RETINAL ARTERY OCCLUSION IN LOIASIS*†

BY

M. J. CORRIGAN AND D. W. HILL

*Moorfields Eye Hospital and The Department of Clinical Ophthalmology,
Institute of Ophthalmology, University of London*

Loa loa, the African eye worm, is a parasite belonging to the group of nematodes. The female worm is 3–6 cm. long, is viviparous and produces large numbers of microfilariae. These measure about 275 μ long by 7 μ in diameter; they invade the peripheral blood stream and are carried to distant sites. In this respect the condition of “*loa loa*” resembles Bancroftian and Malayan filariasis, but differs from onchocerciasis in which the microfilariae travel through the extracellular soft tissue spaces. All microfilariae share the common property of inducing allergy and sensitization (Gentilini, 1962).

Loa loa infestation is usually considered to be benign and scarcely ever leads to ocular damage (Ridley, 1963), but meningo-encephalitis is a well-known complication (Kivits, 1952; van Bogaert, Dubois, Janssens, Radermecker, Tverdy, and Wanson, 1955; Alajouanine, Castaigne, Lhermitte, and Cambier, 1959).

Case Report

A 26-year-old Nigerian student came to hospital with sudden loss of vision in the right eye of 4 hours' duration. Shortly after coming to England, 2 years previously, he had felt unwell and ran a low-grade fever. Microfilariae had been found in the peripheral blood, and he had been treated with a 4-week course of Hetrazan. He had had no other illness and had taken no other drugs.

Examination revealed marked retinal oedema similar to that seen in central retinal artery occlusion, although the large retinal arteries appeared of normal size and were not easily collapsed by digital pressure on the globe. Central vision was reduced to counting fingers, and a dense central scotoma was present, though the peripheral field was full (Fig. 1). A tentative diagnosis of central retinal artery occlusion was

FIG. 1.—Field of vision in the right eye recorded on the day of admission.

* Received for publication June 7, 1967.
† Address for reprints: as above.

made, and retrobulbar Priscol 50 mg. was given. Inhalations of a mixture of 95 per cent. oxygen and 5 per cent. carbon dioxide for an hour produced no improvement. He was admitted to hospital and further ocular examination revealed inactive nummular opacities at all depths of the corneal stroma, suggestive of old onchocercal keratitis. No microfilariae were seen in the cornea or anterior chamber. Ophthalmodynamometry recorded equal pressures in the central retinal arteries of both eyes.

A blood smear revealed frequent *Loa loa* microfilariae without eosinophilia. The erythrocyte sedimentation rate was 7 mm./hour, the Wassermann reaction and Kahn test were negative, and electrophoresis showed no abnormal haemoglobins. The blood pressure was 120/70 mm. Hg, the cardiovascular system was healthy, and no abnormality could be detected in the right carotid artery. A study of the tears did not show any microfilariae.

Fluorescence retinal photography was performed 4 days after the occlusion. Preliminary colour films showed extensive oedema of the retina with haemorrhages in the macular area (Fig. 2). The transit of dye through the right eye was only slightly delayed as compared to the left, but a striking abnormality was seen in the macular area (Fig. 3) where the vessels remained completely empty with sharp end-points to the filling. This area extended by about 3 disc diameters horizontally and 2 disc diameters vertically and coincided with the area in the colour photograph within which the vessels appeared darker and more sharply outlined. The left eye showed a normal transit of dye.

FIG. 2.—Reproduction of a colour transparency of the right retina, photographed 4 days after the onset of loss of vision. There is extensive oedema of the posterior pole, overlapping the disc, though the temporal margin is distinct. The opacity in the macular area is denser, some haemorrhages are seen, and the retinal vessels appear darker but continue to the full extent of their normal distribution.

FIG. 3.—Fluorescence retinal photograph, taken 4 days after the onset of loss of vision, showing the mid-phase of transit. The arterioles are filled and venous filling is beginning. Surrounding the macula there is a large area of capillary closure, bounded by the abrupt termination of fluorescence in the larger vessels.

A 2-week course of Bamazide was given and the microfilariae disappeared from the peripheral blood. The patient did not return for examination for 4 months, by which time the visual field had contracted to a small eccentric area (Fig. 4, opposite), while the visual acuity remained counting fingers. There was obvious optic atrophy with attenuated retinal vessels which were reduced to thin white cords in the macular area (Fig. 5, opposite).

The choroidal pattern showed no disturbance. Repeat fluorescence retinal photography revealed a greatly reduced flow with only the major vessels outlined; neither the smaller vessels nor those at the macula were displayed. There was no significant increase in transit time as compared to the left eye.

