Branch Retinal Artery Occlusion Secondary to a Hollenhorst Plaque

S Y Lee, J J Lee, A C H Koh, S P Chee

ABSTRACT

Retinal arterial circulation obstruction has serious implications. It may result in acute visual loss, but more significantly, it implies that the patient's systemic health needs further review and investigations in order to prevent severe and life-threatening consequences such as myocardial infarction and cerebrovascular accidents. We report a case of a patient with branch retinal artery occlusion with the presence of a Hollenhorst plaque.

Keywords: Emboli, Hollenhorst, Cholesterol, Angiography, Arteriosclerosis

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INTRODUCTION

Obstruction to the retinal arterial vasculature is not common. It is usually asymptomatic but may result in a sudden painless loss of vision which has variable recovery. Retinal emboli are encountered in about 20% of cases[1] and they indicate the presence of significant vascular disease and may forebode a poorer survival rate[2]. The most common type of retinal emboli is the cholesterol embolus, also known as the Hollenhorst plaque[2]. It is difficult and infrequent for emboli to be captured on photography and fundus fluorescein angiography as they are often transient. We present a patient in whom we were fortunate to document the finding of a Hollenhorst plaque and provide a brief discussion on its implications.

CASE REPORT

A 46-year-old Chinese male senior draughtsman presented to the Accident and Emergency Department complaining of sudden painless loss of his superior visual field in the right eye for one week. There were no other ocular or systemic symptoms. He had a history of hypertension for which he was compliant with treatment with oral anti-hypertensives. He was a non-smoker. There was no history of diabetes mellitus, hypercholesterolaemia, heart disease, previous cerebrovascular accidents or previous amaurosis fugax.

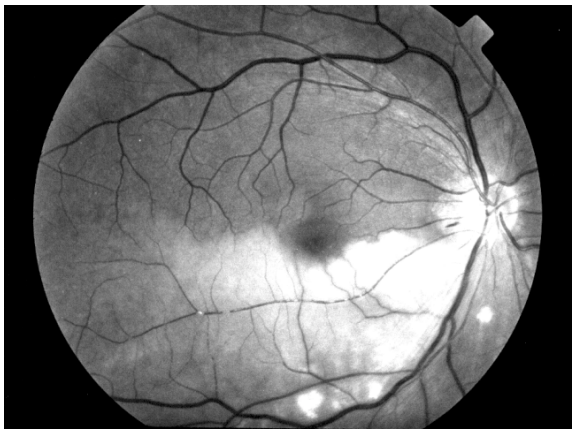

Fig. 1 Fundal photograph of the right retina showing retinal ischaemia.

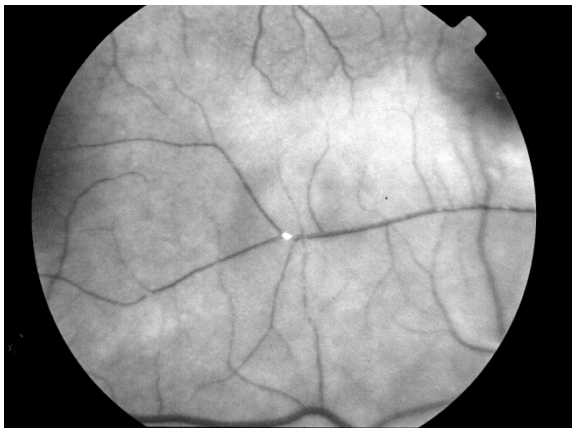

Fig. 2 High magnification of the inferior temporal arteriole showing the Hollenhorst plaque.

He had a visual acuity of 6/6 bilaterally. Examination of the anterior segment of both eyes did not reveal any abnormality. The intraocular pressures were also normal. There was no relative afferent pupillary defect detected.

