Mitral valve prolapse and visual loss

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Abstract

**Purpose:** To evaluate the correlation between mitral valve prolapse and permanent or temporary visual loss in young patients.

**Materials and methods:** Thirty two patients (age range, 16 to 41 years) with permanent or temporary visual loss were included in this study. Eye examination, including fluorescein angiography, and general medical examination, including echocardiography, were performed.

**Results:** Eight patients had central vein occlusion, 15 had branch retinal vein occlusion, and no pathology was found for 9. Seven patients showed mitral valve prolapse on echocardiography.

**Conclusion:** Mitral valve prolapse is thought to be one of the most important reason for the clinical appearance of permanent or temporary (amaurosis fugax) visual loss in young patients.

**Key words:** Mitral valve prolapse, Visual loss

Introduction

Mitral valve prolapse (MVP) is a retrograde displacement of one or more of the mitral valve leaflets into the left atrium during systole. Although various causes of MVP syndrome have been documented, including connective tissue disorders, rheumatic and congenital heart disease, cardiomyopathy, and coronary artery disease, the cause is unknown for the majority of patients. Mitral valve prolapse, which is variously termed the systolic click murmur syndrome, Barlow’s syndrome, floppy valve syndrome, or mitral leaflet syndrome, is more common among females and has been noted in a wide range of ages, although it is most commonly seen between the ages of 14 and 30 years. Most patients are asymptomatic and remain so for their entire lives. Electrocardiography is generally normal. Echocardiography and angiocardiology are important diagnostic methods.

One of the important complications of MVP is thromboembolism, which may lead to cerebral ischemic attack and visual disturbance. Amaurosis fugax, retinal vessel occlusion, and choroidal arteriolar occlusions have been reported in patients with MVP.

Materials and methods

Thirty two patients who were admitted to the ophthalmology department at the Cerrahpasa Medical Faculty complaining of temporary or permanent visual loss were included in this study. All patients underwent ophthalmic examination, including Goldman perimetry and fundus fluorescein angiography. In addition, complete blood count, VDRL, erythrocyte sedimentation rate, antinuclear antibody, rheumatoid factor, and plain chest X-ray were obtained for all patients. Cardiological consultation and echocardiography were also performed.

Results

The ages of the 32 patients (12 male and 20 female) ranged from 16 years to 41 years (median, 31.25 ± 3.30 years). Visual acuity ranged from counting fingers at 2 feet to 20/20 according to the Snellen chart. Rubeosis iridis secondary to central retinal vein occlusion was diagnosed for 2 patients; anterior segment examinations were normal for the remaining patients. Intraocular pressures ranged from 12 mm Hg to 18 mm Hg (mean, 16.32 ± 2.10 mm Hg) except for the 2 patients with rubeosis iridis. Nine patients had a normal fundus appearance, while 8 had central retinal vein occlusion and 15 had branch retinal vein occlusion. Patients with central and branch retinal vein occlusions had corresponding scotomas on Goldman perimetry. Seven
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The ages of these patients (2 male and 5 female) ranged from 16 to 31 years (median, 23.42 ± 4.90 years; Table 1). Visual acuity ranged from counting fingers at 2 feet to 20/20 according to the Snellen chart. One patient had central retinal vein occlusion and 2 patients had branch retinal vein occlusion. Four patients had a normal fundus appearance.

Discussion

In the differential diagnosis of temporary visual loss and retinal vessel occlusion seen in young patients, infective endocarditis and atrial fibrillation due to rheumatic valvular disease should be considered. Other rare causes of embolism are atrial myxomatous disease, anmiotic fluid, and fat emboli. Oral contraceptive use, blood dyscrasias, systemic lupus eryhematosus, polyarteritis nodosus, and rheumatoid arthritis should also be considered. Mitral valve prolapse progressing asymptomaticly may lead to visual complications. After excluding other causes of vascular occlusion, MVP was the only reason for the visual disturbances in this study. Wolof et al were the first authors to report the relationship between MVP and retinal vascular thrombosis in 2 patients.9 Caltrider et al10 and Hopkins et al11 reported that emboli secondary to MVP may lead to retinal occlusive diseases. Butt et al reported the relationship between retinal arterial occlusion and MVP in Marfan’s syndrome.12 Schimkat et al emphasized that hyperagglutination of platelets may lead to retinal vessel occlusion in their study of 5 patients with MVP.13 Van Rhee et al reported a MVP patient with bilateral retinal arterial occlusion.14 Greven et al diagnosed MVP with transesophageal echocardiography in patients with bilateral retinal arterial occlusion that could not be diagnosed by superficial echocardiography.15

Asymptomatic MVP is most easily diagnosed with echocardiography. In their study, Dorsee et al reported 7 patients with MVP among 101 healthy males.16 In another study, 10% of healthy women had MVP.17 As mentioned above, the female to male ratio was 5:2 in our study. Wilhelm reported 7 of 10 patients with ischemic ophthalmic disease and the remainder had amaurosis fugax.18 Mitral valve prolapse was the only risk factor that was indentified. Similarly, we did not find any other cause for retinal vein occlusion and temporary visual loss.

After detecting visual disturbances due to embolism in 2 of 10 MVP patients with acute cerebral ischemic attacks, Vallat et al pointed out the importance of a full cardiovascular evaluation and the necessity of echocardiography as part of the routine examination for these patients.19 In a study of 15 patients with central retinal vein occlusion younger than 50 years, Gonder et al reported that 11 patients had thrombocyte dysfunction and 7 had an echographically demonstrated MVP.20 In our study, 7 patients (21.8%) younger than 45 years had MVP determined by echocardiography.

In the light of these evaluations, it seems best to consider MVP in the differential diagnosis of young patients with temporary visual loss (amaurosis fugax) or visual loss due to retinal artery or deep vein thrombosis and to prescribe acetyl salicylic acid in relation to the platelet hyperactivity.

References

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