FIG. 4.—Field of vision of the right eye recorded 4 months after the onset of loss of vision.

FIG. 5.—Reproduction of a colour transparency of the right eye 4 months after the onset of loss of vision, showing extreme optic atrophy with attenuation of the retinal vessels, particularly the arterioles.

Comment

The appearances of the retinal vessels seen by fluorescence photography must represent multiple obstruction in the small arterioles, for the unfilled vessels are still visible in the colour film as dark columns. The phenomenon resembles that observed in experimental embolic occlusions in animals (Ashton, Dollery, Henkind, Hill, Paterson, Ramalho, and Shakib, 1966). Whether the closure was embolic or due to the pressure of oedema in the tissues is debatable, but the sharp edge to the area of capillary filling suggested embolism. The final state of generalized atrophy, arising from an initial picture of central retinal artery occlusion, is consistent with massive embolism of the retinal arterioles.

Loiasis is a well known cause of visual and cerebral vascular lesions, but involvement of the retinal vessels is a great rarity, only two other case reports being found in the literature.

Langlois, Perrouy, Daoulas, and Berton (1962) described a sailor who had loiasis for many years. He suddenly developed bilateral cerebellar, right-sided pyramidal and sensory tract signs, and a right central retinal artery occlusion. In the absence of any other disease, and with the presence of a marked eosinophilia, Langlois suggested microfilarial emboli as the common aetiology. Histological proof was not available.

Toussaint and Danis (1965) described the case of a 38-year-old male who had spent some time in the Congo and had contracted loiasis. He was admitted to hospital in a comatose state, and numerous *Loa loa* microfilariae were found in the peripheral blood and cerebrospinal fluid. The fundus showed retinal oedema and large superficial haemorrhagic sheets of variable form disseminated throughout the retina alternating with areas of yellowish exudates. There were also a few whitish filaments simulating obstructed arterioles.

Despite treatment with Hetrazan, antihistamines, and cortisone, the patient died on the seventh day after admission. Pathological studies revealed numerous vascular lesions in the cerebral cortex. Many capillaries were distended by microfilariae and surrounded by chronic inflammatory cells including several multinucleate giant cells of the foreign-body type. The eye showed retinal and subretinal oedema with a few microfilariae free in the subretinal fluid. The lumen of a large

number of vessels contained microfilariae, and some were ensheathed with lymphocytes. The choroid contained only an occasional microfilaria and the remainder of the eye was normal. A retinal digest preparation confirmed the presence of a large number of microfilariae within the vessels, although the distribution did not seem conditioned by anatomical or mechanical factors. The proximal part of the retinal vascular system had a normal appearance, whereas the distal vessels displayed a large number of bead-like micro-aneurysms and a greatly reduced number of mural cells. The lumen of many aneurysms was totally or partially occluded by coiled microfilariae.

The present report adds to the evidence of microfilarial embolization by a clinical demonstration of obstructed flow in the retinal vessels within a few days of the onset of loss of vision, thus supporting the pathological study already recorded.

Summary

A 26-year-old Nigerian presented with acute retinal ischaemia. *Loa loa* microfilariae were found in the bloodstream and fluorescence retinal photography suggested extensive embolic occlusion. Two previously recorded cases, one with full pathological studies, are reviewed.

We are indebted to Mr. C. A. G. Cook, under whose care the patient was treated, for his permission and encouragement to record this case.

REFERENCES

- ALAJOUANINE, T., CASTAIGNE, P., LHERMITTE, F., and CAMBIER, J. (1959). *Rev. Neurol.*, **101**, 656.
 ASHTON, N., DOLLERY, C. T., HENKIND, P., HILL, D. W., PATERSON, J. W., RAMALHO, P. S., and SHAKIB, M. (1966). *Brit. J. Ophthalm.*, **50**, 286.
 BOGAERT, L. VAN, DUBOIS, A., JANSSENS, P. G., RADERMECKER, J., TVERDY, G., and WANSON, M. (1955). *J. Neurol. Neurosurg. Psychiat.*, **18**, 103.
 GENTILINI, M. (1962). "Les filarioses pathogènes de l'homme". Arnette, Paris.
 KIVITS, M. (1952). *Ann. soc. belge Méd. trop.*, **32**, 235.
 LANGLOIS, M., PERROUTY, P., DAOULAS, R., and BERTON, M. (1962). *Rev. Neurol.*, **107**, 381.
 RIDLEY, H. (1963). In "Modern Ophthalmology", ed. A. Sorsby, vol. 2, p. 246. Butterworths, London.
 TOUSSAINT, D., and DANIS, P. (1965). *Arch. Ophthalm. (Chicago)*, **74**, 470.