

Visual Field Examination During Transient Migrainous Visual Loss

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A transient episode of bilateral amaurosis fugax, or transient visual loss, occurred in a patient with ophthalmic migraine associated with mitral valve prolapse while computerized visual field testing was performed. This fortunate finding illustrated characteristic defects which are compared with the patient's visual field tested 24 hours later. Statistical analysis of both examinations is stressed here.

Key Words: Ophthalmic migraine—Mitral valve prolapse—Computerized visual field—Transient visual loss—Amaurosis fugax.

CASE REPORT

A 40-year-old woman was referred to our Neuro-ophthalmology Unit suffering episodes of transient visual loss. She had a history of three previous episodes in the previous year, the first one referred to as darkness in the right eye and two others described as a simultaneous concentric reduction of the visual field in both eyes. Visual symptoms lasted for about 15–20 minutes and cleared when a frontal migraine followed. Headaches also had occurred without visual aura a number of times in the previous 5 years.

Ophthalmological examination revealed 20/20 visual acuity in both eyes. The pupils were equal and reactive. Ocular movements were normal as were the slit lamp and fundus examination. Goldman perimetry showed only enlarged blind spots (Fig. 1). Ocular tension was 12 in the right eye and 13 in the left eye.

Mitral valve prolapse was detected by bidimensional echocardiography. Clinical and neurological exams were otherwise normal.

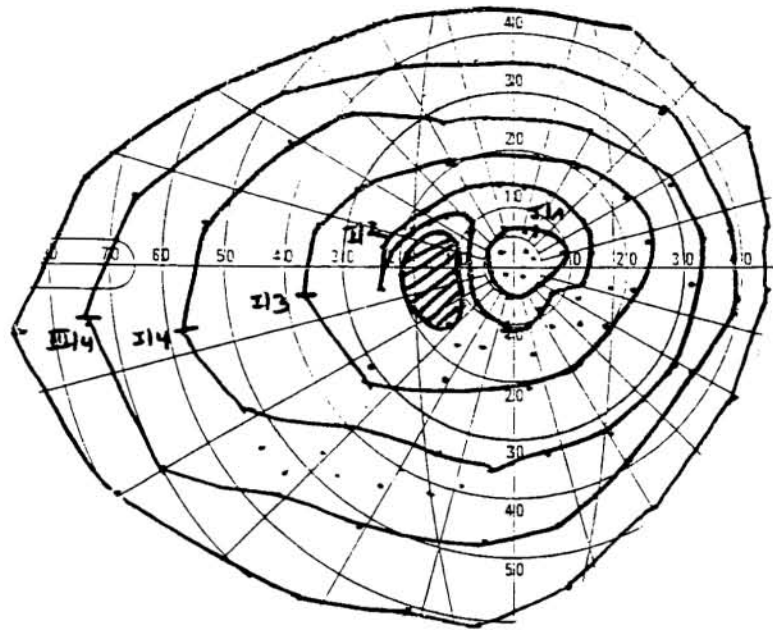
During routine visual field testing 4 months after the first perimetry, bilateral blurring of vision started. Computerized visual field with a central 30° screening strategy was performed, this time with a DIGILAB-750 Automated Perimeter. The visual symptoms lasted for about 30 minutes and allowed the program to be completed on both eyes before they cleared (Fig. 2).

With this program, a general-dotted depression was noted sparing the central 5°. Mountains and valleys occurring in the island of vision during the visual blurring were irregular in shape but similar on both eyes, having a homonymous type distribution. A mild frontal headache followed. The visual field cleared 24 hours later, as seen on type of examination (Fig. 3). Enlargement of the blind spot was still noted in both eyes and the borders of the central 30° were not completely normal.

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FIG. 1. Goldman perimetry obtained 4 months previously. Note bilateral enlargement of blindspot.



