Chronic ocular ischaemia

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SUMMARY The ocular changes due to chronic ischaemia are described in seven patients, aged between 59 and 77 years, with severe carotid artery disease. Doppler sonography was used to confirm the presence of high-grade arterial stenosis or occlusion in all the patients. The treatment of chronic ocular ischaemia is discussed, including the role of carotid bypass surgery, which is mentioned briefly. Three patients died of cardiovascular or cerebrovascular disease within 6 to 18 months of presentation.

The role of extracranial carotid artery disease as a source of ocular pathology has been increasingly recognised over the past 20 years.¹ The commonest ocular manifestation of distant arterial disease is embolic, and the dramatic symptoms accompanied in some cases by the finding of retinal emboli usually enables the correct diagnosis to be made. Less frequently occlusion of one or more of the major arteries in the neck may produce ocular ischaemia. This disease entity has also been well documented,^{2,3} but the bewildering variety of terms employed by different authors tends to obscure the fact that they are all referring to the same basic condition. It is probable that the diagnosis of chronic ocular ischaemia is sometimes missed or delayed, as some of the following cases show, particularly since many ophthalmologists confine their examination to the patient's eyes and rarely carry or use a stethoscope.

Seven patients with chronic ocular ischaemia are described in order to illustrate the wide spectrum of symptoms and signs which may be encountered in this condition. All patients were studied by Doppler sonography, which is an accurate, non-invasive method of detecting occlusive disease of the carotid circulation.⁴

Material and methods

All seven patients were examined personally in the University Eye Clinic by G.D.S. Doppler studies were performed in the University Department of Neurology by H.R.M. A Delalande continuous wave directional device emitting at 4 MHz was used to explore the common, internal, and external carotid

arteries, the supratrochlear and, in some cases, the supraorbital arteries, as well as the vertebral and subclavian arteries. Pulsatile and mean flow velocity and direction of blood flow were recorded on paper, while stenoses and turbulence were detected by ear from the audio output.

Case reports

CASE 1

A 75-year-old man presented in September 1979 complaining of misty vision in the right eye for two months. He gave a history of intermittent claudication. Blood pressure was 180/80 mmHg.

Findings: RV 6/36+cc, VL 6/12p cc. The right eye was injected and the cornea oedematous. The anterior chamber was poorly visualised, but the pupils were equal. A hazy view of the right fundus revealed no obvious abnormality. The cornea of the left eye was clear without any guttata, and the left fundus was normal. A diagnosis of right corneal endothelial decompensation of unknown cause was made. Five months later vision in the RE had fallen to hand movements, the eye was still injected, and gross bullous keratopathy had developed (Fig. 1). Anterior segment ischaemia was suspected, and Doppler sonography revealed occlusion of the right internal carotid artery. The pulse curve registered from the right common carotid artery showed no flow in diastole, the right internal carotid artery could not be identified and the right supratrochlear artery showed very slow, antegrade flow which increased considerably during compression of the ipsilateral superficial temporal artery. Flow in the left carotid system was normal. A carotid angiogram was not performed because vascular surgery was felt not to be

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Fig. 1 Case 1. right eye with gross bullous keratopathy due to chronic ischaemia.

Fig. 2 Case 2. Chronic ischaemia of the left fundus: fleck-shaped haemorrhages above and temporal to the fovea.

Fig. 3 Case 2. Chronic ischaemia of the left fundus: marked retinal arterial narrowing and irregularity with scattered fleck-shaped haemorrhages.

indicated. Three months later the condition of the RE was unchanged. Intraocular pressure was 15 mmHg on the right and 16 mmHg on the left. In January 1983, 3½ years after the onset of symptoms, the right cornea was still grossly oedematous, while the LE remained normal. Repeat Doppler sonography in September 1983 showed no change. Flow in the right common carotid artery, measured with the Hayashi-Denki QFM system⁵ was 1.93 ml/s compared

with 10.09 ml/s in the left common carotid, indicating marked left to right carotid cross flow intracranially.

