LETTERS TO
THE EDITOR

Bilateral circumscribed haemangioma of the choroid not associated with systemic vascular syndrome

EDITOR,—Circumscribed choroidal haemangioma (CCH) is considered congenital, vascular, relatively rare hamartoma which typically occurs as a localised, monolateral lesion in patients without other vascular malformation. This tumour generally is discovered in adulthood and it is located in the macular area. CCH may be ophthalmoscopically confused with amelanotic melanoma, metastatic tumour, choroidal osteoma, disciform scar, serous detachment, and central serous chorioretinopathy, but may be differentially diagnosed with fluorescein angiography (FA), indocyanine green angiography (ICGA), ultrasonography, and periodic observation.1 The bilateral CCH localisation represents an extremely uncommon condition which, in literature, has been only reported in association with Sturge-Weber syndrome2 3 or Klippel-Trenaunay-Weber syndrome.7 To the best of our knowledge, this is the first documented case of bilateral CCHs in the absence of any other evidence of systemic vascular abnormalities.

CASE REPORT
A 81 year old white man was referred to our institution in June 1999 to undergo conservative therapy because of malignant choroidal melanoma, this represents the first case in whom we diagnosed a bilateral circumscribed vascular hamartoma, which was not associated with any systemic syndrome. During the mid-term follow up (15 months) there were neither ocular nor systemic significant modifications. The echographic and ICGA features of these choroidal lesions, together with the lack of neoplasm or vascular abnormality in another part of the body, lead us to confirm the first documented diagnosis of bilateral CCHs. Last but not least, our findings demonstrate that FA and echography are not always capable of documenting the specific characteristics of small CCH; thus, when this kind of lesion is

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surgery. This specific clinical entity of “capsular contraction syndrome” is usually associated with a reduction in the capsular opening, malposition of the opening, reduction in the equatorial capsular diameter, and possibly intraocular lens (IOL) displacement.

Tractional ciliary body detachment and associated hypotony is an uncommon complication of severe anterior lens capsular contraction. Only three such cases have been reported in the literature. We report a case of tractional ciliary body detachment caused by a severe anterior lens capsule fibrosis, in which Nd:YAG laser anterior capsulotomy was effective in relieving the traction caused by the capsular contraction. We illustrate the value of ultrasound biomicroscopy (UBM) in the diagnosis and management of such conditions.

CASE REPORT

A 72 year old woman with primary open angle glaucoma and previous bilateral trabeculectomies (performed twice in the left eye) was followed up in our clinic since December 1999 for an ischaemic central vein occlusion in her right eye. She had a dense cataract in her left eye, which prevented her from the fundus. The biometry of the left eye showed an axial length of 22.60 mm. Preoperatively intraocular pressures were 15 mm Hg in both eyes. She underwent an uncomplicated phacoemulsification through a superotemporal limbus wound. A capsulorrhesis of about 5 mm was fashioned. A foldable three piece silicone IOL with poly(methylmethacrylate) (PMMA) haptics (Allergan SH40 NF) was implanted “in the bag.” The lens had an optic diameter of 6.0 mm and a haptic diameter of 13.0 mm. In the immediate postoperative period she was noted to have a well centred IOL “in the bag” and fundus showed an inferior hemiretinal vein occlusion involving the macula in the left eye. At this time she had a visual acuity of counting fingers at 2 metres in her right eye and 6/60 in her left eye.

Two and a half months following her cataract surgery she was referred by an optician with deterioration of vision in her left eye. Visual acuity was counting fingers at 2 metres in both eyes. Slit lamp biomicroscopy of the left eye showed a round and quiet anterior chamber. Severe contraction of the CCC opening with eccentric displacement of the CCC orifice was noted and the IOL was displaced superiorly (Fig 1, above). Gonioscopy showed an open iridocorneal angle. There was no evidence of any iris changes or changes at the pupillary border, consistent with pseudoexfoliation in either eyes. Goldmann applanation tonometry revealed an intraocular pressure of 5 mm Hg in the left eye and 14 mm Hg in the right. Posterior segment examination did not show any retinal or choroidal changes. An ischaemic central vein occlusion involving the fovea was noted.

