LETTERS TO THE EDITOR

Gaze evoked amaurosis in dysthyroid orbitopathy

EDITOR,—Gaze evoked amaurosis is an uncommon symptom usually associated with intraconal masses such as cavernous haemangioma[1] or optic nerve sheath meningioma.[2 3] Patients describe transient loss of vision in eccentric positions of gaze with full recovery of vision on returning to the primary position. The cases previously published have all been unilateral. We describe a case of bilateral gaze evoked amaurosis in a patient with dysthyroid orbitopathy.

CASE REPORT

A 62 year old smoker gave a 12 month history of transient bilateral loss of vision on upgaze associated with supraorbital discomfort. His hobby was flying radio controlled model aeroplanes but after crashing two of these during amaurotic episodes he presented to an ophthalmologist. Two years previously he had required admission for cardiac failure associated with thyrotoxicosis but after treatment with iodine-131 and carbimazole his thyroid status had been stabilised.

Ophthalmic examination revealed symmetrical axial proptosis (26 mm), restriction of upgaze, and lid signs consistent with dysthyroid orbitopathy. In primary position the corrected distance acuities were 6/6 in both eyes with 17/17 Ishihara colour plates seen and full visual fields. On upgaze the distance acuities were reduced to less than 6/60 with none of the Ishihara colour plates seen. The intraocular pressures (IOP) increased from 20 mm Hg (both eyes) in primary position to 30 mm Hg (right eye) and 31 mm Hg (left eye) in elevation. Pupil examination using infrared pupillography showed 0.20 mm dilatation of both pupils and 15% reduction in light reflex amplitude on upgaze compared with the primary position. The disc and fundal appearances were normal and did not change with direction of gaze. On general examination the patient had pretibial myxoedema but appeared clinically euthyroid.

The pattern VEP in both eyes was normal in the primary position but reduced in amplitude on upgaze (Fig 1A). Fluorescein angiography showed normal perfusion of the optic disc and retina both in primary position and in upgaze. Orbital computed tomography revealed moderate enlargement and oedema in all extraocular muscles; no tumour or other mass lesion was present (Fig 2). The patient was treated with oral steroids (prednisolone 40 mg/day). One month later there was symptomatic improvement, 2–3 mm less proptosis, and the distance acuities had improved to 6/36 (right eye) and 6/60 (left eye) on upgaze. There was no change in the IOP rise associated with elevation of the eyes. The pupils dilated less on upgaze (0.05 mm) and there was less attenuation of the light reflex amplitude (5%) compared with before treatment. The pattern VEP showed improvement on upgaze (Fig 1B) compared with before treatment. The steroid dose was tapered off over the next 6 months with no relapse of his symptoms. The patient has now returned to his hobby of flying model aeroplanes.

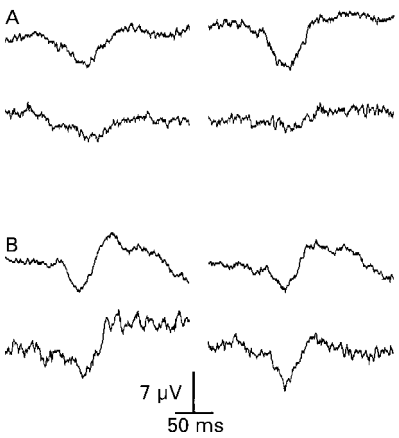

Figure 1 Pattern VEPs from the left eye (left) and from the right eye (right) before treatment (A) and after treatment (B). In each case the upper trace was recorded with the eyes in primary position, the lower trace with the eyes in upgaze.

Figure 2 Computed tomograph of orbits (coronal view).

