Sir,

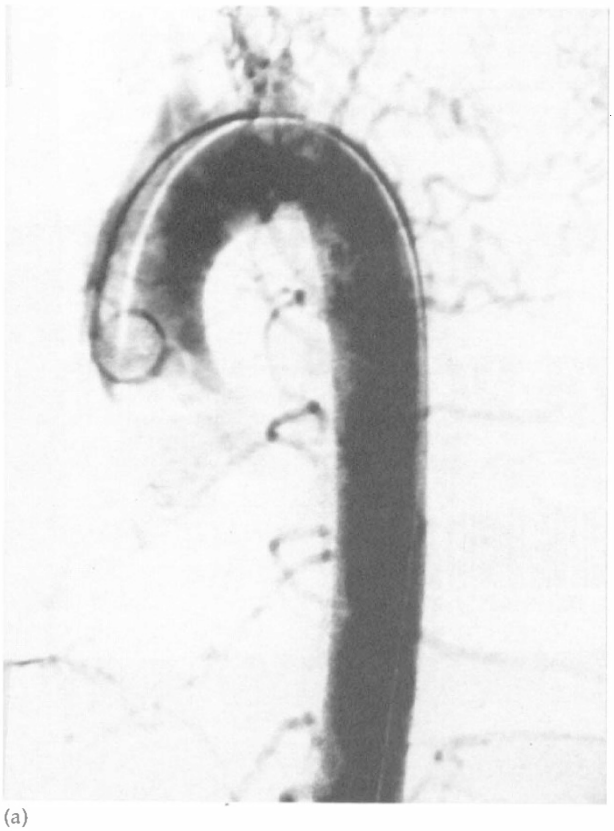
Neovascular glaucoma after bypass surgery in Takayasu's disease

Takayasu's arteritis (TA) is a chronic inflammation of unknown origin that primarily involves the aorta and its major branches.[1] This may lead to progressive obliteration of the aortic arch and its large branches, with diminution of the arterial blood flow in the upper part of the body. Asian and Mediterranean women in the second or third decade of their life are most commonly affected. The ocular findings were first described in 1908 by Takayasu, who was an ophthalmologist.[1] The ocular involvement in TA is secondary to the ocular ischaemia, the most typical finding being ischaemic retinopathy. Other ocular findings in TA may include: cataract formation,[2] anterior ischaemic optic neuropathy[3] and iris neovascularisation.[4,5] Although neovascular glaucoma is said to be a known complication in TA, only one documented case has been previously described.[6]

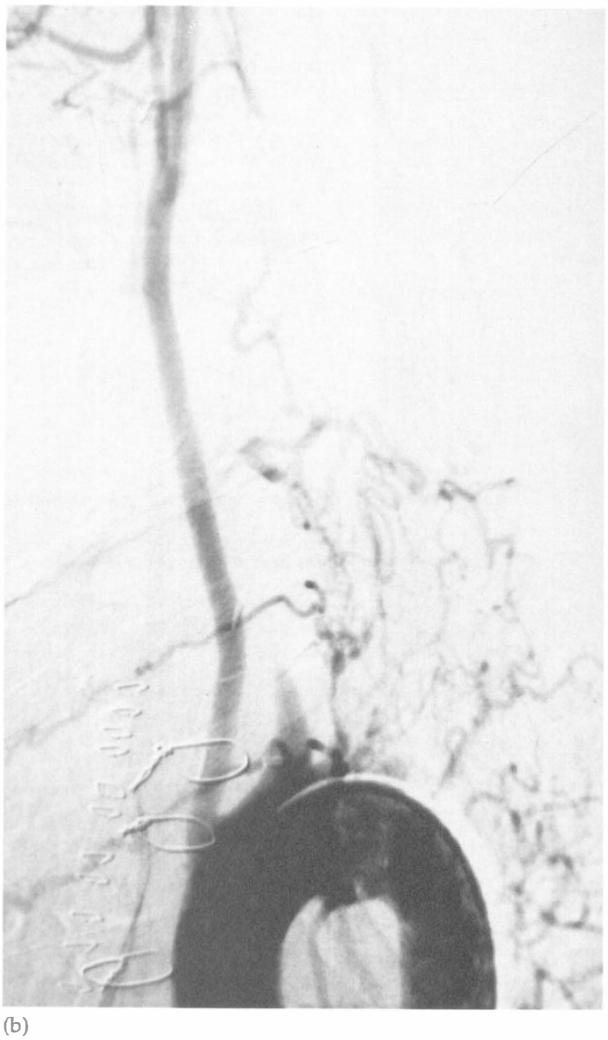
We report an atypical case of neovascular glaucoma occurring after arterial reconstructive surgery in a patient with previous normotensive Takayasu's retinopathy. In this case, treatment with panretinal photocoagulation prior to surgery could not prevent development of neovascular glaucoma.