CASE 2

A 63-year-old man was first seen in April 1977 with a 6-month history of misty vision. He suffered from hypertension and angina pectoris and appeared to be somewhat confused and forgetful. Visual acuity was 6/7.5 in both eyes, which were normal on examination. However, perimetry revealed a homonymous left inferior quadrantanopsia, and a bruit was heard over the right carotid artery. His blood pressure was 200/110 mmHg.

Findings in July 1980: VA 6/9 in both eyes. The left fundus now contained scattered fleck-shaped haemorrhages in the midperiphery (Fig. 2), the arteries were narrow and irregular in calibre (Fig. 3), and sludging of blood was visible in the dilated veins. The right fundus was normal. Intraocular pressure was 14 mmHg on the right and 15 mmHg on the left. Blood pressure was 135/85 mmHg. A CT scan in October 1980 showed generalised cortical atrophy and an old parieto-occipital infarct on the right. A carotid bruit was now audible bilaterally, and the diagnosis of chronic ischaemia of the left fundus due to carotid artery disease was confirmed by Doppler sonography. A stenosis signal with considerable post-stenotic turbulence was detected over the origins of both internal carotid arteries. Flow in the right supra-trochlear artery was retrograde and reverted to antegrade flow during compression of the right facial artery. Flow in the left supra-trochlear artery was in the physiological direction but very slow, with

Fig. 4 Case 3. Left eye. Anterior segment ischaemia with small hyphaema and posterior synechiae (iatrogenic subconjunctival haemorrhage).

Fig. 5 Case 3. Ischaemia of the left fundus: narrow arteries, dilated veins with interrupted columns of blood and retinal oedema.

Fig. 6 Case 4. Left optic disc with prominent new vessels (arrowheads) due to chronic ischaemia. October 1980.

Fig. 7 Case 4. Partial fibrosis of disc new vessels two years after brachiocephalic endarterectomy.

no increase in velocity during compression of the superficial temporal and facial arteries. Stenosis of both internal carotid arteries and, probably, the left external carotid artery was diagnosed. Angiography was not performed. In January 1981 the patient died suddenly of a myocardial infarct. Post-mortem examination revealed a fairly recent occlusion of the right internal carotid artery and a very tight stenosis of the left internal carotid artery.

CASE 3

A 69-year-old man was admitted as an emergency in October 1980 with a diagnosis of left haemorrhagic glaucoma. Owing to the patient's poor memory a full history was only later obtained from his wife. Hypertension had been diagnosed 10 years ago and he had suffered two mild episodes of right hemiparesis, the first in 1977 and a second in March 1980. Angiography performed elsewhere in 1977 had shown a left internal carotid artery stenosis.

Fig. 8 Case 6. Anterior segment ischaemia with rubeosis iridis, ectropion uveae, and posterior synechiae.

Fig. 9 Case 7. Left eye showing dilated episcleral vessels due to chronic ocular ischaemia.

Findings in October 1980: RV 6/6, LV hand movements. The RE was normal. The LE appeared hyperaemic with mild corneal oedema, fine keratic precipitates, and aqueous flare and cells. A small hyphaema was visible associated with early rubeosis iridis. The lens was clear. Fundus examination showed some scattered small blot haemorrhages. The retinal veins appeared to be mildly congested, while the arteries were markedly constricted and pulsated spontaneously on the disc at a systemic blood pressure of 190/100 mmHg. Intraocular pressure was 19 mmHg on the right and 30 mmHg on the left. Fluorescein angiography of the LE showed very slow perfusion of the retinal vessels. Dye did not appear in the arteries until 37 seconds after injection, and the veins never filled completely. Doppler sonography showed complete occlusion of the left internal carotid artery and a well developed ophthalmic collateral circulation with rapid retrograde flow in the left supraorbital and

Fig. 10 Case 7. Left eye. Chronic anterior segment ischaemia with rubeosis iridis (arrowheads) and dense cataract.

supratrochlear arteries, the latter being supplied via the left external carotid artery. Aortic arch angiography confirmed the diagnosis of total occlusion of the left internal carotid and also showed occlusion of both vertebral arteries with tight stenosis of the right internal carotid artery intracranially.