COMMENT

The visual loss in this patient was transient, reversible, and related to the position of the globe in the orbit. The only orbital pathology found on imaging was enlargement of the extraocular muscles related to his dysthyroid orbitopathy. The mechanism of this visual failure remains intriguing. Its rapid onset and reversibility suggest vascular compromise but fluorescein angiography showed normal disc and retinal perfusion on upgaze. Vascular compromise of the retrobulbar optic nerve cannot be ruled out. Of interest, in the rat model retrobulbar ischaemia produces a depolarising conduction block which takes minutes rather than seconds to develop and which would generate photopsia before the visual failure.[4] In previous reports, raised IOP has been invoked as the cause of the visual loss.[5] However, in our patient the rise in IOP was only moderate and persisted after steroid treatment whereas the vision improved. A third possibility is that compression of the optic nerve by enlarged extraocular muscles or stretching of the nerve as a result of dural tethering produced a mechanical conduction block in upgaze. This has been described in peripheral nerves[6] but it classically takes several days for full recovery of function; whether optic nerve axons behave similarly is not known. Gaze evoked amaurosis has not been previously described in thyroid eye disease but was noticed by this patient because it interfered with his hobby of flying radio controlled model aeroplanes.

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Epiphora due to Kaposi's sarcoma of the nasolacrimal duct

EDITOR,—Excessive watering of the eye may be due to lacrimation, mechanical obstruction of the tear drainage, or lacrimal pump failure. We report a case of obstructive epiphora due to Kaposi's sarcoma of the nasolacrimal duct.

CASE REPORT

A 34 year old man who was recently diagnosed as HIV seropositive was referred to the eye department complaining of watering of his left eye. At the time of diagnosis Kaposi's sarcoma lesions were noted on his face, left lower lid, and scalp and the patient was commenced on triple antiretroviral therapy (zidovudine, lamivudine, saquinavir). On examination a Kaposi's sarcoma lesion of the left lower lid with swelling over the lacrimal sac was noted (Fig 1). The patient had a left sac washout, which confirmed left nasolacrimal duct obstruction. At the time of sac washout a blood stained nasal discharge was noted. Examination of the left nostril showed a raised lesion involving the nasal septum, inferior turbinate, and the nasolacrimal duct. Biopsy under local anaesthesia was performed and examination of the specimen confirmed the diagnosis of Kaposi's sarcoma (Fig 2). The patient was commenced on liposomal daunorubicin and his symptoms of epiphora resolved completely.

COMMENT

Epidemiological evidence suggests that Kaposi's sarcoma is caused by a transmissible

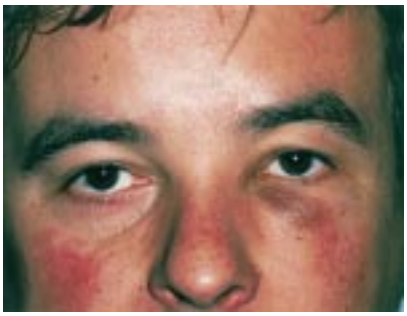

Figure 1 The left eye showed mild periocular swelling and skin discoloration consistent with Kaposi's sarcoma.

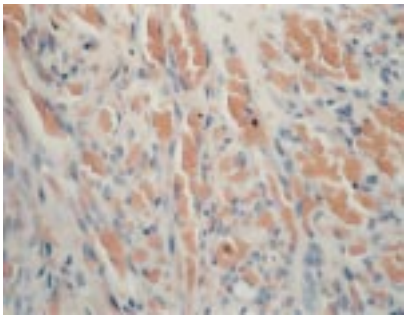

Figure 2 Histological section shows a cellular spindle cell lesion forming vascular channels within the mucosa (Kaposi's sarcoma). (Haematoxylin and eosin stain, original magnification ×250.)

agent.[1] Simpson *et al* considers Kaposi's sarcoma associated herpes virus (herpes virus 8) to be a transmissible cofactor in the pathogenesis of Kaposi's sarcoma.[2]

Kaposi's sarcoma may give rise to the symptoms of epiphora if nasopharyngeal involvement obstructs the nasolacrimal duct. The treatment options include local radiotherapy and systemic cytotoxic chemotherapy. Radiotherapy is likely to lead to a cicatrising course of the external eye and lacrimal outflow system. Systemic therapy with liposomal daunorubicin led to resolution of the symptoms of epiphora in this individual and may be considered the first line therapeutic approach.

