elevation of tissue pressure sufficient to displace the weakened lesion anteriorly.[12]

This case is interesting as the presentation of PCV was precipitated by a bout of forceful sneezing. A broadly similar case was reported by Rosa *et al*[13] whereby the patient was diagnosed to have PCV, and at a later stage developed massive retinal and subretinal haemorrhage following an episode of severe vomiting. In our case, it is possible that a clinically silent lesion may have been present prior to her sneezing event. She did have risk factors, hypertension, and soft drusen. We postulate that an inner choroidal vascular abnormality possibly associated with hypertensive stress in this patient was further aggravated by the Valsalva effect of sneezing, and may have contributed to the aneurysmal bulging of the choroidal arteries.

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Sir,

Spontaneous regression of optic disc neovascularization in Takayasu Arteritis

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A female patient was identified with Takayasu arteritis owing to the presence of disc neovascularization. However, regression of neovascularization may occur without treatment, as in this case, presumably because of vascular remodelling. This phenomenon has not been previously reported.

Case report

A 49-year-old Caucasian female was seen in the ophthalmic department with a 5-year history of diplopia, neck pain, pins and needles in her fingers, and weakness in her arms particularly on lifting. During this time, a vascular surgeon in another centre had discovered right iliac artery disease. An MRI of her carotids and a cervical X-Ray were reported as normal. It was thought that she had severe atherosclerotic disease that was aggravated by her smoking.

She moved house and her new GP discovered that he was unable to find a pulse or blood pressure in either arm. Owing to her varied symptoms, she was referred to the local rheumatology and ophthalmology departments. Carotid, subclavian, and aortic bruits were found. Her arms and right proximal leg pulses were absent. Blood tests showed a normal ESR and haemoglobin, and a raised cholesterol. During her visit to the ophthalmic department, fundal examination revealed neovascularization of her right optic disc and bilateral attenuated arterioles, no other ischaemic signs were seen.

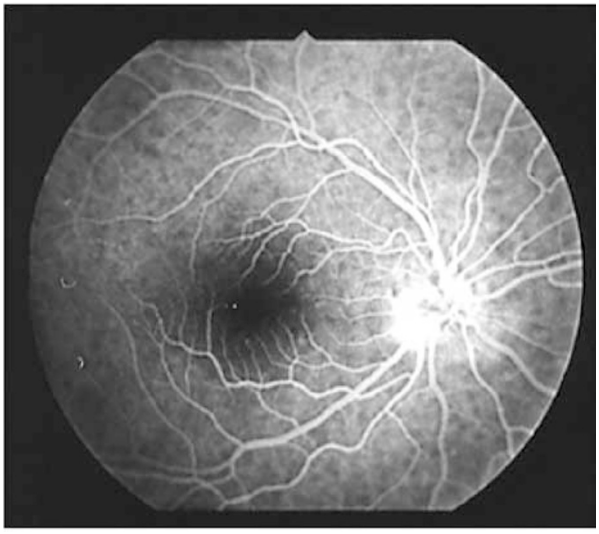

Figure 1 Fluorescein angiogram at 43 s shows disc new vessels.

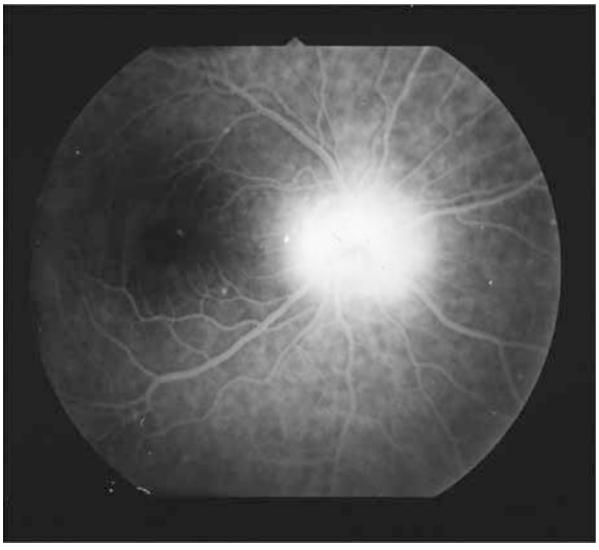

Figure 2 Fluorescein angiogram at 5 min shows new vessel leakage at the disc.

The retinal vasculature collapsed in the right eye with gentle digital pressure, although formal ophthalmodynamometry was not performed. A diagnosis of Takayasu arteritis was made. Fluorescein angiography showed late venous filling and profound leakage from new vessels at the right disc, see Figures 1 and 2.

A magnetic resonance angiogram confirmed the diagnosis of Takayasu's arteritis; it demonstrated that almost all the arteries originating from the aorta were affected. The innominate artery had a subtotal occlusion, the left carotid and left subclavian had occlusions at their origin, the vertebral artery showed reverse flow, the right carotid and subclavian were severely stenosed. The superior mesenteric was occluded, as was the right common iliac. The left common iliac had a 50% stenosis. No active treatment has been instituted by the vascular team thus far as it was felt that the condition is presently inactive.