Case report

A 32-year-old woman of North African origin was referred for ophthalmic assessment before vascular surgery for TA. Diagnosis of TA had been made 4 years earlier, when the patient presented with dizziness, upper limb claudication and absent pulsations of radial, brachial and carotid arteries. Three years later the patient developed left hemiplegia secondary to obstruction of the common carotid artery. After being treated with 30 mg of methylprednisolone and 100 mg of acetylsalicylic acid daily for more than 1 year, the patient was referred to our hospital for vascular surgical management. At admission, no blood pressure could be measured in either arm. Blood pressure as measured in both legs was 110/60 mmHg. The patient complained of severe headache and jaw pain while chewing. General physical examination was normal except for the sequelae of left hemiplegia. Arteriography showed an almost complete occlusion of the innominate artery, left



(a)



(b)

Fig. 1. *Aortography revealing (a) bilateral obstruction of the common carotid arteries and (b) surgical reperfusion of the right carotid.*

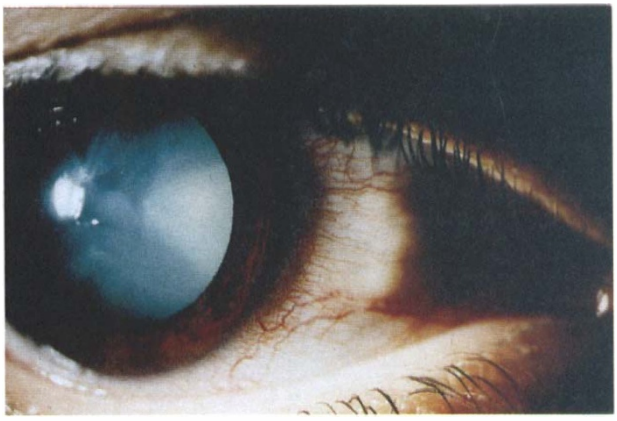


Fig. 2. *Rubeosis iridis and mature cataract, secondary to the ciliary hypoperfusion.*

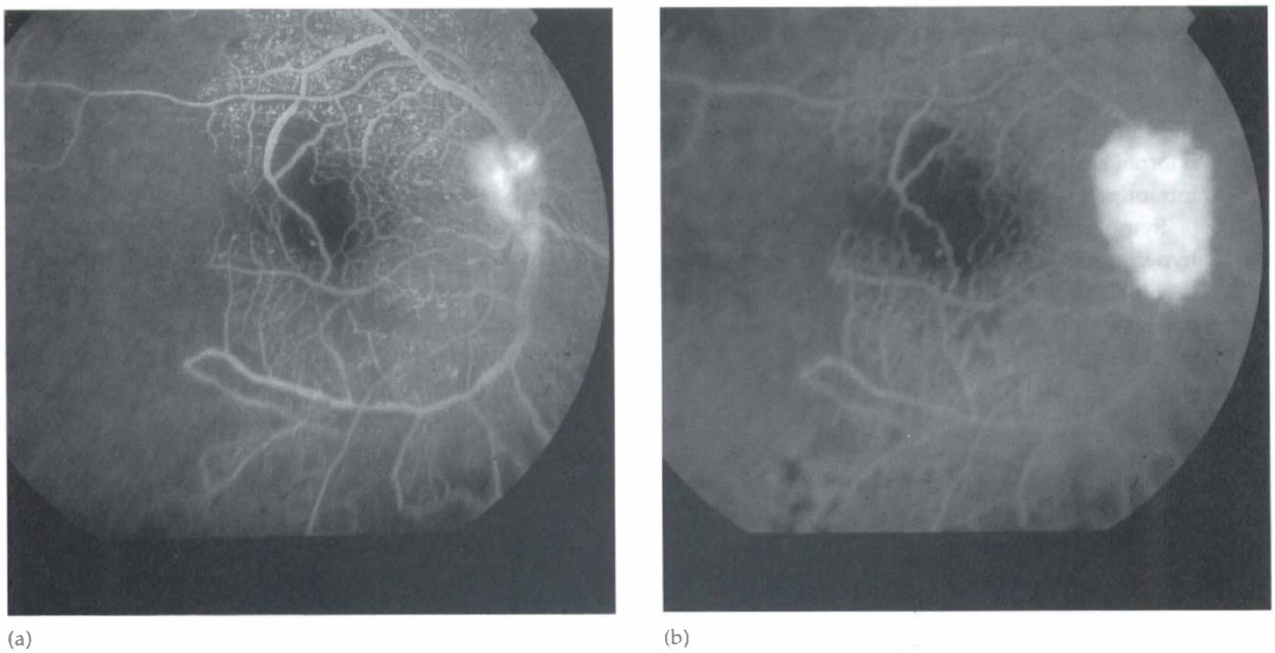
common carotid, left subclavian artery and right vertebral artery, and stenosis of the left vertebral artery (Fig. 1a).