Examination of the fundi after pupillary dilatation with mydriatics revealed ischaemia of the inferior temporal retina of the right eye (Fig. 1). The affected part of the retina was white and the retinal vessels were attenuated. A single embolus was seen along the inferior temporal arteriole at its bifurcation, occupying the entire vessel at the point causing occlusion (Fig. 2). It was pale and refractile. There was associated "cattle-trucking" or "boxcarring" of the involved arteriole

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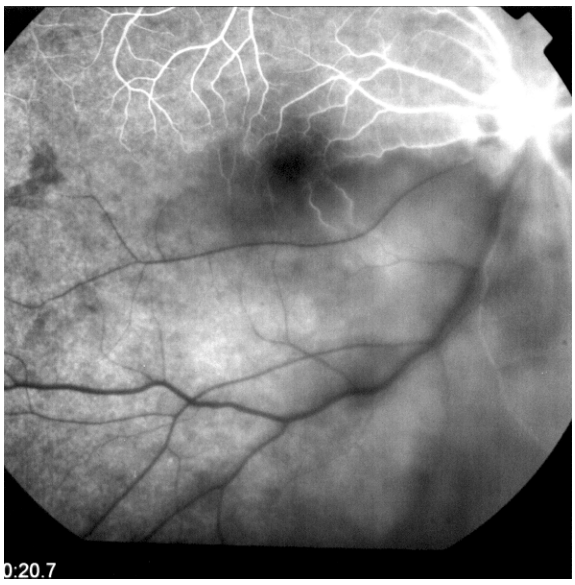

Fig. 3 Fluorescein angiogram at 20.7 seconds showing non-perfusion of the inferotemporal retina.

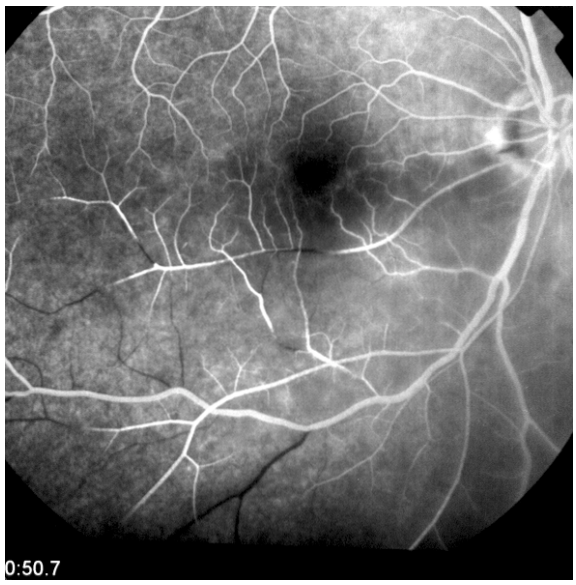

Fig. 4 Fluorescein angiogram at 50.7 seconds showing retrograde filling of the vessels in the inferotemporal retina.

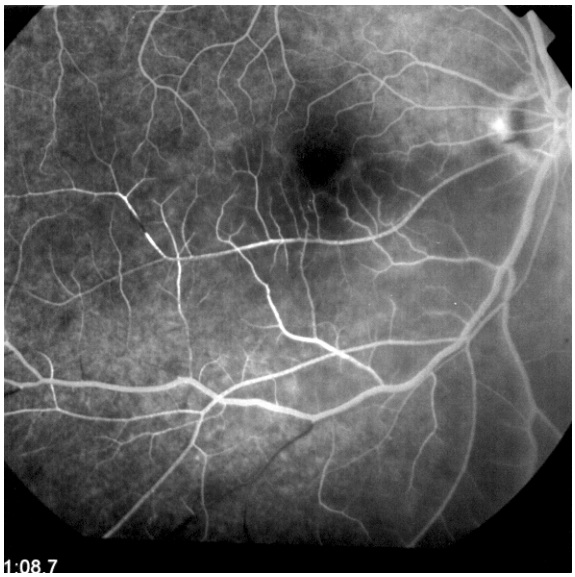

Fig. 5 Fluorescein angiogram at 1 minute 8.7 seconds showing segmental filling of the involved vessel.

