Carotid artery disease and glaucoma

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SUMMARY Five patients with evidence of severe bilateral carotid artery disease and increased intraocular pressure have been followed up for 3 to 12 years without the development of glaucomatous disc or field changes. Although it is frequently stated that glaucoma patients with low systemic or ophthalmic artery blood pressures show rapid progression of their eye disease, our patients show that this is not necessarily true. Other more important and largely unknown susceptibility factors determine the occurrence of glaucoma in patients with raised intraocular pressure.

The management of patients with raised intraocular pressure, normal optic discs, and full visual fields remains a controversial topic. Fewer than 10% of these patients with so-called ocular hypertension will ultimately develop glaucomatous disc and field changes, and individual susceptibility to a given level of intraocular pressure varies greatly. One susceptibility factor that is frequently mentioned is blood pressure. It has been stated that patients with low systemic or ophthalmic artery blood pressure are prone to develop glaucomatous disc and field changes, and patients with carotid artery disease have been used as evidence that arterial pressure is an important factor in the development of glaucoma or low-tension glaucoma (Harrington, 1959; 1961; Francois and Neetens, 1970). The purpose of this report is to describe 5 patients with increased intraocular pressures and severe bilateral carotid artery disease who have been followed up for 3 to 12 years without the development of glaucomatous disc or field changes.

Case reports

Case 1
A 58-year-old man had a 33-year history of mild systemic hypertension and a 2-year history of diabetes mellitus. In 1975 the patient reported the onset of transient ischaemic attacks. A right carotid bruit was noted, and cerebral angiography showed severe stenosis of the right common carotid artery and total occlusion of the left common carotid artery. In March 1976 a right carotid endarterectomy was performed without complication.

The patient’s ophthalmological history disclosed the presence of increased intraocular pressure of 12 years’ duration. For the first 7 years of this period he was treated intermittently with pilocarpine and epinephrine drops. For the last 5 years he had received no glaucoma therapy. During this time his intraocular pressures were measured in the low- to mid-twenties. Ophthalmological evaluation on 24 February 1977 showed a visual acuity in the right eye of 20/25 and 20/20 in the left. Applanation tensions were 22 mmHg in both eyes. Fundus examination showed a cup/disc ratio of 0-2 bilaterally. Goldmann fields were normal. Ophthalmodynamometry showed a reading of 140/50 in the right eye and 65/30 in the left. (This and all subsequent ophthalmodynamometry readings were measured with the Baillart instrument. Values are expressed in scale units.) Blood pressure was 140/95 mmHg. Despite the asymmetry of his ophthalmic artery pressures (related to previous unilateral surgery) no asymmetry of the discs was detected.

Case 2
A 62-year-old woman had a long-standing history of mild systemic hypertension and arteriosclerotic heart disease. In September 1974 she first complained of amaurosis fugax in the right eye. A diminished right carotid pulse with a right carotid bruit was present. Cerebral angiography showed bilateral carotid artery disease. Ophthalmodynamometric measurements were 80/10 in the right eye and 90/10 in the left. Blood pressure was 130/80 mmHg. A right carotid endarterectomy was performed in October 1974 with resultant relief of the amaurosis.
but no change in the ophthalmodynamometry measurements.

The patient's past eye history disclosed increased intraocular pressure first detected in 1974. Her intraocular pressures, with no treatment, were consistently measured in the mid- to the high-twenties. Fundus evaluation showed normal discs with a cup/disc ratio of 0.1 bilaterally. There was slight but definite retinal venous tortuosity. Goldmann fields were normal. With the exception of a very brief trial of 2% pilocarpine she remained untreated for 3 years, and no change in her discs or fields was noted.

CASE 3
A 59-year-old man was seen in February 1977 with a 1-year history of amaurosis fugax in his right eye. A loud left carotid bruit was noted. Ophthalmodynamometry revealed a diastolic reading of 35 in the right eye and 48 in the left. A cerebral angiogram showed 90% occlusion of the right common carotid artery and a large ulcerative plaque in the left common carotid artery.

The patient had a history of raised intraocular pressures for 11 years. Throughout this time his pressures were consistently measured in the low twenties while receiving 4% pilocarpine 4 times a day. On several occasions when the pilocarpine therapy had been stopped the pressures were measured in the low thirties. Eye evaluation February 1977 showed bilateral vision of 20/20. His applanation pressures on pilocarpine therapy were 25 mmHg in both eyes. There was slight but definite retinal venous tortuosity. Goldmann fields were normal. His discs showed a cup/disc ratio of 0.3 in both eyes. This was unchanged from previous examinations. The patient was then referred for vascular surgery.

