Mitral Valve Prolapse and Cerebral Infarction

ROBERT G. HART AND J. DONALD EASTON

Establishing or excluding the presence of MVP in an individual patient is often difficult, as diagnostic standards are controversial. We have recently evaluated two young adults with unexplained cerebral infarction who illustrate this dilemma: one had definite auscultatory findings of MVP in the opinion of a senior cardiologist but had four M-mode and three 2-D echocardiograms that were normal; the other had no auscultatory findings, a normal 2-D echocardiogram, but MVP on M-mode echocardiography.

Auscultatory findings are absent in about 20% of patients with M-mode echocardiographic evidence of MVP. However, MVP has been reported in 6 to 21% of asymptomatic females using M-mode echocardiographic testing, emphasizing the variation in diagnostic criteria (criteria vary from 2mm to 5mm posterior displacement). Angiocardiographic diagnosis of MVP is also poorly quantifiable with no standardized criteria being used. Recently 2-D echocardiography has been proposed as the diagnostic standard, but it has not been generally confirmed as such due to variations in technical expertise.

Notably, most reports of MVP-related cerebral ischemia do not specify the diagnostic criteria used. At present, the aggregate data from careful auscultation by expert physicians and M-mode and 2-D echocardiography are most useful. The presence of typical auscultatory findings and clear M-mode echocardiographic evidence would seem to make the diagnosis definite. The presence of only auscultatory or echocardiographic evidence should be considered probable MVP. The ultimate role of 2-D echocardiography awaits further data.

Routine echocardiography is not indicated in unselected, older stroke patients, in whom cerebrovascular atherosclerosis is the likely mechanism even in the presence of MVP. Even if one assumes a similar incidence of MVP-associated cerebral ischemia for all age groups, it is calculated that only 1% of cerebral infarctions would be associated with MVP in unselected patients, explaining the low yield of routine echocardiography in such patients. If no etiology for cerebral ischemia is found after a search for common causes, echocardiography seeking MVP may be warranted, although the relationship of MVP to ischemia should still be regarded with some skepticism.

There are currently no reliable clinical, laboratory or echocardiographic indicators to distinguish which people with MVP are prone to stroke. A relationship...
between MVP-associated cerebral ischemia and migraine has been noted in a small number of patients. Platelet abnormalities have been reported in MVP patients with and without cerebral ischemic symptoms, as well as in young stroke patients without MVP. A linked mesenchymal dysplasia involving platelets and collagen of the mitral valve has been postulated.

An embolic mechanism for the cause of MVP-associated stroke is supported by angiographic evidence. Valvular thrombi forming on the abnormal valves presumably constitute the typical embolus. Two-dimensional echocardiography in patients with MVP-associated cerebral ischemia has visualized valvular lesions that suggest small thrombi and histologically verified thrombus has been found attached to the valve. However, MVP-associated atrial fibrillation could cause left atrial clot as a source of larger emboli. Further, it is possible that MVP is only a marker of, and not the cause of, hyperaggregable platelets.

The treatment of patients with MVP-associated cerebral ischemia has been largely empiric. Recurrent events have been reported, often separated by years, but the natural history is uncertain. The strategy of initial therapy with platelet antiaggregation agents as outlined by Barnett and Sandok and Giuliani seems reasonable pending further information.

Robert G. Hart, M.D.
The Comprehensive Stroke Center of Oregon, The Oregon Health Sciences University Portland, Oregon.
J. Donald Easton, M.D.
Department of Neurology University of Missouri, Health Sciences Center, Columbia, Missouri 65212

References
Mitral valve prolapse and cerebral infarction.
R G Hart and J D Easton

Stroke. 1982;13:429-430
doi: 10.1161/01.STR.13.4.429

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/13/4/429.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/