DISCUSSION

Monocular or binocular transient visual loss in migraineurs with mitral valve prolapse is a known association occurring usually before age 45–50 (1–5). A variety of visual symptoms are described: gray-/blackouts, seeing dark, through fog/rain or water, fortress-shaped scotoma, general constriction of visual field, etc. (6–9). These events can occur in either eye or in both simultaneously; they

can also occur in a different eye each episode. Visual symptoms can also be a positive phenomenon as scintillations, phosphenes, etc. Because these episodes are transient, they can seldom be plotted on visual field examination. The risk of stroke has been mentioned (3,6) in those cases where a permanent visual field impairment occurs. In this case, the patient's description of constriction of her visual field correlates with the collapse observed around the central 5°. The blurring cleared 24

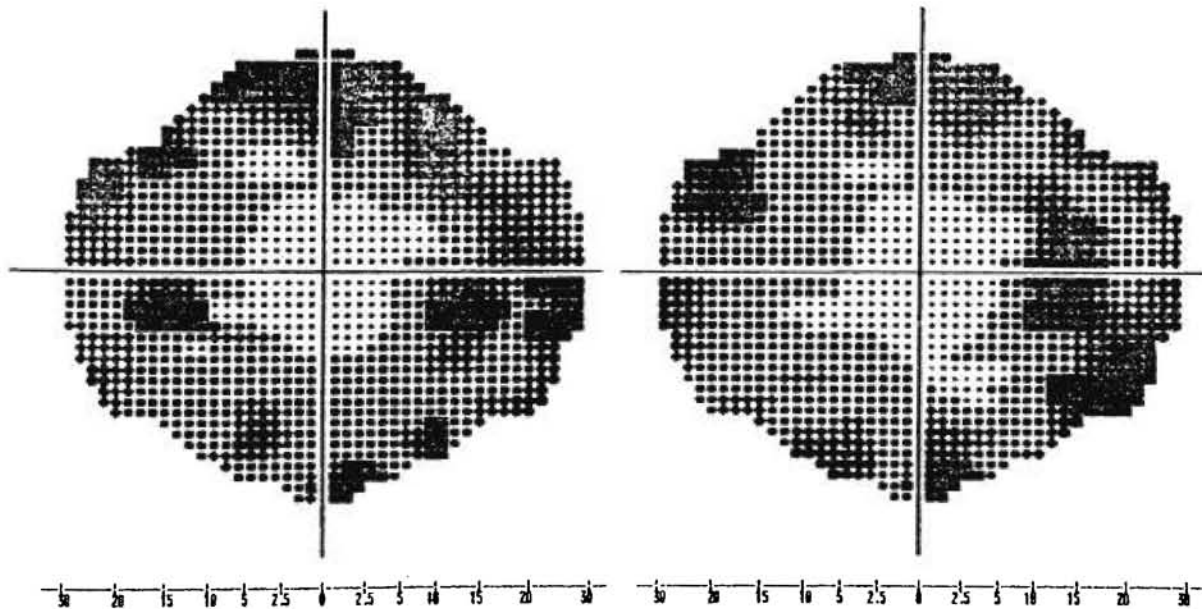


FIG. 2. Computerized visual field (central-30° screening strategy) performed during the visual obscuration episode. Note concentric-dotted depression around central 5° on both eyes. Defects adopt a homonymous distribution.

