RETINAL ARTERY EMBOLISM

By S. M. Chu

SYNOPSIS

A case of transient monocular blindness due probably to cholesterol emboli to the retinal artery is described. Though no source of emboli was demonstrated, the most likely source from atheromatous plagues in the internal carotid artery or its branches cannot be excluded. A brief review of the aetiology of the condition is given.

INTRODUCTION

Transient monocular blindness, or better known amaurosis fugax, is not an uncommon condition. It has been known as early as 1835 and referred to then as "periodic amaurosis" by Middlemore. Various diseases were believed to have caused this, among which were glaucoma, giant-cell arteritis, heart disease, migraine, hysteria and more rarely ergot poisoning, malaria, syphilis, and tobacco intoxication. For many years the interest shown by most workers was centred on the nature of the retinal circulation in cases of transient monocular blindness rather than on the underlying disease states. In 1864 Hughlings Jackson in attempting to explain an attack of blindness lasting five minutes in one of his patients postulated that attacks of transient blindness were due to retinal ischaemia from vasospasm. That sudden loss of vision could be attributed to embolism of the retinal artery was first observed by Fisher in 1959. He repeatedly examined the retina of a patient during a transient attack of monocular blindness and observed the passage of a white substance through the retinal arteries. The substance fragmented as it moved to the periphery and disappeared with the patient's recovery of vision. Russell (1961) described a similar finding in two patients with carotid artery disease and he thought that these substances were platelet aggregates. Further support of platelet embolisation was provided by McBrien *et al* (1963) when they studied a case of transient monocular blindness associated with carotid artery stenosis. Post mortem flat-sections were taken of the retinal vessels where emboli were impacted. They found that these emboli consisted of finely granular material which stained negatively for fibrin and positively for PAS, and appeared to consist of conglutination of platelets.

Hollenhorst (1961) described bright yellow refractile emboli in the retinal arterioles and believed these emboli were crystals of cholesterol arising from ulcerating atherosclerotic plagues. David *et al* (1963) were able to demonstrate cholesterol emboli to the retina at necropsy on one of their patients.

The following is a case report of a patient with recurrent transient monocular blindness due to the presence of refractile emboli.

CASE REPORT

The patient is a 50 year old Caucasian who had been in good health before. While watching television one evening, he suddenly noticed blurring of vision in the left eye. The next morning he noted he had total loss of vision in that eye. He was seen by a general practitioner by which time he had regained his vision. He developed several attacks of transient blindness within the same day, each episode of blindness lasting a few minutes. He noted that the left upper field was more blurred during the attacks. He was referred to the University Hospital for investigation on the same day. He had no other symptoms.

His father died probably of coronary heart disease and his mother had an attack of sudden blindness of one eye six years previously with no recovery of her sight.

On physical examination, he was a well built individual. His blood pressure measured 110/80 mmHg. and the pulse was regular with a rate of 80 per minute. All peripheral pulses were palpable and equal. Both carotids were easily felt and no bruit was heard. Examination of the heart revealed no abnormality. There were no neurological deficits and examination of the eyes showed both pupils to be equal and reactive to light. Visual acuity on the right was 6/18 and on the left it was markedly diminished; he could only count fingers. Fundoscopic examination revealed a normal disc on the

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left side, the macula appeared slightly oedematous and two refractile plugs were seen along the inferior branches of the retinal artery, one at the second bifurcation and the other more peripherally (Fig. 1). The calibre of the vessels were normal. The right fundus was normal. His left visual field was full at the periphery but a central field defect was present (Fig. 2). Laboratory investigations showed a Hb. of 15·1 gm./100 ml. and a platelet count of 177×10^3/cmm. The serum cholesterol was 320 mg.% and his blood Kahn test was negative. The electrocardiogram was normal. An arch aortogram done via the right femoral artery showed normal calibre of both carotids and vertebral arteries. A selective carotid angiogram was not done.

Repeated fundoscopic examination revealed fresh embolisation by these white refractile plugs along the course of the left inferior temporal branch of the retinal artery. During this period, and over the next few days, the visual loss of the left eye remained static.

On the day of admission the patient was commenced on heparin and warfarin therapy but this was stopped following the transfer of the patient to another centre at his request. The subsequent course of his illness was not known.

