may add weight to the tenuous evidence that a major vessel can be occluded in a young patient with an embolic stroke associated with mitral valve prolapse.

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