Strokes and Mitral Valve Prolapse
Coincidence or Relation?

George Kouvaras, M.D., F.I.C.A. and George Bacoulas, M.D.

SUMMARY

Four cases with cerebral ischaemic events and mitral valve prolapse are described. Full investigations failed to reveal any other potential cause for these cerebral events.

Additional Indexing Words:
Strokes Mitral valve prolapse Cerebral ischaemic events

THE mitral valve leaflet prolapse syndrome may be a cause of endocarditis, paroxysmal or persistent arrhythmias and sudden death.\(^{5,15,23}\)

Four cases of cerebral ischaemic events were reported in 1974 to be associated with mitral valve leaflet prolapse.\(^{8}\) Although it is difficult to definitely link cerebral ischaemic events and mitral valve leaflet prolapse, the number of cases claiming an association between these phenomena has been steadily increasing.\(^{5,6,13,14,18}\) We describe 4 patients with cerebral ischaemic events where a mitral valve leaflet prolapse was the presumed etiological cause. Detailed investigations failed to detect any other potential cause.

CASE REPORT

Case 1

A 30-year-old manual worker developed two episodes of weakness of the right arm and right leg over a period of 4 months. The first episode lasted approximately 10 min, but the second lasted longer and was accompanied by dysarthria. The patient was admitted to hospital about 30 min after the onset of the last episode. Neurological examination showed moderate weakness and impaired movement of the right arm. The following day, the patient was asymptomatic and the signs from his arm had improved. Cardiovascular examination demonstrated a late systolic murmur in the mitral area and an intermittent mid-systolic click. The two-dimensional echocardiogram revealed prolapse of the anterior leaflet of the mitral valve. A full

From the Cardiology Department of the Public Hospital of Athens, Greece.
Address for reprint: G. Kouvaras, M.D., Spartis 127, TT 18546, Piraeus, Greece.
Received for publication February 15, 1985.
Table I. Diagnostic Examinations of Cerebral Ischaemic Events

1) Neurologic and cardiovascular examination.
2) Laboratory.
   CBC, platelet count, ESR, PT, PTT, fibrinogen, urinalysis, urea, glucose, serum,
   cholesterol triglycerides, antinuclear antibody, LE cells, protein electrophoresis,
   three blood cultures.
3) Chest X ray.
4) ECG.
5) CT scan.
6) Angiography of aortic arch and cerebral vessels.
7) ECG.
8) Echocardiogram.
9) Cardiac monitoring (48 h).

examination of the patient was normal (Table I). Dipyridamole and aspirin
were administered to the patient and 6 months after his discharge from
hospital he remains asymptomatic.

Case 2
A 32-year-old housewife was admitted because of the acute onset of left-
sided hemiplegia. She had never taken contraceptive pills and her medical
history was unremarkable. Neurological examination showed a left-sided
hemiplegia with left homonymous hemianopia. Cerebral angiography show-
ed a completely occluded right middle cerebral artery (Fig. 2).

Fig. 1. Echocardiogram of the second patient (parasternal long axis
view) showing prolapse of both cusps of the mitral valve. RV=right ven-
tricle; LA=left atrium; LV=left ventricle; MV=mitral valve.
Fig. 2. Cerebral angiography of the second patient. The right middle cerebral artery is occluded and is not opacified.

The aortic arch and the rest of the cerebral vessels were normal. A moderate sized right temporal infarct was seen on the CT scan. Cardiovascular examination revealed a mid-to-late systolic murmur at the cardiac apex. The two-dimensional echocardiogram showed prolapse of both leaflets of the mitral valve (Fig. 1). The rest of the investigations were normal. The patient remained in hospital for 20 days, her condition improved and 1 year after her discharge she has a residual left-sided hemiparesis. The patient has been maintained on aspirin and dipyridamole therapy since her discharge from hospital. She has also been receiving physiotherapy.

Case 3

A 32-year-old woman was admitted with acute onset of Wernicke’s aphasia and a right hemiparesis. She reported one previous episode of weakness and clumsiness of her right hand lasting for several minutes which occurred about 1 year prior to admission. Cardiac auscultation revealed a mild mid-systolic murmur and two-dimensional echocardiography disclosed prolapse of the posterior leaflet of the mitral valve. A CT-scan demonstrated a low-density area in the left posterior parietal cortex. No other abnormalities were found in the examination. This situation was resolved in 24 hours and the patient has been asymptomatic for 6 months after discharge. She has been receiving dipyridamole and aspirin.