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Ochrobactrum anthropi endophthalmitis after vitreous surgery

EDITOR,—*Ochrobactrum anthropi* is a non-fermentative, motile, strictly aerobic, oxidase positive Gram negative bacillus.[1] In 1980, the first case of human infection with *O anthropi* was described.[2] Since then, there have been some reports and this bacillus has been considered as a possible cause of opportunistic infection. There are only two reports of *O anthropi* endophthalmitis, one was metastatic endophthalmitis in a patient with a central venous catheter,[3] and the other was after cataract surgery.[4] We describe a case of unilateral endophthalmitis caused by *O anthropi*, which was diagnosed after two vitreous surgery procedures.

CASE REPORT

A 64 year old man complained of visual loss in his left eye in January 1998. He was diagnosed with uveitis and treated with oral prednisolone, topical betamethasone and atropine, and subconjunctival injection of dexamethasone. As the inflammation had not resolved, he was transferred to our institution. He had a medical history of bacterial endocarditis caused by *Streptococcus haemolyticus* in April 1997 and underwent placement of a central venous catheter for 1 month. Mitral valvuloplasty had been performed in October 1997.

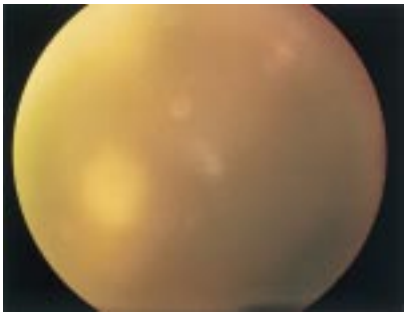

Figure 1 Vitreous haze with a lobulated white mass in the inferior vitreous.

His visual acuity was right eye 20/20 and left eye 20/100. The left eye had anterior chamber inflammation with flare (1+) and cells (2+), keratic precipitates, and prominent vitritis with a lobulated white mass. The right eye was normal. A clinical diagnosis of fungal endophthalmitis was made in the left eye. Medication was changed to intravenous fluconazole, topical betamethasone, and subconjunctival injection of dexamethasone, but vitreous haze was still present with this treatment (Fig 1). A pars plana vitrectomy with removal of the lens and intravitreal fluconazole irrigation was performed on 14 April 1998. The next day he had severe pain in his left eye and headache. Left visual acuity reduced to light perception, intraocular pressure was 42 mm Hg, and marked inflammation with hypopyon was observed. As a bacterial endophthalmitis was suspected, he underwent the second vitrectomy on 16 April 1998 with intravitreal imipenem irrigation. Vitreous cultures grew *O anthropi*. The isolate was sensitive to cefmetazole, cefbuperazone, imipenem, minocycline, levofloxacin, gentamicin, tobramycin, and amikacin, and resistant to ampicillin, piperacillin, cefazolin, cefotiam, flomoxef, and ceftazidime. He was treated with intravenous imipenem, oral minocycline, and ciprofloxacin, successively, and the intraocular inflammation subsided. Four months after the second vitrectomy, his left visual acuity was 20/30.

COMMENT

The natural habitat of *O anthropi* has not yet been established. It is commonly found in environmental and hospital water sources.[1 2] This organism has been isolated from clinical specimens, including blood, urine, faeces, and sputum. Most cases of *O anthropi* sepsis were reported to relate to indwelling catheters for venous access or other permanent medical devices.[5–7] As for the infectious routes, there are two possibilities in our case. One is contamination during mitral valvuloplasty. Indeed, a lobulated white mass in the vitreous seen before the first vitrectomy (Fig 1) is similar to that in the case of Berman *et al*.[3] In the past, however, *O anthropi* endophthalmitis occurred within 3 weeks after placement of a central venous catheter.[3] Endophthalmitis occurred in our case more than 70 days after the mitral valvuloplasty. Moreover, *O anthropi* was detected from in the vitreous sample only at the second vitreous procedure. Accordingly, contamination in our case may have been caused during the first vitreous surgery procedure. Bacterial endophthalmitis after vitreous surgery is very rare; its frequency is about 0.2%.[8 9] The main organisms causing endophthalmitis are *Pseudomonas aeruginosa*, *Staphylococcus epidermidis*, and *S aureus*.[8 9] However, one should look out for infections induced by attenuated bacteria such as *O anthropi* after vitrectomy.