The ophthalmic history revealed recent progressive painless visual loss in the right eye. Best corrected visual acuity was light perception in the right eye and 20/20 in the left eye. At the slip-lamp examination there were no dilated episcleral vessels; the corneas were clear and no inflammation was detectable in either anterior segment. Biomicroscopy disclosed severe right rubeosis iridis and a mature right cataract (Fig. 2). Gonioscopy found extensive anterior synechiae, and the right angle was almost occluded by neovascularisation. However, intraocular pressure as measured with Goldmann applanation tonometry was 10 mmHg in both eyes. The right fundus was not visible because of total cataract. Examination of the left eye found no iris neovascularisation, a wide open angle and no cataract; fundus biomicroscopy disclosed retinal ischaemia but no visible proliferative retinopathy.

In order to visualise the right retina, routine phacoemulsification and intraocular lens implantation was performed. However, post-operative visual recovery in the right eye was poor (20/200). Fluorescein angiography showed proliferative retinopathy in the right eye (Fig. 3a). The left retina was ischaemic, with microaneurysms, dilated retinal vessels, arteriovenous shunts as well as mild prepapillary neovascularisation. Arm-to-retina time was 24 s in the right eye and 15 s in the left eye. Because of the proliferative retinopathy, bilateral panretinal photocoagulation was rapidly completed, prior to the vascular reperfusion surgery.

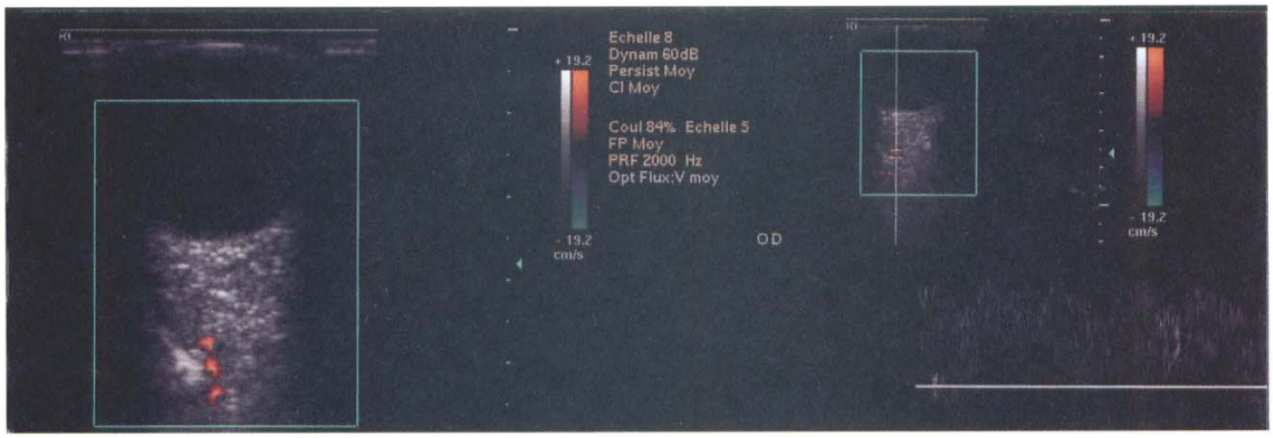
Additional information concerning the ocular haemodynamic changes was obtained using colour Doppler imaging of cervical and intracranial arteries before and after reconstructive surgery. Prior to surgery, the blood flow velocity in all the arteries was diminished. There was a dramatic decrease in the peak systolic velocities, predominating in the right carotid. However, the blood flow remained anterograde in the ophthalmic arteries (Fig. 4a).