Five days after admission posterior synechiae developed in the LE (Fig. 4), the rubeosis slowly increased, and four weeks later intraocular pressure reached 50 mmHg, after which the eye became blind. Under treatment the pressure fell to around 30 mmHg. The central retinal artery still pulsated weakly, but the columns of blood in the veins were discontinuous (Fig. 5) and the retina was oedematous, with a cherry red spot centrally. An intermittent left-sided headache was thought to be ischaemic in origin, since it did not correlate with the intraocular pressure.

The patient was not considered suitable for surgery. In May 1981 he died suddenly, and at necropsy both internal carotid arteries were seen to be occluded.

CASE 4

A completely asymptomatic 59-year-old woman was found to have forward new vessels on the left disc when she visited her ophthalmologist for glasses. There was no history of cerebro- or cardiovascular disease, and a glucose tolerance test was normal.

Findings in October 1980: VA 6/6 cc both eyes. The RE was normal as was the left anterior segment. A prominent vascularised glial veil arose from the left disc (Fig. 6). The major vessels at the posterior pole were unremarkable apart from focal constriction of the superotemporal artery but microaneurysms, telangiectases, and areas of vascular closure were

evident peripherally. Bilateral carotid bruits were noted, and the brachial blood pressure measured 80/65 mmHg on the right and 120/80 on the left. Doppler sonography showed a brachiocephalic steal syndrome. Angiography confirmed total occlusion of the brachiocephalic trunk, associated with retrograde flow down the right vertebral artery, which thereby supplied the subclavian and common carotid arteries. In addition the left common carotid artery was occluded in its middle third, so that cerebral perfusion was totally dependent on the left vertebral artery. A brachiocephalic endarterectomy was performed in October 1981, and Doppler sonography in November 1981 and March 1983 showed physiological flow in the right vertebral and common carotid arteries. The fibrovascular frond on the left disc persisted, although shrinkage of the previously fine capillary network at the tip of the frond was accompanied by an increase in the amount of glial tissue (Fig. 7). Vision in the LE remained full.

CASE 5

A 77-year-old man presented in April 1981 with a two-week history of pain above the left eye and reduced vision. Three years previously he had suffered a transient episode of aphasia.

Findings: RV 6/12 cc (later recorded as 6/6p), LV 6/12p cc. The right eye was normal. Apart from conjunctival injection the left anterior segment was also reported as normal. Five days later the left supra-orbital pain was worse and vision had fallen to 6/18. Examination now showed folds in Descemet's membrane, aqueous flare, and rubeosis iridis with haemorrhage and new vessels obscuring the chamber angle. Multiple small haemorrhages were scattered throughout the left fundus, and the retinal veins appeared congested. The intraocular pressure was 14 mmHg in each eye but fell to 4 mmHg on the left a week later when posterior synechiae had started to form. Doppler sonography showed total occlusion of the left internal carotid artery, which could not be identified with the transducer. High diastolic flow in the left external carotid artery indicated collateral circulation, as did retrograde flow in the left supratrochlear and supraorbital arteries, which reverted to antegrade during compression of the left facial and superficial temporal arteries respectively. The right carotid artery was normal. The blood pressure was 130/60 mmHg in the presence of atrial fibrillation.

Six weeks after the patient was first seen new vessels were noted on the left disc. The intraocular pressure abruptly rose to 30 mmHg causing headache and vomiting. A single Diamox Sustet (acetazolamide) daily lowered the pressure to 5 mmHg, which promptly rose again to 30 mmHg when treatment was stopped. Vision fell to counting fingers. The rubeosis

regressed, and by October 1981 the intraocular pressure stabilised at around 20 mmHg without acetazolamide, even though the chamber angle was totally occluded. Spontaneous pulsation of the central retinal artery became visible with sludging of blood in the arteries and veins. By October 1982 vision in the left eye had fallen to light perception and a cataract had developed. One year later the eye was blind, with a pressure of 25 mmHg.