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Scleral perforation following trans-scleral cyclodiode

EDITOR,—In recent years, the diode laser utilising 810 nm wavelength has emerged as an increasingly popular and effective tool for treating severe cases of glaucoma which are resistant to other conservative or surgical therapeutic options.[1 2] The desired effect (as with other lasers used in this field) is thermal heating and coagulation necrosis of the ciliary epithelium (laser cyclophotocoagulation). However, the laser scleral transmission is increased by the contact method (compared with the non-contact method), allowing for less total energy application while obtaining the same desired effect.[3]

The side effects of trans-scleral cyclodiode laser range from common ones such as mild iritis[4] to rare ones including phthisis bulbi.[1] To the best of our knowledge, there has only been one reported case of scleral perforation due to trans-scleral diode cyclophotocoagulation. This was following contact delivery of the laser using the original quartz G-probe (diameter 600 μm) and settings of 2 W for 2 seconds per application.[5] The patient in question had scleral thinning following previous cataract surgery. It was thought that the sharp edge of the probe had cut conjunctival vessels causing bleeding and contamination of the probe head. Thin adherent debris was then carbonised allowing the laser tip temperature to rise to 300°C, sufficient to cause scleral perforation. This case report led to the redesigning of the laser probe tip in order to protect the vascular structures from its sharp edges.

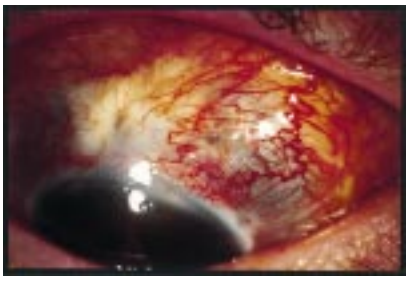

Figure 1 Post laser photograph showing the extensive severe superonasal scleral thinning which existed before application of cyclodiode laser. The blue Vicryl suture is just visible to the left of centre of picture overlying the zone where the perforation occurred.

CASE REPORT

We would like to report a second case of scleral perforation following contact trans-scleral cyclodiode treatment. It concerns a 37 year old white man with buphthalmos whose left eye had been enucleated at the age of 13. His other ocular history included right trabeculectomy (age 4 years) and right giant retinal tear repair with vitrectomy, silicone oil, and 360° indirect laser (age 35 years). He had severe scleral thinning through 180° superiorly (see Fig 1), thought to have been caused by his buphthalmos and previous ocular surgery. Despite maximal medical therapy and removal of the silicone oil, his intraocular pressure was poorly controlled (>30 mm Hg). His best corrected visual acuity in the right eye had dropped from 6/36 in 1979 to 6/60 in 1998. This eye was treated with the currently marketed contact G-probe attachment of the Iris medical diode laser (Mountain View, CA, USA) using 14 applications of 2 seconds' duration and 2 W power each. The eye was transilluminated before application of the laser but ciliary body identification was difficult due to the extensively thinned sclera. There were three audible "pops" from the first 13 applications, some of which were applied to the thinned scleral zones and following the last planned application at the superonasal limbus, a gush of aqueous was seen. Closer microscopic inspection confirmed a round, "punched out" full thickness perforation through conjunctiva, sclera, and choroid immediately posterior to the laser site. This defect was closed with two 10.0 Vicryl (Ethicon) sutures. Following the perforation, the patient developed a large choroidal haemorrhage (confirmed on ultrasound). A week after the laser, the scleral leak recurred and was successfully sealed using three 10.0 nylon (Alcon) sutures. Eight weeks later, his intraocular pressure was well controlled (10 mm Hg) using topical levobunolol and oral acetazolamide and the choroidal haemorrhage had resolved. The scleral wound has remained closed with no further leaks.