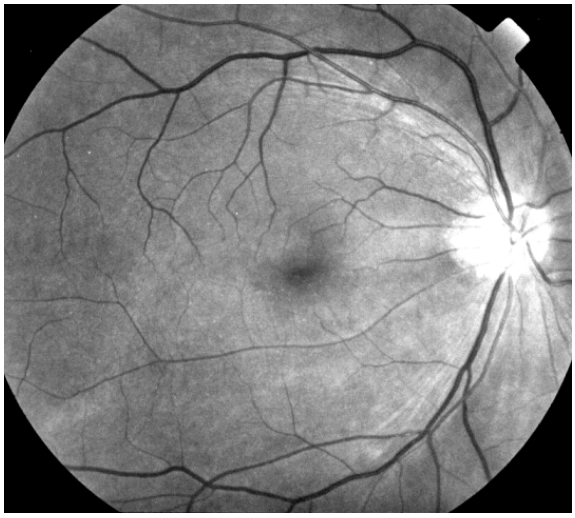

Fig. 6 Fundal photograph at 3 months review.

and adjacent areas of cotton wool spots. A splinter haemorrhage was noted on retina just temporal to disc. The fovea was uninvolved and the presence of a cilioretinal artery was noted. We did not detect any other emboli in the rest of the retina and there was no abnormality found in the fellow eye.

Systemic examination did not reveal any abnormality. In particular, the heart rate and blood pressure were normal. The heart sounds were normal and there were no murmurs auscultated. There was no carotid bruit. Fundus fluorescein angiography revealed normal choroidal filling. There was non-perfusion of the inferior temporal arteriole (Fig. 3). The flow in this arcade was re-established by retrograde fill from 45 seconds (Fig. 4). An embolus was clearly documented at the bifurcation and there was segmental filling of the affected vessel (Fig. 5). The perifoveal capillaries were not involved in the ischaemia of the retina Blood investigations were essentially normal. Random blood glucose level was 5.0mmol/L (normal). The fasting serum lipid levels were not significantly raised, total cholesterol 6.59mmol/L (mildly raised) and cholesterol-high density lipoproteins (HDL) ratio was 5.54. The erythrocyte sedimentation rate (ESR) was 34mm/h (within normal). The chest x-ray and electrocardiogram were normal.

The patient was subsequently referred to the neurologist who performed a carotid Doppler ultrasound which showed normal vessels without stenosis. Review by the cardiologist did not reveal any significant cardiac disease. The patient was started on Aspirin 300mg/day and continued with his anti-hypertensive treatment.

At three months follow-up, the patient still had good visual acuity of 6/6 in both eyes. There was no

relative afferent pupillary defect detected. The embolus had disappeared. There was resolution of the acute ischaemic features in the right retina, replaced by early pigmentary changes in that area (Fig. 6). Examination did not reveal other emboli in either eye of the patient. There were no subsequent reports of other thromboembolic episodes, in particular there was no amaurosis fugax or transient ischaemic attacks.

DISCUSSION

Hollenhorst plaques are the commonest type of retinal emboli encountered[1]. They are cholesterol crystals[3,4] that are 10 to 250 μm in diameter[5] and seen as orange refractile particles. On their own, they are less likely to cause vascular occlusion except at the smaller vessels but this probability increases greatly when they are associated with fibrin and platelets[6]. The most common source of these emboli is from ulcerative atherosclerotic plaques of the proximal carotid arteries[2,6].

The other commonly encountered types of retinal emboli include calcific emboli and fibrinoplatelet emboli. Others such as mycotic emboli, fat and exogenous material such as air or Teflon, are rarely seen. Calcific emboli are more solid compared to the cholesterol emboli[5]. They have a dirty yellowish-white look and a chunky appearance, predisposing them to greater probability of vascular occlusion in the larger first- or second-order arterioles[1,7], particularly around the optic disc. While the cholesterol emboli tend to disappear with time, calcific emboli are permanent. Calcific emboli usually arise from calcified aortic valves. Other sources include the aorta and the carotid vessels.