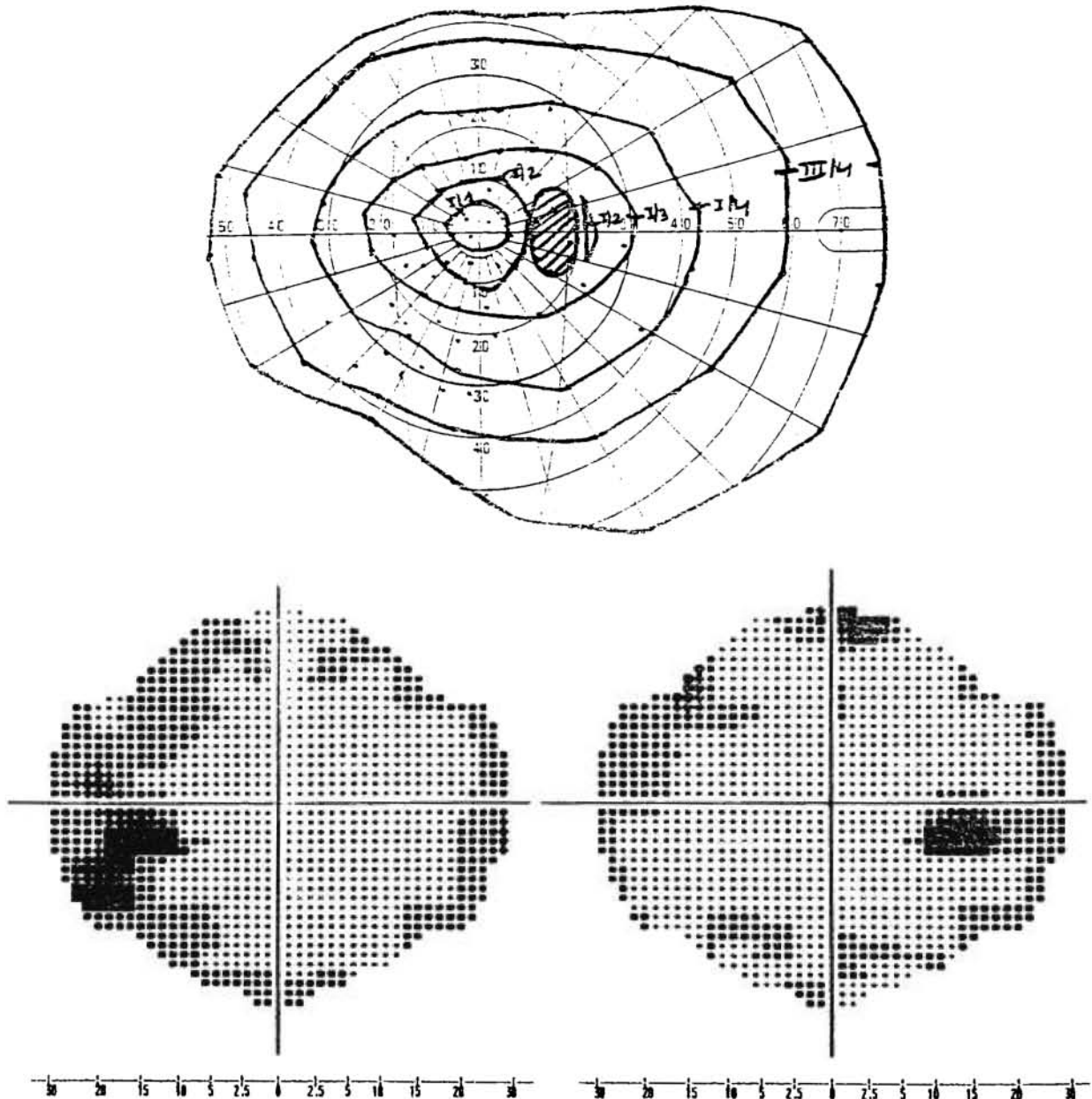


FIG. 3. Computerized visual field (central-30° screening strategy) obtained 24 hours later. Previous defects cleared (see Fig. 2); blindspots remain enlarged.

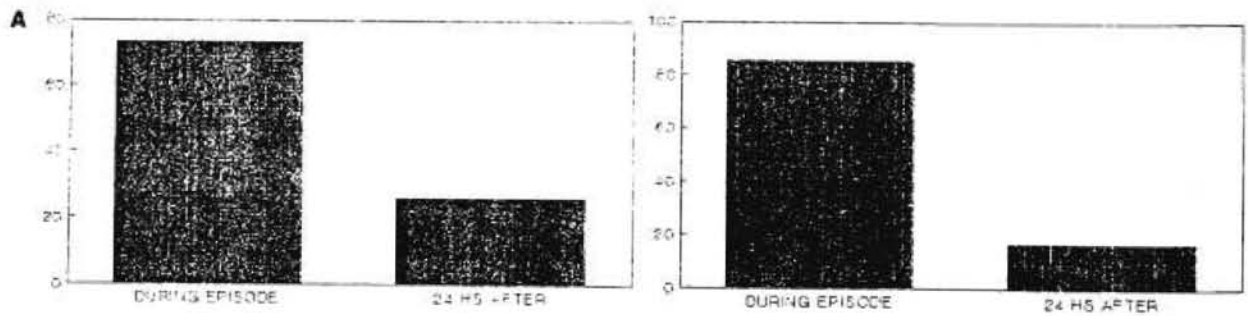


FIG. 4. Graphics of right (up) (A) and left (down) (B) eyes Defect Score (DS). Being a product of Standard Deviation Loss Variance (SD) LV and Mean Defect (MD), DS captures the values of loss distribution. The average loss is a more sensitive indicator of total loss. $DS = (SD) LV \times MD = dB^2$.