COMMENT

Trans-scleral "cyclodiode" photocoagulation has emerged as an effective method of controlling intraocular pressure and pain in refractory glaucoma. With its increasing use, more patients may be at risk of so far rare, but significant, complications. To the best of our knowledge, this report is the first case of scleral perforation using the new contact G-probe attachment.

Scleral perforation appears therefore to be a rare but significant complication of contact trans-scleral cyclodiode treatment. In both this and the previously reported case, pre-existing scleral thinning was the common risk factor. Parma *et al* [6] looked at the effect of cyclodiode therapy on cadaver eyes. They showed that approximately 40% less energy is needed to achieve ciliary photocoagulation in thin sclera (that is, half to a third full thickness) compared with normal thickness sclera.

Our patient was being treated as part of a standard protocol in use in our unit as part of a prospective trial of cyclodiode therapy. We had previously treated other eyes with thin sclera without incident at the energy levels utilised for the case described above although none has had such extensive thinning as this case. Similarly thinned areas in the same eye had been treated before the event which occurred on the final scheduled application. No conjunctival haemorrhages had been noted before the perforation and the probe tip had not been inspected between applications. It is difficult to know whether the perforation in our case was due to mechanical pressure, carbonisation of debris at the laser tip, or both. We would now warn against treating areas of an eye with severe scleral thinning as there are no known "correction factors" that can be utilised at present. If treatment is absolutely necessary, a lower laser power setting should be used (we would suggest 50%) and minimal pressure applied with the G-probe. Furthermore, care should be taken to ensure that the probe tip is clean before each application in such eyes in order to prevent carbonisation of debris.

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Globe perforation during botulinum toxin injection

EDITOR,—Botulinum neurotoxin A, when injected into striated muscle, prevents acetylcholine release causing flaccid paresis of the muscle.[1] Scott termed this procedure "chemodenervation" and introduced the technique for treatment of strabismus in 1979.[2] The procedure has been reported to be safe with no systemic and minimal local complications. Complications commonly reported following botulinum injection to horizontal muscle are ptosis and vertical muscle involvement.[3] We report a case of globe perforation following botulinum injection into the medial rectus muscle in a woman with high myopia.

CASE REPORT

A 46 year old woman presented with a 6 year history of diplopia, worse on dextroversion. Prism treatment by her optometrist had partly alleviated her symptoms. She was found to have bilateral abduction weakness, worse on the right side. She was a high myope wearing a −19.5 D lens in her right eye and −17 D in her left eye. She was normotensive, normoglycaemic, and had no neurological abnormalities on clinical examination. A decision was made to perform botulinum injection to her right medial rectus muscle to achieve temporary, or possibly longer lasting, symptomatic improvement.

After informed consent the eye was anaesthetised with 1% amethocaine hydrochloride eye drops. Under electromyographic (EMG) control a 27 gauge monopolar needle was advanced into the medial rectus muscle with no undue resistance. A good EMG response was obtained from the muscle. The signal increased on adduction and decreased on abduction of the eye. Botulinum toxin 2.5 units was injected. The injection was performed by a surgeon experienced in using the technique (BWF).

Immediately after treatment the patient complained of red floaters in the right eye "like drops of blood". Fundus examination revealed a small vitreous haemorrhage with a small retinal haemorrhage in the nasal retina at the equator (Fig 1). There was no obvious retinal tear. A diagnosis of globe perforation was made. There was poor uptake of laser energy by the atrophic retinal pigment epithelium and cryotherapy was therefore performed to the probable entry site. On follow up the retina remained flat. The motility response to the botulinum injection was poor. Paralysis of accommodation and dilatation of the pupil developed which resolved 10 months after the procedure.

COMMENT

Botulinum injection is regarded as a procedure with a low incidence of morbidity.[4] Post injection ptosis and diplopia are transient complications. No eye has been reported to have lost vision as a result of botulinum injection. Accidental perforation of the globe is an acknowledged complication of peribulbar and retrobulbar anaesthesia[5]) and strabismus surgery.[6] High myopia is a strong risk factor for globe perforation in peribulbar anaesthesia.[7] Our case shows that globe perforation can occur with botulinum injection as with any other periocular surgical procedure.

