Sir,

Retinal arterial macroaneurysm at the site of a retinal artery embolus

Retinal arterial macroaneurysms represent a distinct clinical entity arising from a main retinal arteriole within the first three orders of bifurcation.[1] They are seen in the elderly and rarely before the age of 60 years with a marked female predominance. There is a strong association with hypertension and generalised arteriosclerotic vascular disease, with up to 75% of patients being hypertensive.[2]

Retinal arterial macroaneurysms may exhibit exudative or haemorrhagic activity. The exudative process can cause oedema, lipid deposition, and serous neurosensory retinal detachment. Approximately 50% of patients have an associated retinal haemorrhage, typically one disc diameter in size surrounding the macroaneurysm. Subretinal, preretinal or vitreous haemorrhage may be seen in association with intraretinal haemorrhage.[2,3] If there is macular involvement, patients may complain of insidious or sudden-onset decreased vision.[2,3]

This report describes the fundal findings in a 20-year-old patient with documented retinal artery macroaneurysm and retinal artery embolus in one eye with subsequent development of another retinal artery macroaneurysm at the same location of the embolus in the same eye. The medical history of this patient was previously reported in part.[4]

Case report

A 20-year-old man was initially seen in December 1991. He complained of sudden loss of vision in the right eye of 3 days' duration. He had no history of trauma to the eye, nor did he have any systemic complaints. The patient reported a similar episode due to intraocular haemorrhage in the same eye 3 years earlier, followed by gradual restoration of vision. Visual acuity in the right eye was counting fingers at 1 m; vision in the left eye was 20/20. Intraocular pressure and slit-lamp examination of the anterior segment were normal in both eyes.

The left eye was ophthalmoscopically normal. Ophthalmoscopy of the right eye disclosed a normal optic disc. There was evidence of haemorrhage under the internal limiting membrane in the macular region with a horizontal fluid level. Another boat-shaped area of subhyaloid haemorrhage was noted inferiorly. Along the lower temporal arteriole there was a large aneurysmal dilatation close to the inferior edge of the optic disc and there was a focal area of periarterial sheathing distal to the macroaneurysm. A small embolus was noted at the site of the second bifurcation of the upper temporal arteriole (Fig. 1). On fluorescein angiography, the haemorrhage obscured choroidal fluorescence. The aneurysmal dilatation filled in the early arterial phase and became hyperfluorescent. In late views, there was minimal leakage of dye (Fig. 2). The patient was studied for systemic diseases. Cardiovascular evaluation revealed normal blood pressure with an ejection systolic murmur over the pulmonary area. Echocardiographic findings were consistent with congenital pulmonary stenosis and an atrial septal defect.

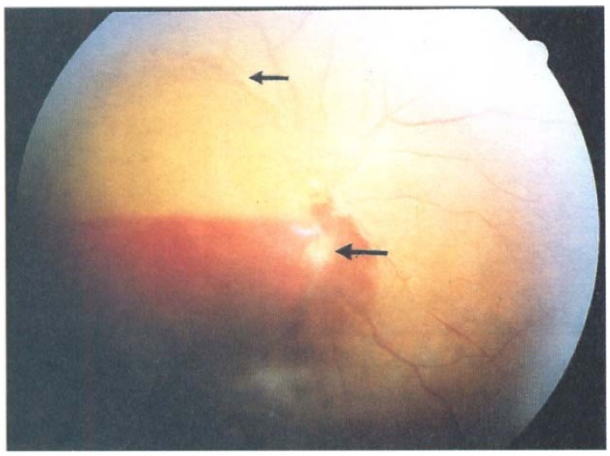

Fig. 1. *Initial examination in December 1991. Note the preretinal haemorrhage with a fluid level. The macroaneurysm at the level of the lower temporal arteriole is seen through the haemorrhage (large arrow). Note the presence of an embolus at the site of the second bifurcation of the upper temporal arteriole (small arrow). Reproduced with permission from Abu El-Asrar* et al.[4]

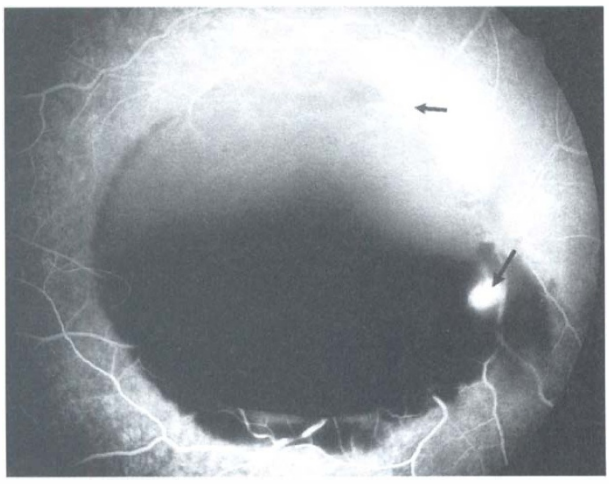

Fig. 2. *Arteriovenous phase of the fluorescein angiogram demonstrates the macroaneurysm as a hyperfluorescent area (large arrow). The hypofluorescent area is due to the preretinal haemorrhage. The embolus at the site of the second bifurcation of the upper temporal arteriole is delineated (small arrow). Reproduced with permission from Abu El-Asrar* et al.[4]