Uneventful vascular surgery was performed 4 weeks after the complete bilateral retinal photocoagulation. It consisted in a right aorto-carotid bypass using the ipsilateral superficial femoral artery as an autograft. Post-operative aortography (Fig. 1b) showed a functional bypass. Nevertheless, 6 h after the surgery the patient complained of a red painful right eye. At examination, right rubeosis iridis was still present and intraocular pressure was 40 mmHg in the right eye. Gonioscopy showed an unchanged, almost closed right angle. Examination of the left eye remained unchanged. Fluorescein angiography of the right eye found leakage from the prepapillary vessels (Fig. 3b) that was not present pre-operatively.

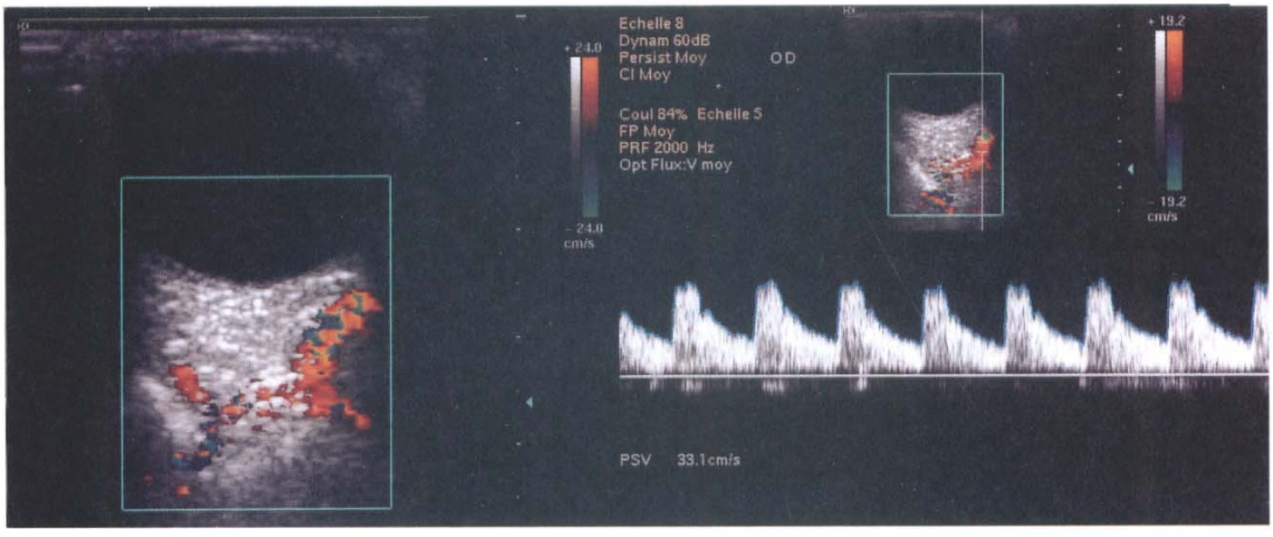


(a) (b)

Fig. 3. *(a) Pre-operative retinal ischaemia and (b) intense reperfusion of the preretinal neovessels and development of neovascular glaucoma.*



(a)



(b)

FIg. 4. *Colour Doppler imaging of the arterial flow in the ophthalmic artery before and after reperfusion surgery.*

Carotid surgery resulted in a dramatic increase in the blood flow in the right internal carotid artery, middle cerebral artery, and all the vessels of the ipsilateral orbit, confirmed with colour Doppler imaging. The blood peak systolic velocities increased in the right internal carotid artery from 32 cm/s pre-operatively to 123 cm/s post-operatively. In the right ophthalmic artery the peak systolic velocity (PSV) increased from 22 cm/s to 32 cm/s (Fig. 4b). Systolic and medium velocities of the left internal carotid, middle communicating artery and intraorbital arteries remained unchanged.