In our patient the injection was performed by an experienced surgeon and the procedure

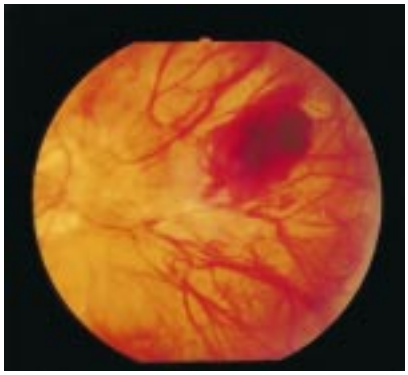

Figure 1 Disc and nasal retina of the right eye showing retinal haemorrhage following botulinum injection.

was performed in a standard fashion using EMG control. Before injection there was good muscle signal. It has been shown that EMG signals may be recorded in most cases once the needle contacted the conjunctiva.[8] Using a monopolar electrode needle, the reference electrode is located centimetres away from the active electrode and the needle can record signals that are closer to the needle electrode than the reference electrode even if they are several millimetres away from the needle electrode. In our patient it is likely that the tip of the needle was in the vitreous cavity at some stage during the procedure and an EMG response was present at that time. The most common potentially vision impairing complication of globe perforation is retinal detachment. Retinal detachment has been reported in globe perforations associated with peribulbar anaesthesia and after strabismus surgery.[9] The theoretical risk of causing a globe perforation is greater with botulinum injection into an extraocular muscle than it is with peribulbar injection. The needle enters the muscle just behind the insertion and the sclera is at its thinnest at this part (0.3 mm). Any movement of the eye by the patient, with the needle in this position may result in an inadvertent perforation of the globe, especially in patients who are likely to have thin sclera, such as high myopes. Demonstration of increased signal by movement of the eye into the field of action of the muscle to be injected should probably be avoided for this reason.

Management of patients with scleral perforations is controversial. Some authors recommend that they should be treated with indirect diode laser or transscleral cryotherapy regardless of the depth of perforation, to reduce the incidence of retinal detachment.[9] However, animal experiments have found a higher incidence of retinal detachment following heavy cryotherapy,[10] and suggest cryotherapy should be used only if there is vitreous haemorrhage or the patient has a predisposing risk factor for retinal detachment. Our patient was a high myope and she had a small retinal and vitreous haemorrhage which increased her risk of developing a detachment. Globe perforation, although rare, is a complication that can occur with botulinum injection into an extraocular muscle and surgeons doing the procedure and their patients should be aware of this. The risk is higher in myopic eyes, as the equator of the globe is more posterior than usual, and the sclera thinner.

Botulinum toxin injection to an extraocular muscle should be approached with extreme caution in highly myopic eyes, and all movement of the eye should be avoided during the procedure.

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Diagnosis of corticosteroid resistant ocular sarcoidosis by chorioretinal biopsy

EDITOR,—Sarcoidosis, a multisystem granulomatous disease, involves the eye in approximately 25% to 50% of patients[1 2] and produces numerous ocular findings.[3] Definitive diagnosis requires a non-caseating granuloma in the absence of mycobacterial infection on biopsy. Although most patients have abnormal chest *x* rays, ocular sarcoidosis can precede pulmonary involvement, making diagnosis difficult. The following patient with ocular sarcoidosis—unresponsive to corticosteroids and cyclosporine—was diagnosed only after chorioretinal biopsy; she responded to azathioprine therapy.

CASE REPORT

A 32 year old white woman had a 12 year history of granulomatous uveitis in both eyes, resistant to treatment with topical, periocular, and systemic corticosteroids, as well as systemic cyclosporine. The patient presented with intermediate uveitis of the left eye, complicated by cystoid macular oedema, posterior subcapsular cataract, and epiretinal membrane formation. In 1986, she underwent lensectomy, vitrectomy, and membrane peeling with poor visual outcome. Laboratory evaluation, including chest *x* ray and serum angiotensin converting enzyme level, was normal.