Fibrino-platelet emboli are grayish-white and very mobile. They move rapidly through the vasculature and tend to fragment as they pass from one bifurcation to another, resulting in fleeting episodes of symptoms[8]. It is also why they are seen less frequently on examination of the fundi.

Retinal emboli may or may not present with visual loss. It is not unusual to find retinal emboli incidentally on routine ophthalmic review in the clinic. O'Donnell and Mitchell[7] did a retrospective review of 207 consecutive patients with retinal emboli. They found that 63% of these patients were in fact asymptomatic. Sixty-nine percent had single emboli. The emboli were most frequently encountered along the course of a retinal arteriole (49%) and 41% were found at bifurcation points. The majority of the patients had a systemic disease such as hypertension, diabetes mellitus or ischaemic heart disease, and smoked. In this series, 73% were found to have significant carotid artery stenosis on Doppler ultrasound.

Frequently these patients present with visual loss and a background history of systemic disease. The systemic illnesses that have been identified as risk factors for retinal arterial occlusive are many. These include systemic hypertension (53-70%[7,9]), diabetes mellitus (21-55%[5,9]), coronary heart disease (37-46%[9]), stroke (63%[9]), smoking (60-84%[7]) and obesity. Up to 63%[9] of patients have been reported to have a history of stroke or previous cerebrovascular event including transient ischaemic attacks and amaurosis fugax.

Pfaffenbach and Hollenhorst[9] studied the morbidity and survivorship of patients with retinal cholesterol emboli and found that they had significantly higher mortality than patients without. They found that at the time of diagnosis, 93% of the patients already had significant arteriosclerotic disease and 65% died over a 6 to 15 year follow-up, mainly from carotid disease or cerebrovascular accidents. Other more recent studies have also shown significant carotid artery stenosis in 22-53% of patients[10-12] and carotid end-arterectomy has been advocated for these patients to prevent future cerebral embolism. Schwarz et al reviewed a series of 64 patients with Hollenhorst plaques to determine if carotid endarterectomy prevented the recurrence of emboli or symptoms[13]. They found that 24 patients out of 28 who underwent the surgery remained asymptomatic and the remaining 4 developed new ipsilateral Hollenhorst plaques subsequently.

Anticoagulation with an anti-platelet aggregating drug has been advocated to reduce the risk of cerebrovascular accidents, myocardial infarction and vascular deaths, particularly in patients with carotid ulceration without significant stenosis[6] and patients who are unfit for surgery. Aspirin has so far been the unrivalled drug of choice although the dosage has yet to be agreed upon.

Management of the eye with branch retinal artery occlusion is largely conservative. The prognosis for visual acuity is usually good because of the development of collaterals and the presence of intact perifoveal capillaries. The main aim of the management of these patients is to identify the source of the emboli and treat any systemic disease before fatalities occur.

This patient had only two risk factors for atherosclerotic disease – hypertension and hyperlipidaemia. He had normal carotid vessels on ultrasonography. Various studies have shown that up to two-thirds of patients with retinal arterial occlusive disease can be found to have normal carotid ultrasonograms[14,15]. This may be explained by the possibility of the level of occlusion at the ophthalmic artery, which was not imaged. The frequency of retinal ischaemia and cardiac pathology has not been well established although there have been reported cases of associations[10]. Fortunately for our patient, he did not have cardiac disease. Nevertheless, he was started on

Aspirin to prevent further embolic disease.

Therefore it is imperative that patients with retinal vaso-occlusive disease be screened systemically for disease that may have a significant effect on his future health. As many of these patients first present to the ophthalmologist, further referral to the neurologist and cardiologist is advisable. These patients then need to be treated for their systemic illness as well as with treatment directed at the embolism, ie carotid endarterectomy or anticoagulant therapy.

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