Symptomatic treatment of the right intraocular hypertension with systemic acetazolamide and topical timolol 0.5% lowered the intraocular pressure to 20 mmHg, which medically stabilised at the 6 week follow-up examination.

Comment

TA has an unknown origin, but an autoimmune aetiology is favoured, probably triggered by streptococcal or tuberculous infection.[3] Chronic inflammation in large arteries arising from the aorta results in stenosis and ischaemia. Prodromal pre-occlusive symptoms include fatigue, weight loss, myalgias and arthralgias. Later systemic symptoms include arm claudication, jaw claudication, transient ischaemic attacks and stroke. Physical examination may reveal absent pulses in the radial, brachial or carotid arteries. Therefore, blood pressure may not be detectable in the upper limbs.

Ophthalmic manifestations are considered to be late and inconstant occurrences in TA.[7] Functional symptoms such as transient obscurations when assuming an erect posture are described, due to cephalic hypoperfusion.[7] The most common ocular findings in patients with TA are related to the characteristic Takayasu's ischaemic retinopathy, found in 30% of patients.[8,9]

Studies of intraocular pressure in Takayasu's retinopathy show that in 50% of the eyes its value is less than 10 mmHg.[8] The intraocular pressure in TA decreases with the progression of the disease.[9] It has been postulated that low intraocular pressure at late stages of the disease might be the result of aqueous humour hyposecretion, due to ciliary hypoperfusion.[9] This may be an explanation for the low incidence of raised intraocular pressure in TA when iris neovascularisation occurs.

This report represents, to our knowledge, the first documented case of neovascular glaucoma occurring immediately after surgical repair of an obstructed carotid artery in TA. At admission, the patient presented with

typical ocular changes secondary to TA. Unilateral mature cataract associated with proliferative retinopathy was ipsilateral to the most severely obstructed carotid. Cataract formation, probably caused by reduction in lens nutrition secondary to reduced blood supply, is considered to be a rare event in TA.[2]

We used colour Doppler imaging of flow characteristics in the carotids, the intracranial and orbital arteries to assess haemodynamic changes induced by reconstructive surgery. Severe pre-operative reduction of the PSV in the ophthalmic artery was suggestive of high-grade (more than 70%) ipsilateral carotid stenosis. This finding is consistent with other studies conducted in patients with ocular ischaemic syndrome related to carotid occlusive disease.[10,11] Nevertheless, the marked reduction in PSV noted in both the ophthalmic arteries and the arteries of the circle of Willis, without ophthalmic flow reversal, suggested global hypoperfusion in the internal and external carotid arteries. Post-operative restoration of normal flow in the right carotid improved the flow in the ipsilateral ophthalmic artery and the middle cerebral artery. The flow in the left ophthalmic artery remained unchanged; the PSV in the left middle cerebral artery slightly increased, probably due to a functional circle of Willis.

In the ischaemic retinopathy induced by the ocular ischaemic syndrome, a multidisciplinary approach combining panretinal photocoagulation and reconstructive surgery may be beneficial.[12–14] In our case we performed panretinal photocoagulation prior to vascular reperfusion surgery. Panretinal photocoagulation is indicated to reduce the stimulus for the production of neovascularisation, and to decrease the risk of hypertensive neovascular glaucoma.[15] In our case, vascular surgery was delayed 4 weeks after the photocoagulation to allow scarring of the laser impacts. Despite this delay, the patient developed intraocular hypertension, ipsilaterally to the arterial graft. Endarterectomy has been reported as being responsible for neovascular glaucoma in patients with carotid occlusive disease, because of sudden reperfusion of the ciliary body.[15] Surprisingly, in previous reports of vascular bypass surgery in patients with Takayasu's disease, intraocular pressure did not rise after surgery, even though prior retinal laser photocoagulation was not performed.[5,7,16]

A possible explanation for the occurrence of neovascular glaucoma in our patient might be that she was operated on at a very advanced stage of the disease, and the right angle was already compromised by the presence of rubeosis. Post-operative carotid perfusion resulted in sudden and massive raise of ciliary secretion while engorgement of vessels in the angle caused additional obstruction in the impaired meshwork. Further observations are needed to establish the potential risk factors of developing intraocular hypertension after restoration of arterial carotid perfusion in eyes with ischaemic ocular syndrome in TA.