CASE 6

A 70-year-old man presented in July 1981 with misty left vision of two days' duration. He was under treatment for hypertension and diabetes mellitus and had been on anticoagulants since 1971 following a right femoral endarterectomy. He suffered a transient right hemiparesis with aphasia in October 1980, when Doppler sonography showed occlusion of the left internal carotid artery.

Findings in July 1981: RV 6/6, LV counting fingers. The RE was normal. The LE showed conjunctival injection with corneal epithelial oedema, aqueous flare, and rubeosis iridis. The chamber angle was almost totally occluded. Fundus examination showed scattered fleck haemorrhages, dilated veins, marked irregularity in arterial calibre, and a spontaneous arterial pulse on the disc. The intraocular pressure was 22 mmHg on the right and 42 mmHg on the left. Doppler sonography again showed total occlusion of the left internal carotid artery with a well developed collateral circulation via the supratrochlear and supraorbital arteries, which were fed by the ipsilateral external carotid artery. The right carotid artery was normal.

The intraocular pressure was reduced to under 20 mmHg with acetazolamide but vision fell to light perception over the following weeks. The rubeosis increased (Fig. 8) and the retinal circulation deteriorated until retinal oedema developed, with sludging visible in the veins. By April 1982 the LE was blind but remained pain-free with an intraocular pressure of 25 mmHg, when acetazolamide was stopped. Five months later a cataract had developed. In December 1982 the patient died of a myocardial infarct.

CASE 7

A 71-year-old man was referred in May 1983 with a left cataract allegedly due to ocular contusion. Fourteen months previously vision in the left eye had been recorded as 6/60 and scattered haemorrhages were seen in the left fundus. Two months later, in April 1982, rubeosis iridis was noted in the left eye, but the intraocular pressure was normal—13 mmHg on the right and 11 mmHg on the left. The retinal haemorrhages were ascribed to 'arteriosclerosis.'

The normotensive patient was under treatment for ischaemic heart disease.

Findings in May 1983: RV 6/6, LV light perception. The RE was completely normal. The episcleral vessels of the LE were dilated (Fig. 9) but the cornea was clear and the anterior chamber quiet. The pupil was dilated and immobile due to rubeosis, which had also totally occluded the chamber angle. A dense cataract precluded any view of the fundus (Fig. 10). The intraocular pressure was 16 mmHg in each eye. A bruit was audible over the left carotid artery and pulses in the left arm were weak. Chronic ischaemia affecting the LE and stenosis of the left subclavian artery were diagnosed. Doppler sonography performed four months later revealed occlusion of the left internal carotid artery and an incomplete subclavian steal syndrome with bidirectional flow in the left vertebral artery. In October 1983 vision in the LE was unchanged. The anterior chamber now contained a heavy flare, with some cells, but the intraocular pressure was still 16 mmHg in each eye.

Discussion

These seven cases illustrate the wide range of symptoms and signs which may be encountered in patients with chronic ocular ischaemia. The acute presentation of case 3, with the picture of neovascular glaucoma, was in striking contrast to the chance finding of retinal haemorrhages in case 2 and disc new vessels in case 4, both patients being totally asymptomatic. Case 5 presented with a two-week history of left-sided headache, which was presumably due to cranial ischaemia, since the left eye initially showed little abnormality. Painless loss of vision was the presenting feature in the remaining three patients, being due to corneal oedema in case 1, retinal ischaemia in case 6, and a combination of retinal ischaemia with subsequent cataract formation in case 7.

The large number of different names applied by various authors to chronic ocular ischaemia clearly reflects the variable expression of this condition. The terms ischaemic glaucoma,⁶ ischaemic ocular inflammation,³ and neovascular glaucoma⁷ signify predominantly anterior segment ischaemia, whereas retinal ischaemia has been described as venous stasis retinopathy,² hypotensive retinopathy,⁸ or collateral flow retinopathy.⁹ Affection of the whole eye has been called ischaemic oculopathy¹⁰ or ischaemic ophthalmopathy.¹¹ It would seem more sensible to replace this confusing array of terms with the single description chronic ocular ischaemia.