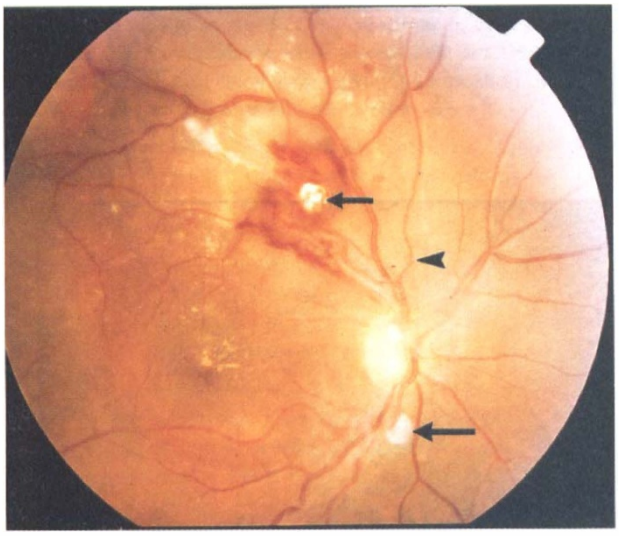

Fig. 3. *January 1999. Original aneurysm appears fibrosed (large arrow), and new fibrosed macroaneurysm is present at the previous site of the embolus (small arrow) surrounded by intraretinal haemorrhage and exudates with neurosurgery retinal detachment involving the macula. Note the presence of a new embolus (arrowhead).*

Four months later, the haemorrhage resorbed and visual acuity improved to 20/20. On ophthalmoscopy, the macroaneurysm became white and fibrosed, with a proximal area of periarterial sheathing. A follow-up fluorescein angiography revealed slow and incomplete filling of the aneurysmal dilatation, suggestive of partial obliteration of the lumen. Oral anticoagulant therapy with warfarin was initiated. The patient discontinued the treatment and was lost to follow-up.

In January 1999, the patient presented with the complaint of insidious diminution of vision in the same eye; visual acuity had decreased to 20/200. He was healthy and had no systemic complaints. Ophthalmoscopy revealed the development of a second arterial macroaneurysm at the same location as the embolus at the second bifurcation of the superotemporal retinal artery. This macroaneurysm was fibrosed and

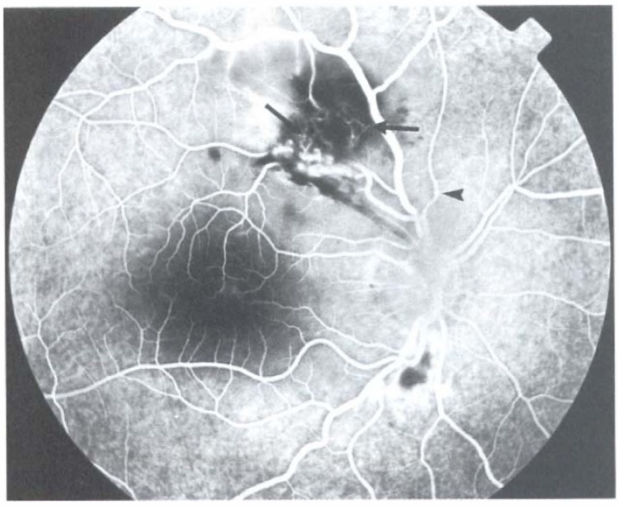

Fig. 4. *Arteriovenous phase of the fluorescein angiogram shows non-filling of the original and new aneurysms and small telangiectatic vessels surrounding the new aneurysm (arrows). The new embolus is delineated (arrowhead).*

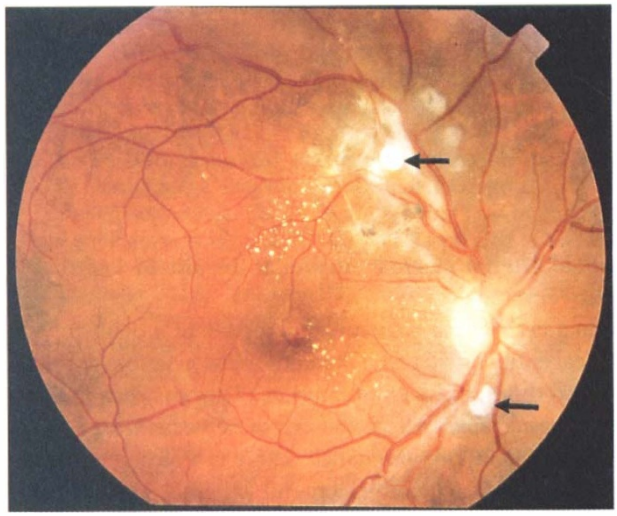

Fig. 5. *Six months after treatment. Note the resolution of haemorrhage and serous macular detachment. Fibrotic macroaneurysms are seen (arrows).*

surrounded by intraretinal haemorrhage. Hard exudates were scattered around the lesion with serous neurosensory retinal detachment involving the macula. There was a fibrous scar representing the old macroaneurysm along the lower temporal arteriole. In addition, a new small embolus was noted (Fig. 3). Fluorescein angiography disclosed non-filling of the old and new macroaneurysms and blocking of choroidal fluorescence by the retinal haemorrhage. The new macroaneurysm was surrounded by dilated telangiectatic capillaries that leaked dye in the later phases of angiography (Fig. 4).