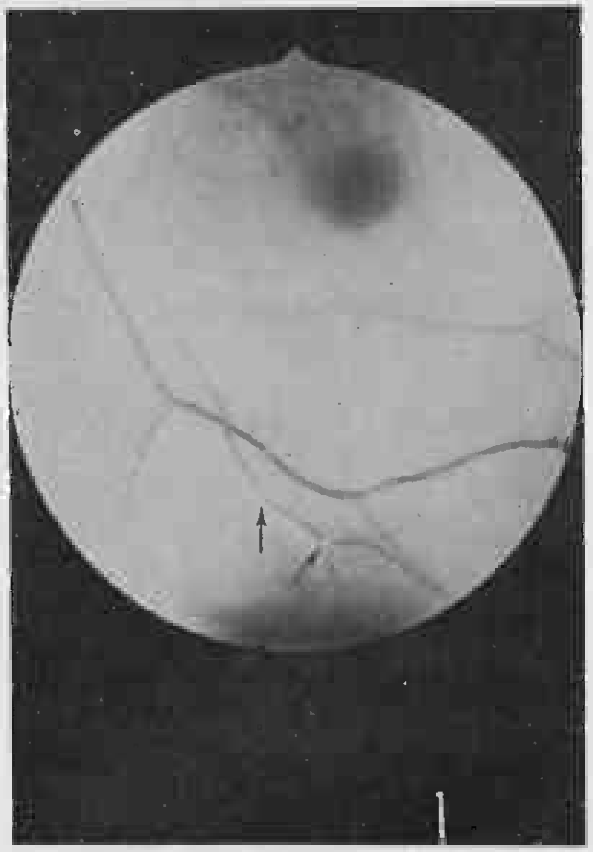

Fig. 1. Photograph of the left fundus showing the refractile emboli (arrows) in the inferior temporal branch of the retinal artery. Notice also oedema of the macula.

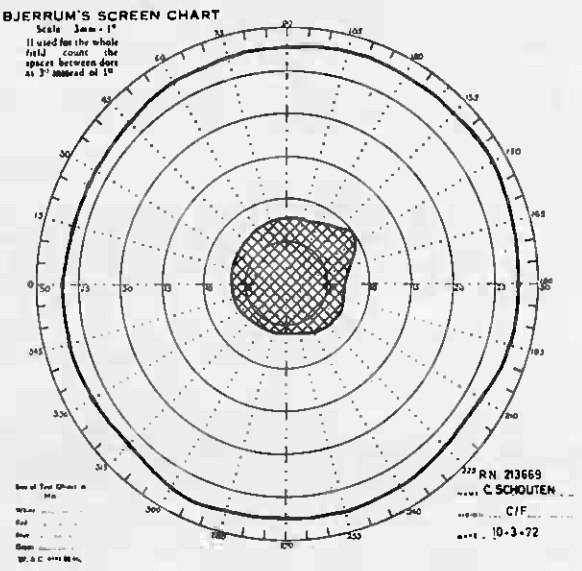

Fig. 2. Bjerrum's screen chart showing central scotoma and full peripheral field.

DISCUSSION

The association of transient monocular blindness and carotid artery disease had been well documented (Fisher, 1952), Hollenhorst (1960), Marshall *et al* (1968). The emboli observed in this patient who developed residual visual loss were refractile in nature. It was therefore very likely that these emboli were cholesterol crystals and could have arisen from atheromatous plagues in the carotid artery. Russell (1968) classified the types of emboli and their origin into three categories. The first variety, often associated with aortic valve stenosis, presents as a single white plug impacting in a retinal artery causing complete obstruction over a variable segment. The resulting field defect is usually permanent. The second variety consists of platelet-fibrin aggregates from mural thrombi in the carotid arteries. Since they traverse through the retinal circulation within a few minutes, they usually cause transient visual symptoms with no residual visual loss. The third variety and the commonest is usually derived from ulcerating atheromatous lesions in the carotid arteries, and multiple refractile cholesterol plugs cause embolisation of the smaller vessels, chiefly the branches on the temporal side of the discs. The symptoms are more variable in severity and frequency and may be associated with field defects. There are however some patients who have definite refractile emboli in the retinal vessels in

whom carotid artery lesions are not demonstrable. A few of them have hypertension or diabetes mellitus. The source of emboli in this group is uncertain. Although the aortogram in this patient did not show any lesion in the arch of the aorta, the origins of the carotids, the common carotid arteries or the proximal portion of the internal carotid arteries, this does not exclude totally the presence of atheromatous plagues in the carotids or the ophthalmic and central retinal arteries, and he would probably fit into the third category of Russell's classification.

The treatment of patients with transient ocular blindness is largely one of prevention of further embolisation. The use of anticoagulant therapy is of doubtful value (Marshall *et al*, 1968). Aspirin and dipyridamole (Persantin) can both impair platelet aggregation by inhibition of release of A.D.P. from stimulated platelets. Their use in the prevention of embolisation in cerebral vascular disease (Acheson *et al*, 1969), heart disease (Sbar and Schlant, 1967) and patients who have undergone cardiac valve replacement (Sullivan, 1968) have shown variable results. Harrison *et al* (1971) have reported favourable response with aspirin in 2 patients with transient monocular blindness, but until proper controlled trials are carried out the value of these agents is difficult to assess.

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