Case 4

A 29-year-old salesgirl was admitted because of an onset of weakness of her right arm with dysarthria. These symptoms lasted about 15 min. Car-
diagnostic auscultation revealed a 2/6 mid-systolic murmur and two-dimensional echocardiography demonstrated prolapse of the posterior leaflet of the mitral valve. No other abnormalities were detected. The patient refused medication and she was discharged recently from hospital.

DISCUSSION

Mitral valve leaflet prolapse was recognized as a clinical cardiological syndrome in the early 1960s. Its incidence in the general population is between 5–10%, and it is considered one of the most common cardiac diseases. It has been reported to be familial. Although the long-term prognosis of the individual patient having the disease is uncertain, this condition is not considered benign because of its numerous complications. According to anatomical data the leaflets become voluminous and redundant and are characterized by myxomatous degeneration.

Several publications in the last decade have suggested there is an association between transient cerebral ischaemic episodes, visual disturbances and mitral valve leaflet prolapse.

Three mechanisms have been proposed for the production of cerebral ischaemic lesions in the mitral valve prolapse patient. First, thrombogenesis takes place at the region of the abnormal valve and the cerebral events result from a non-infective thromboembolism. Necropsy studies have reported torn endocardium in 35 cases with mucoid degenerating atrioventricular valves. The torn endocardium was overlying the abnormal valves and adherent fibrin and red blood cells were found in 10 of these cases.

Other pathological studies have demonstrated that fissures and thrombi develop on the myxomatous valve. Steele et al demonstrated that the platelet survival time is shortened in patients with mitral valve leaflet prolapse and thromboembolism. Reductions in platelet survival time makes one more prone to thromboembolism. A second possible mechanism is bacterial endocarditis may occur. Such cases have been reported.

A third possible mechanism involves disturbances of the cardiac rhythm, which is a well-known complication of mitral valve leaflet prolapse, but the ensuing neurological symptoms in these cases have to be diffuse, or in rare occasions focally located.

The most likely mechanism involved in our cases is non-infective thromboembolism because of the focal presentation of symptoms and angiographic findings. Subacute endocarditis was rejected as a potential cause because of the negative blood cultures and the recovery of the patients without administration of antibiotics. Arrhythmias should not be implicated because the
occurrence of focal neurological signs mitigates such a hemodynamic explanation. We cannot, of course, rule out the occurrence of paroxysmal atrial fibrillation which may be associated with thromboembolism, although the fact that our patients have never been aware of rhythm disturbances and the normal cardiac monitoring data argue against such a possibility.

Facts which suggest against the coincidental occurrence of cerebral ischaemic lesions and mitral valve leaflet prolapse but favour a cause-and-effect relationship in our cases are: A) Detailed and careful investigations failed to prove any other intracardiac or extracardiac potential causes. B) The young age of our patients and the angiographic findings reduce the likelihood of the preponderance of overall arteriosclerotic disease.

Barnett et al\textsuperscript{4} found a 30\% incidence of mitral valve leaflet prolapse in 60 patients younger than 45 years of age who suffered from cerebral ischaemic events for which no other cause was detected. Tharakan et al\textsuperscript{22} also found a 23.8\% incidence of mitral valve leaflet prolapse in 31 cases of stroke in young patients in which there were no other risk factors.

In our opinion, although the pathological corroboration would be the ideal proof that cerebral ischaemic events may be a complication of mitral valve leaflet prolapse, there is enough circumstantial evidence to suggest an association between the two.

Thus, cardiovascular examination should be of great importance in diagnostic evaluation of patients with cerebral ischaemic events and mitral valve leaflet prolapse should always be suspected and investigated.

\textbf{References}

5. Barnett HJM, Jones MW, Boughner DR: Cerebral ischaemic events associated with prolapsing mitral valve. Trans Am Neural Assoc \textbf{100}: 84, 1975
10. Davies MJ, Moore BP, Brainbridge MV: The floppy mitral valve. Study of incidence,
pathology and complications in surgical, necropsy and forensic material. Br Heart J 40: 368, 1978