The small incompetent vessels surrounding the new aneurysm were treated using the yellow wavelength (577 nm) laser. The laser parameters consisted of 500 μm spot size, 0.2 s duration and moderate intensity. The patient was started on warfarin as recommended by the cardiologist. Six months later, visual acuity in the right eye improved to 20/30. Ophthalmoscopy of the right fundus showed resolution of haemorrhage and serous macular detachment (Fig. 5).

Comment

This case showed several interesting features: (1) it demonstrated the occurrence of an arterial macroaneurysm in a young patient; (2) the point where the second macroaneurysm arose corresponded to the site of an embolus at the second bifurcation of the superotemporal retinal artery; (3) in the same eye, the first macroaneurysm that was located closer to the optic disc exhibited haemorrhagic activity, and the second macroaneurysm exhibited exudative activity. Lavin *et al.*[5] noted that haemorrhagic macroaneurysms were located significantly closer to the optic disc than other macroaneurysms. Arteries close to the disc have larger diameters and higher flow rates than peripheral vessels. These factors will increase transmural stress in these arteries, and may contribute to haemorrhage.

The precise pathogenesis of retinal arterial macroaneurysms is not well known and the vessel abnormalities that precede their development have rarely been demonstrated. Focal arterial wall damage is the likely precursor of retinal artery macroaneurysm. Macroaneurysm formation from the exact site of a previous retinal artery embolus has been described in only four cases including the present one.[6–8] It is postulated that local vessel wall damage from emboli predisposes to aneurysmal dilatation. The incidence of carotid atheromatous plaques in patients with retinal artery macroaneurysms was higher than that found in a similar asymptomatic hypertensive population, supporting the theory that retinal artery macroaneurysms may be of embolic origin.[9] Cardiovascular evaluation of the patient reported here revealed a congenital atrial septal defect; this may have caused retinal arterial embolic episodes, leading to focal arterial wall damage and development of macroaneurysms.

Most macroaneurysms exhibit vascular leakage resulting in retinal oedema, and the secondary accumulation of lipid exudate. The oedema may be due to direct aneurysmal leakage or leakage from small incompetent vessels surrounding the aneurysm.[2,10] These microvascular changes resemble the reorganisation of small vessel networks which have been shown in experimental embolic arterial occlusions.[11] Palestine *et al.*[10] recommended photocoagulation to the small, incompetent vessels surrounding the aneurysm (which contribute to retinal swelling). It is generally agreed that the visual prognosis is poorer in patients who experience visual loss from extensive macular oedema or exudate than in patients with decreased vision secondary to macular or vitreous haemorrhages.[2,3] In the case described here, resolution of haemorrhage, and serous macular detachment with improvement in visual acuity, followed yellow wavelength photocoagulation treatment of the leaky, telangiectatic blood vessels surrounding the macroaneurysm.

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Sir,

Ultrastructural analysis of opacities seen in a hydrophilic acrylic intraocular lens

Intraocular lens (IOL) optic opacities have been observed in foldable acrylic IOLs. In some patients they can cause glare, reduced visual acuity and contrast sensitivity. In this article we present an electron microscopic study of opacities in an explanted hydrophilic acrylic IOL.

Case report

A 67-year-old patient was referred to our department in December 1998 because of progressive visual loss in her left, pseudophakic eye. The cataract operation was performed in January 1998 in another clinic. According to the surgical report, the operation was uneventful. A one-piece acrylic IOL was implanted: SC60B-OUV (DGR Incorporated, St Petersburg, FL).

Slit lamp biomicroscopy of the left eye revealed a clear cornea and no inflammation in the anterior chamber. The IOL was in the capsular bag. The IOL optic was opaque due to many tiny whitish opacities. Both haptics were clear. Visual acuity (VA) was 20/400, IOP was 12 mmHg. The IOL was explanted, and exchanged for a PMMA IOL.

The IOL was analysed with a Philips PSEM 500 scanning electron microscope (Philips, Eindhoven, Holland), a Zeiss EM902 transmission electron microscope (Zeiss, Oberkochen, Germany) and a Hitachi H600 transmission electron microscope (Hitachi, Dusseldorf, Germany) equipped with a Kevex 7000 energy-dispersive X-ray microanalysis system (Getac, Mainz, Germany).

The IOL optic was opaque due to many tiny whitish opacities (Fig. 1). Both haptics were clear, as was the edge of the IOL optic. On scanning and transmission electron