References

1. Takayasu M. Case of unusual changes of the central vessels in the retina. Acta Ophthalmol Jpn 1908;12:554.
2. Bapat V, Tendolkar AG. Turning a blind eye. Lancet 1997;349:1740.
3. Schmidt M, Fox AJ, David N. Bilateral anterior ischaemic optic neuropathy as a presentation of Takayasu's disease. J Neuro-ophthalmol 1997;17:156–61.
4. Bodker FS, Tessler HH, Shapiro M. Ocular complications of Takayasu's disease in a hispanic woman. Am J Ophthalmol 1993;115:676–7.
5. Tanaka T, Shimizu K. Retinal arteriovenous shunts in Takayasu's disease. Ophthalmology 1987;94:1380–8.
6. Karwatowski W, Jampol L, Mani H, Weinreb RN. Neovascular glaucoma in Takayasu's disease. Retina 1995;15:353–4.
7. Lewis JR, Glaser JS, Schatz NJ, Hutson DG. Pulseless (Takayasu's) disease with ophthalmic manifestations. J Clin Neuro-ophthalmol 1993;13:242–9.
8. Sagar S, Kar S, Gupta A, Sharma B. Ocular changes in Takayasu's arteritis in India. Jpn J Ophthalmol 1994;38:97–102.
9. Uyama M, Asayama K. Retinal vascular changes in Takayasu's disease (pulseless disease), occurrence and evolution of the lesion. International symposium on fluoroescence angiography, Ghent, 28 March–1 April 1976.
10. Ho AC, Lieb WE, Flaharty PM, Sergott RC, Brown GC, Bosley TM, Savino PJ. Colour doppler imaging of the ocular ischemic syndrome. Ophthalmology 1992;99:1453–62.
11. Mawn LA, Hedges TR, Rand W, Heggerick PA. Orbital colour imaging in carotid occlusive disease. Arch Ophthalmol 1997;115:492–6.
12. Johnston ME, Gonder JR, Canny CL. Successful treatment of the ocular ischemic syndrome with panretinal photocoagulation and cerebrovascular surgery. Can J Ophthalmol 1988;23:114–9.
13. Sivalingan A, Brown GC, Margargal LE. The ocular ischemic syndrome: visual prognosis and the effect of treatment. Int Ophthalmol 1991;15:15–20.
14. Biousse V. Carotid disease and the eye. Curr Opin Ophthalmol 1997;8:16–26.
15. Nguyen QH, Hamed LM, Sherwood MB, Roseman RL. Neovascular glaucoma after carotid endarterectomy. Ophthalmic Surg Lasers 1996;27:881–4.
16. Didier T, Brasseur G, Charlin JF, Hufault D, Langlois J. Artérite de Takayasu: évolution des signes ophtalmologiques après greffe vasculaire. A propos d'une observation. Bull Soc Ophtalmol Fr 1986;86:909–11.

D. Milea
N. Cassoux
P. Lehoang
Department of Ophthalmology
Hôpital de la Pitié-Salpêtrière
Paris, France

S. Gerber
C. Marsault
Department of Neuroradiology
Hôpital de la Pitié-Salpêtrière
Paris, France

J.-C. Piette
Z. Amoura
Department of Internal Medicine
Hôpital de la Pitié-Salpêtrière
Paris, France

E. Kieffer
Department of Vascular Surgery
Hôpital de la Pitié-Salpêtrière
Paris, France

Phuc Lehoang, MD, PhD ✉
Service d'Ophtalmologie
Hôpital de la Pitié-Salpêtrière
47–83 Boulevard de l'Hôpital
F-75651 Paris Cedex 13, France

Fax: +33 1 42 16 32 01