The diagnosis of chronic ocular ischaemia can usually be made clinically, on the basis of history and physical signs. A past history of cardiovascular or

cerebrovascular disease can be elicited from most of these patients, though in the presence of poor cerebral perfusion history taking may be difficult.

Any part of the eye may be affected by chronic ischaemia, but it is uncommon for both eyes to be involved, despite the presence of bilateral vascular disease. Dilated episcleral vessels are a frequent finding¹² and have even been described as a sign of ipsilateral carotid artery occlusion in seven patients with cerebrovascular insufficiency but no other evidence of ocular ischaemia apart from low ophthalmodynamometry readings.¹³ The significance of corneal oedema is usually obvious when a patient presents with the classical picture of anterior segment ischaemia. However, case 1 presented with the initially inexplicable finding of corneal endothelial decompensation in a normotensive eye with no evidence of rubeosis and no history of trauma. The endothelium in the unaffected left eye was morphologically normal, thus excluding a dystrophy. The diagnosis of ocular ischaemia was considered only when a history of intermittent claudication was obtained.

The discovery of extensive peripheral anterior synechiae associated with rubeosis in an eye with normal or low intraocular pressure is strongly suggestive of ischaemia, although a long-standing retinal detachment must be excluded. Diminished production of aqueous by an ischaemic ciliary body is presumably the reason why the intraocular pressure may remain delicately balanced between glaucoma and hypotony, as in case 7. Should the pressure rise, it can often be reduced with a carbonic anhydrase inhibitor, as in cases 5 and 6.

The retinal changes caused by chronic ischaemia may take several forms, possibly reflecting the rate at which ischaemia develops, and must be differentiated from those due to retinal arterial and venous occlusion, and diabetic retinopathy. The effects of ischaemia developing over a short period of time are exemplified by case 3, which presented with neovascular glaucoma which was initially ascribed to occlusion of the central retinal artery. However, secondary glaucoma following arterial occlusion is uncommon, occurring in only 1% of cases.¹⁴ Although Smith⁶ applied the term 'ischaemic glaucoma' to neovascular glaucoma caused, apparently, by central retinal artery occlusion associated with ipsilateral carotid occlusion, it was Knox³ who suggested that obstruction of the ophthalmic or carotid arteries might be the common denominator in some of these cases, causing both the rubeosis and the retinal artery occlusion. Indeed Hayreh and Podhajsky¹⁵ believe that rubeosis is hardly ever caused by isolated central retinal artery occlusion, reflecting, instead, severe carotid artery disease.

The chance finding of blot haemorrhages distributed in the midperiphery of the retina in case 2 associated with a congested appearance of the veins which showed sludging probably indicates ischaemia of more gradual onset. This appearance, which has been misleadingly called venous stasis retinopathy,² must be distinguished from that due to central retinal vein thrombosis.¹⁶ Regrettably this same term, venous stasis retinopathy, was subsequently applied to certain cases of central retinal vein thrombosis,¹⁷ thereby compounding the confusion.

The retinal changes seen in case 4, with forward new vessels on the left disc and peripheral vascular closure, bear some resemblance to diabetic retinopathy and, by analogy, probably resulted from ischaemia of very gradual onset. However, diabetic retinopathy is usually bilateral, and the microvascular changes are most marked at the posterior pole, whereas the retinopathy due to chronic ocular ischaemia is usually unilateral, and the microvascular changes are predominantly peripheral. Furthermore, diabetes can be excluded by a glucose tolerance test.