Figure 1 Retinal photograph of the left eye shows substantial vitreous haze. Scattered lesions at the levels of the deep retina and choroid (arrows), as well as mottling of retinal pigment epithelium, are seen in the posterior pole.

In 1991, she developed active uveitis in the right eye with visual acuity of 20/25; cystoid macular oedema developed, with scattered lesions in the deep retina. The left eye decreased to hand movement. Both eyes showed moderate anterior uveitis, vitritis, and chorioretinal lesions (Fig 1), and a diagnosis of multifocal choroiditis was made.

Chorioretinal lesions increased in size and number in both eyes, despite cyclosporine, and vision in the right eye worsened to 20/60. She developed peripheral retinal detachment, and a chorioretinal biopsy was performed on the left eye.

Biopsy examination demonstrated a well defined, non-caseating choroidal granuloma (Fig 2), predominantly macrophages surrounded by CD4+ T lymphocytes and a few plasma cells. The retina was gliotic, infiltrated with scattered lymphocytes and plasma cells. Higher expression of Fas, FasL, and Bax, as well as lower expression of Bcl-2 and DNA fragmentation were detected in the granuloma. No micro-organisms were found. Cultures and stains for bacteria, mycobacteria,

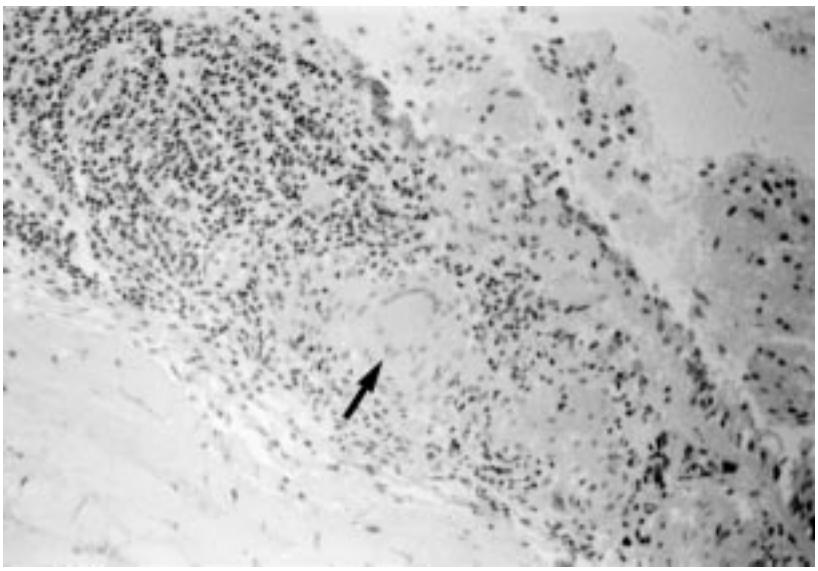

Figure 2 Photomicrograph of a chorioretinal biopsy from the left eye shows a well defined, non-caseating choroidal granuloma predominantly consisting of macrophages and a giant cell (arrow) surrounded by lymphocytes and a few plasma cells. The retina is gliotic, infiltrated with scattered lymphocytes and plasma cells. No micro-organisms were found. (Haematoxylin and eosin; original magnification ×200.)

and fungi were negative. These findings suggest that apoptosis occurs in choroidal granuloma and plays a regulatory role in limiting ocular inflammation.[4]

Upon diagnosis of ocular sarcoidosis and treatment with azathioprine, the patient's vision improved to 20/40 in her right eye and has remained stable for 4 years.

COMMENT

This case illustrates several points: ocular sarcoidosis can occur in the absence of pulmonary signs or symptoms and causes a plethora of ocular findings; the disease eludes accurate diagnosis—in this case, a chorioretinal biopsy excluded chronic infection and led to accurate diagnosis[5]; and ocular sarcoidosis can resist corticosteroid therapy. This patient's disease progressed despite treatment with periocular and systemic corticosteroids and cyclosporine, but responded to azathioprine. Azathioprine has been used successfully to treat sight threatening ocular sarcoidosis at 1–1.25 mg/kg/day.[6] Although sarcoidosis responds to corticosteroids, other immunosuppressive agents may be needed to control ocular disease.