CASE 4
A 61-year-old woman had a long-term history of type 4 hyperlipoproteinaemia and a 12-year history of hypertension. In December 1976 she was evaluated because of episodes of diplopia, vertigo, and dizziness, dating back to 1969. She had also noted occasional episodes of amaurosis fugax. Ophthalmodynamometry showed a reading of 80/40 in the right eye and 80/38 in the left. Blood pressure was 150/92 mmHg. The left carotid pulse was diminished. A cerebral angiogram showed severe stenosis of both common carotid arteries.

Her past eye history disclosed increased intraocular pressure that had been present since 1969. Her intraocular pressures were consistently measured in the mid- to upper-twenties. Ophthalmological examination on 6 December 1976 showed a bilateral visual acuity of 20/25. Applanation tensions were 26 in the right eye and 27 in the left. The cup/disc ratio was right eye 0.3, left eye 0.4. A left posterior vitreous detachment and mottling of pigment in the left superior macular was present. Goldmann perimetry and tangent screen testing were normal.

CASE 5
A 54-year-old woman had a history of transient ischaemic attacks for the past several years. In December 1975 a loud bruit was noted over the right common carotid artery and a faint bruit over the left common carotid artery. Cerebral angiography showed severe bilateral stenosis of the internal carotid arteries. The patient's past eye history disclosed raised intraocular pressures that had been greater in the right than left eye since at least 1972. Her pressure measurements in the right eye had varied between 20 and 36 mmHg and in the left eye between 19 and 25 mmHg. Only the right eye had been intermittently treated with pilocarpine, and pressures determined while the patient was receiving medication were measured in the low twenties. Ophthalmological examination on 2 December 1975 showed a best corrected bilateral vision of 20/60. The right iris showed a diffuse loss of pigment with pigment deposition on the corneal endothelium and the angle. The left iris was normal. The chamber angles were wide open with the possibility of a subtle 360° angle recession in the right eye. Ophthalmodynamometry showed readings in the right eye of 110/40 and 125/50 in the left. Blood pressure was 180/80 mmHg. Ophthalmological examination in December 1975 showed multiple Hollenhorst plaques in both eyes. The arterioles showed arteriovenous nicking and increased light reflex. The cup/disc ratio was 0.2 in both eyes (unchanged from 1972). Tangent screen testing revealed diffuse constriction bilaterally with no glaucomatous changes. The patient was followed up with treatment consisting of 2% pilocarpine in the right eye only, 4 times daily. Her last examination was March 1977. Her pressures during that time were in the 20 to 25 mmHg range in both eyes. There was no change in the discs or fields.

Discussion
All 5 of these patients have had amaurosis fugax or transient ischaemic attacks with documented bilateral carotid arterial occlusive disease. Patients 1, 2, and 3 had unilateral carotid endarterectomy, while Patients 4 and 5 were considered not to be candidates for surgery. After right carotid endarterectomy Patient 1 showed a pronounced difference in ophthalmic artery pressures between the 2 eyes; Patient 2 showed very low diastolic readings. Definite venous
tortuosity, presumably mild venous stasis retinopathy (Kearns and Hollenhorst, 1963) was seen in Patients 2 and 3, and Hollenhorst plaques were noted in Patient 5.

All patients had raised intraocular pressure. These elevations had prompted intermittent treatment (by other ophthalmologists) in Patients 1, 2, and 5 (right eye only) and constant treatment in Patient 3. Despite the increased intraocular pressure in association with significant carotid artery disease no glaucomatous disc or field changes have been noted. Follow-up of the ocular hypertension in these patients has varied from 3 to 12 years.

The importance of the level of systemic blood pressure in the development of glaucomatous changes is unknown. Although it has been suggested that shock may cause glaucomatous disc and field changes (Drance et al., 1973), the occurrence of 'glaucomatous changes' after an episode of systemic hypotension has been questioned (Jampol et al., 1978). It is also frequently stated that patients with low ophthalmic arterial blood pressures will show rapid progression of their glaucoma. This is also unproved. Drance (1962) compared the diastolic ophthalmic artery pressures of both eyes of a group of 12 patients with advanced glaucoma in 1 eye and early glaucoma in the other eye. He found no difference in pressure between the 2 eyes.

The significance of the ophthalmic artery blood pressure has been supported largely by case reports (Harrington, 1959; 1961; François and Neetens, 1970) of glaucoma patients with low blood pressure or carotid artery disease whose conditions progress rapidly. However, in most instances systemic or ophthalmic artery blood pressures have not proved useful prognosticators of future glaucomatous loss; many patients with glaucoma or low-tension glaucoma actually have systemic hypertension (Leighton and Phillips, 1972).

Our 5 patients with carotid artery disease and ocular hypertension demonstrate that carotid occlusive disease and low ophthalmic artery pressures, per se, in a patient with increased intraocular pressure do not necessarily indicate future glaucomatous changes (at least during follow-up periods of several years). Other (largely unknown) susceptibility factors are present that determine the occurrence of glaucomatous disc and field changes.

References