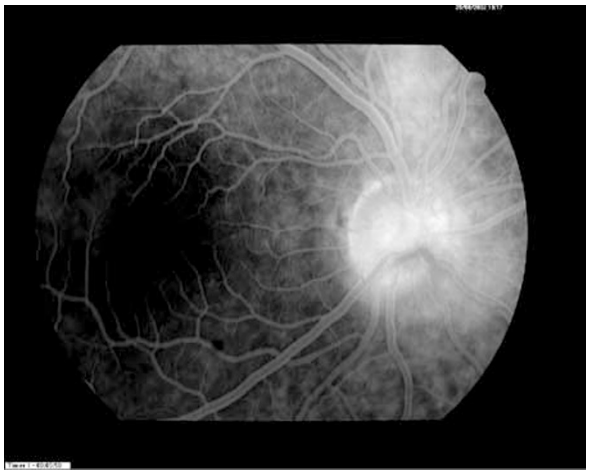

Figure 3 Fluorescein angiogram at 5 min 58 s shows considerable regression of disc leakage, taken 10 months after Figures 1 and 2.

At her most recent examination, there was significant regression of her neovascularization in the absence of any treatment (Figure 3). However, the venous filling time was still delayed. She continues to undergo periodic examination.

Comment

Takayasu arteritis is an extremely rare condition with an occurrence rate of about two per million. It typically affects young people, the symptoms usually begin before the age of 40. Asians are more commonly affected and the male-to-female ratio is 1 : 9. The diagnosis is often delayed; one study commented that there was a median delay of diagnosis of 10 months from the onset of signs of large vessel inflammation.[1] It is a chronic inflammatory disorder that affects the large blood vessels such as the aorta and its branches. It is a continuous or patchy granulomatous inflammation in the vessel media. In the active phase, there are lymphocytes, histocytes, and multinucleated giant cells. During the chronic phase sclerosing fibrosis appears. It causes vessel narrowing, occlusion, and aneurysm formation. In the eye, corneal oedema, anterior chamber cells, retinal cotton wool spots, haemorrhages, microaneurysms, arteriovenous malformations, ischaemic optic neuropathy, iris and

posterior segment neovascularization, and subsequent vitreous haemorrhage may occur.

This condition is frequently divided into two phases that often overlap. Early symptoms are systemic and nonspecific. These include fever, poor appetite, fatigue, weight loss, joint pains, and night sweats. During this time many patients have a raised ESR, this was found in 72% of one group of patients with active disease, and in 56% of those with inactive disease.[2] There are no specific markers for the disease.[3] The gold standard test is vessel biopsy, but this is only possible if surgical intervention is needed. Therefore, disease activity is difficult to establish. Some patients that are classified as in remission may not be; one publication reported that histology specimens showed that 44% of those presumed to be in remission were not.[3] Occlusive symptoms can occur later, secondary to narrowing of the arteries.

The condition can be confirmed by radiographic means such as arteriography, computerized tomography, and magnetic resonance imaging.

A number of options exist to control this disorder. Initially, prednisolone can be instigated; if this alone is unsatisfactory then additional drugs such as methotrexate, azathioprine, and cyclophosphamide can be used. With steroid treatment alone, one study observed that remission was achieved in 60% of patients but half of these subsequently relapsed. With additional cytotoxic drugs remission was accomplished in 40%. However, 23% of treated patients never attained remission.[1] Various mortality rates have been stated; a group of 120 patients studied had a rate of 82.9% over 15 years.[4] Optic complications can improve with systemic therapy, carotid endarterectomy and pan retinal photocoagulation may be considered. Spontaneous regression of new vessels as in this case has not been previously reported.

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Sir,

Marcus Gunn syndrome

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The Marcus Gunn syndrome, described by Gunn in 1883, is one of the more common congenital oculofacial synkineses, and accounts for approximately 5% of all congenital ptosis.[1]

Typically, the infant is noted soon after birth to have a ptotic lid, which elevates spontaneously when sucking or chewing. The lid elevates on contraction of the lateral (external) pterygoid muscle, which mediates opening, forward, and contralateral movement of the jaw. Less commonly, the lid elevates upon closure of the mouth, which is mediated by the medial (internal) pterygoid. A very rare variant, 'inverse' Marcus Gunn syndrome is characterized by the lid falling on opening the mouth, because of inhibiton of the levator muscle in association with lateral pterygoid contraction. Although bilateral cases of classical Marcus Gunn are well documented, there are, to our knowledge, only two previous reports of a patient with the classical syndrome on one side and an inverse Marcus Gunn synkinesis on the other.[1,2] This report describes the case of a 4 year-old boy with what could be described as 'see-saw' Marcus Gunn syndrome.

Case report

A 4-year-old boy, presented with a right congenital ptosis. His mother was concerned about the cosmetic appearance of the lid as well as anomalous movements of the lids when eating. The child was otherwise well, had no history of other ocular problems, and there was no family history of similarly affected individuals.

Examination showed his visual acuities to be 6/9 right 6/6 left. He had a 3–4 mm right ptosis, with typical Marcus Gunn syndrome on the right side, the lid elevating to the normal position with jaw opening. On the left side, the lid height was normal at rest, but became ptotic when the mouth was opened (Figure 1). There was no contraction of the orbicularis oculi. This resulted in