Spontaneous pulsation of the central retinal artery was visible in three patients (cases 3, 5, and 6) with normal or only moderately raised intraocular pressure. This finding, in the presence of a normal blood pressure, signifies reduced ophthalmic artery pressure, due almost invariably to carotid artery obstruction. Indeed, measurement of ophthalmic artery pressure by ophthalmodynamometry is widely practised as a simple means of assessing carotid perfusion. However, the accuracy of this method, compared with carotid angiography, is only 70–75%,¹⁸ mainly due to a high incidence of false negative results.

A clinical diagnosis of chronic ocular ischaemia implies the presence of severe carotid artery disease,¹⁹ which clearly requires further investigation without at the same time exposing all patients to the risks of carotid angiography.²⁰ Doppler sonographic examination of the carotid arteries in the neck, the periorbital branches of the internal carotid artery, and, when indicated, the subclavian and vertebral arteries enables high grade stenosis (>50%) or occlusion of the common or internal carotid arteries to be diagnosed with an accuracy of 95 to 97%.^{21–23} Many American investigators restrict their Doppler examination to the supraorbital arteries,^{24–26} resulting in a higher incidence of false positive and false negative results. In the present series of patients carotid angiography was reserved for those in whom vascular surgery was contemplated.

The reported incidence of chronic ocular ischaemia in several series of patients with carotid arterial obstruction varies between 4 and 18% depending on the criteria used to define ischaemia and also the

composition of the different groups of patients. Homolateral retinopathy (cotton-wool patches and reduced ophthalmic artery pressure) was observed in 23 out of 235 patients with symptomatic occlusive disease of the carotid artery.²⁷ Venous stasis retinopathy was noted in 22 out of 600 patients with 'intermittent insufficiency or thrombosis in the carotid arterial system'² and in 13 out of 72 patients awaiting vascular surgery for carotid artery occlusion.⁹ No figures exist for the incidence of chronic ocular ischaemia in the general population, but six of the seven patients described here presented over a period of two years at a hospital serving a population of 400 000.

The prognosis for eyes affected by chronic ischaemia is generally poor, though improvement following treatment has been reported in a few cases. Mild ischaemic retinopathy (VA 6/7.5) in a 60-year-old patient resolved following carotid endarterectomy,²⁸ optic disc new vessels regressed after pan-retinal photocoagulation in a 56-year-old man,²⁹ and 'some decrease in retinopathy' was reported in 13 patients after carotid bypass surgery.⁹ However, significant loss of vision due to ischaemia is probably not reversible. Thus no improvement in ocular function was seen after carotid bypass surgery in six patients with ocular ischaemia.¹⁰ Successful vascular surgery may even exacerbate the condition of an ischaemic eye, as in one patient with rubeosis iridis and hypotony who developed glaucoma following carotid bypass surgery,¹⁰ presumably due to an increase in the ciliary body blood supply. The results of cataract surgery in such patients are also disappointing owing to coexisting retinal ischaemia. Lens extraction in two patients led to a very modest increase in visual acuity, while a vitreous haemorrhage occurred postoperatively in a third patient whose eye was later enucleated.³

It is doubtful whether chronic ocular ischaemia *per se* represents an indication for major vascular surgery, such as endarterectomy, which may in any case be contraindicated owing to the widespread nature of the vascular disease and the advanced age of many patients. However, all such patients should be considered for carotid bypass surgery in an attempt to safeguard cerebral perfusion. This recently developed procedure³⁰ involves anastomosing the superficial temporal artery to a branch of the middle cerebral artery and has a high success rate with few complications.³¹ Ophthalmic artery pressure, however, does not increase significantly following this operation.⁹

In view of the serious arterial disease always present in patients with chronic ocular ischaemia it might be anticipated that the prognosis for life is guarded, and indeed three of the present series of seven patients died within six months to 1½ years of presentation.

Carotid bypass surgery had not been performed in these patients, but the cause of death was myocardial ischaemia in two cases and only one died of cerebrovascular disease.

Despite the rather depressing and therapeutically resistant course of chronic ocular ischaemia it remains important to recognise this condition, as case 4 illustrates. The observation of optic disc new vessels in this asymptomatic patient led to the discovery and timely operative relief of a precariously reduced cerebral blood supply.

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