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Relation between blood flow velocities in the ophthalmic artery and in nailfold capillaries

EDITOR,—Studies on visual field and peripheral circulation had led to the hypothesis that the eye might be involved in the vasospastic syndrome,[1] a potential contributor to glaucomatous optic neuropathy.[2] In some patients with peripheral vasospasms, visual field defects worsened after cold provocation, and, often, both peripheral vasospasms and visual field defects improved after calcium channel blocker treatment.[1] Because these and newer observations[3] suggest some parallels in ocular and digital blood flow regulation, the relation between ophthalmic artery and nailfold capillary blood flow velocities was evaluated.

Fifty patients with primary open angle glaucoma with a mean age of 67 (SD 15) years were examined. Excluded were patients with previous filtering surgery or systemic and cardiovascular diseases. Blood flow velocity in nailfold capillaries was assessed in one randomly chosen finger of the right hand (totally arbitrary choice) by means of nailfold capillaroscopy.[4] The velocities measured in each visible vessel were averaged. Blood flow velocity in the ophthalmic arteries was assessed by means of colour Doppler imaging (CDI). The ophthalmic artery was traced nasal to the optic nerve, 10–15 mm posterior to the globe. CDI measurements of the right eye of each patient were considered for further analysis (same side as nailfold capillaroscopy). Peak systolic velocity (PSV), end diastolic velocity (EDV), mean velocity (MV), and resistivity index (RI) (RI = (PSV − EDV)/PSV) were computed. Linear correlations between blood cell velocity in nailfold capillaries and ophthalmic artery were assessed (Spearman's rank order correlation factor).[5]

The mean blood cell velocity in nailfold capillaries was 0.45 (SD 0.32) mm/s. The mean PSV was 37.11 (7.33) cm/s. The mean EDV was 7.7 (3.06) cm/s. The average MV was 15.30 (7.80) cm/s. The mean RI was 0.79 (0.07). EDV (R=0.31; p=0.034) and MV (R=0.38; p=0.007) correlated with blood cell velocity in nailfold capillaries. PSV (R=0.14; p=0.35) and RI (−0.25; p=0.09) did not correlate with nailfold capillary blood cell velocity. Systemic blood pressure (mean systolic blood pressure 120.5 (11.4) mm Hg; mean diastolic blood pressure 73.4 (13.6) mm Hg) did not vary significantly during blood flow assessments.

COMMENT

The results suggest a relation between nailfold capillary blood cell velocity and blood flow velocity in the ophthalmic artery. Although the ophthalmic artery is the only routinely assessed retrobulbar vessel in CDI which does not enter the eye, its contribution to choroidal blood flow is important.[6] Because vasospastic dysregulation seems to be much more common in the ciliary circulation than in the retinal vasculature,[2] it appeared reasonable to evaluate this vessel, especially because this vessel is assessed much more reliably than ciliary vessels during CDI.[7] RI and PSV did not correlate with nailfold capillary blood cell velocity. However, RI is not a measure of velocity. PSV represents a unique event in arterial blood flow, and, possibly, capillary blood flow fluctuations may not be related directly to very brief moments during the cardiac cycle.[8] Although the measurement of EDV is less reproducible compared with PSV and RI,[7] a fact which is expected to alter a potential correlation, nailfold capillary blood cell velocity correlated with EDV and MV in the ophthalmic artery. The relatively constant blood flow during the diastole might predict more closely blood cell velocity in the capillary bed. Mean velocity is not related to unique moments in the cardiac cycle and seems to reflect even more closely blood cell velocity in capillary vessels. Although the present study suggests some common alterations in various vascular beds in glaucoma patients, special caution is warranted. Dysregulative phenomena might well alter various vascular beds in a comparable fashion, but local factors will always influence regional blood flow. Consequently, further studies will have to confirm these results and unravel the exact basis for such a relation.

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