TABLE 1. Statistical matching of data obtained from visual fields performed during and after the obscuration (VISISTAT statistics program).

	RE		LE	
	a	b	a	b
Mean sensitivity (db)	12.0	17.5	11.5	17.4
Mean defect (db)	14.0	8.49	14.4	8.55
Loss variance SD (db)	5.234	3.063	5.911	1.958
Loss variance (db*db)	27.40	9.383	34.94	3.839
Defect score (db*db)	73.34	26.02	85.49	16.76

^a, per-episode data; ^b, 24 hours later. (See details in text.)

hours later even though the borders of the central 30° were not as clear as expected. A big blind spot was present before, during and after the event. This finding was mentioned by Tipping et al. (6). In our series mitral valve examining prolapse with visual symptoms (unpublished data), big blind spot constitutes the commonest finding on visual field examination (Fig. 4). Tipping et al. (6) mention the need for more than 24 hours for visual fields to clear in some cases. This might explain the irregular borders of the 30° study in the postepisode test.

Statistical data are shown in Table 1. The degree of loss is reflected as mean sensitivity and mean defect indices and distribution of defects is indicated by loss variance. The defect score, which combines mean defect and loss variance, is the main indicator of total loss.

Amaurosis fugax is the illustrative term to describe most of these phenomena. Much has been stressed in the literature to separate amaurosis fugax occurring in one eye due to carotid disease from transient visual events of other origins. The intrinsic meaning of the words "amaurosis fugax" can hardly be restricted to one etiology (10-13). Regardless of the terminology, visual symptoms

associated with mitral valve prolapse have been termed amaurosis fugax (1,2,6). Our case, whatever the name of the transient phenomenon, was a fortunate and useful increase in our knowledge of amaurosis fugax.

REFERENCES

1. Lesser RL, Heinmann MH, Borkowski H Jr, Cohen LS. Mitral valve prolapse and amaurosis fugax. In Smith JL, ed. *Neuro-ophthalmology focus 1982*. New York: Masson, 1981.
2. Barnett HJ, Boughner DR, Taylor DW, Cooper PE, Kostuk WJ, Nichol PM. Further evidence relating mitral-valve prolapse to cerebral ischemic events. *N Engl J Med* 1980;302:139-44.
3. Caltrider ND, Irvine AR, Kline HJ, Rosenblatt A. Retinal emboli in patients with mitral valve prolapse. *Am J Ophthalmol* 1980;90:534-9.
4. Grosogeat Y. Cerebral ischemic accidents of cardiac origin. *Neuroradiology* 1985;27:579-82.
5. Bugousslavsky J, Hachinski VC, Barnett HJM. Causes cardiaques et arterielles de cecite monoculaire transitoire. *Rev Neurol (Paris)* 1985;141:774-9.
6. Tippin J, Corbett JJ, Kerber RE, Thompson HS. Amaurosis fugax and ocular infarction in adolescents and young adults. *Ann Neurol* 1989;26:69-77.
7. Hupp SL, Kline LB, Corbett JJ. Visual disturbances of migraine. *Surv Ophthalmol* 1989;33:221-36.
8. Hoyt WF. Ocular symptoms and signs. In: Wylie EJ, Ehrnfeld WK, eds. *Extracranial occlusive cerebrovascular disease: diagnosis and management*. Philadelphia: W.B. Saunders, 1979:36-88.
9. Glaser JS. *Neuro-ophthalmology*. Hagerstown: Harper & Row, 1978:67-70.
10. Muci-Mendoza R. Occlusive vascular disease of the retina and optic nerve. *Curr Opin Ophthalmol* 1990;1:435-9.
11. Miller FC. Transient monocular blindness versus "amaurosis fugax". *Neurology* 1989;39:1622-3.
12. Sandok BA, Trautmann JC, Ramirez-Lessepas M, Sundt TM, Houser OW. *Am J Ophthalmol* 1974;79:137-42.
13. Hurwitz BJ, Heyman A, Wilkinson WE, Haynes CS, Utley CM. Comparison of amaurosis fugax and transient cerebral ischemia: a prospective clinical and arteriographic study. *Ann Neurol* 1985;18:698-704.
14. Halgliger IO, Flammer J. Increase of the short-term fluctuation of the differential light threshold around a physiologic scotoma. *Am J Ophthalmol* 1989;107:417-20.