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Figure 3 (Left eye). (A) Standardised A-scan ultrasonography at 1660 m/s demonstrates the temporal paramacular solid lesion of the choroid, with its high and regular internal reflectivity, consistent with the presence of an angiomatos benign lesion. The maximum thickness of this circumscribed choroidal haemangioma is 3.32 mm. (B) Late phase fluorescein angiogram shows an ill defined hypofluorescent and hypofluorescent area in correspondence with choroidal haemangioma (arrows). (C) Early indocyanine green photograph reveals a rapid and complete fill up of the lesion. The haemangioma has a “mulberry appearance” at the stage of maximal fluorescence. (D) Late phase indocyanine green angiogram shows clearing of the dye from the tumour, associated with diffusion into the choroidal and subretinal space (“washout” phenomenon).
Capsular contractile syndrome as a complication of anterior segment laser capsulorhexis: Report of five cases

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In general, shrinkage of the anterior capsule according to Davidson7 is produced by an imbalance between the centrifugal and centripetal forces on the capsular bag. Although the pathogenic mechanism responsible for excessive capsule fibrosis and contracture are not well understood, several histopathological studies have identified the cell types associated with pseudophakic fibrosis.8 9 Frezzotti et al10 attributed constriction of the anterior capsule opening to fibrogenic transformation of the subcapsular and equatorial lens epithelial cells (LECs). Nishi and Nishi7 suggested that this fibrosis might be induced by interleukin 1 or 6 in an autocrine manner. The following three main factors may account for anterior capsule contraction: (1) IOL material, (2) IOL design, and (3) CCC opening. The sphincter effect of an intact capsulorhexis seems to be important in creating significant capsule shrinkage. Some authors believe that the initial diameter of the CCC is an important factor in its pathogenesis. It is postulated that the more epithelium that is left the greater the potential for capsule contraction.11 12 The IOL optic composition may influence the development of anterior capsule fibrosis. Davidson12 suggested that one piece PMMA IOL with a large optic would help counterbalance the centripetal forces of capsular fibrosis. Werner et al13 in their histopathological study comparing different IOL styles found that the rate of anterior capsule contraction was relatively high with plate-haptic silicone lenses. The lowest rate was noted with the three piece acrylic optic PMMA haptic IOLs. In their histopathological grading of anterior capsule contraction with IOL materials and designs, silicone optic-PMMA haptic IOL as used in this case was rated third after plate haptic silicone lenses with large holes and small holes.

Anterior capsule shrinkage shifts the relative position of the lens equator, moving it to a more anterior location. This centripetal movement induces an inward pulling force on the zonular apparatus. Depending on the strength of the zonulotomy, a counteracting force might result. We feel that the smaller capsulorrhesis size and the use of silicone IOL predisposed our patient to develop severe anterior lens capsule contraction. Severe anterior lens capsule contraction can exert centripetal traction on the ciliary body resulting in a ciliary body detachment. In this case Nd:YAG radial anterior capsulotomy was helpful in relieving the phimosis and thereby removing the tractionsal force on the ciliary body.

The authors have no proprietary interest in any of the products described in this paper.

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Angle closure in fellow eye with prophylactic plicarpine treatment

EDITOR,—Prophylactic pilocarpine is often used in patients presenting with unilateral primary acute angle closure until definitive treatment with either peripheral iridotomy can be performed. We present two cases of unilateral primary acute angle closure glaucoma treated with prophylactic plicarpine that subsequently developed angle closure in the fellow eye within 24 hours of admission.

CASE REPORTS

Case 1
An 81 year old woman was referred from the ophthalmic unit in the right eye with visual acuity of 6/9 in the left eye and near vision without complaint.

The right cornea was oedematous with intraocular pressures (IOP) of 56 mm Hg in the right and 17 mm Hg in the left. The iridocorneal angle was closed on the right eye, and narrow on gonioscopy (grade 1 inferiorly and closed superiorly) on the left, with bilateral moderate nucleosclerotic cataracts.

She was treated with intravenous Diamox 500 mg, topical levobunolol, 2% pilocarpine, and dexamethasone 0.1%. Review 1 hour later showed decreased oedema with IOP of right eye 41 mm Hg and left eye 15 mm Hg.

Prophylactic 2% pilocarpine four times daily was started in the fellow eye and she was admitted to hospital. On review 8 hours after admission her IOP was 16 mm Hg in the right eye and 46 mm Hg in the left. The left cornea had minimal oedema and closed iridocorneal angle on gonioscopy. A Nd:YAG laser peripheral iridotomy was performed in the left eye that night with subsequent resolution of the attack.

Case 2
A 46 year old hypermetropic woman (right eye +2.75DS/−0.5 × 160 left eye +4.5DS) with no significant ocular history presented to casualty with intermittent visual disturbance, followed by pain, redness, and decreased vision in the left eye. Visual acuity on presentation was right eye 6/9 and left eye 6/24. The left cornea was hazy with a shallow anterior chamber and IOP of 62 mm Hg. The right iridocorneal angle was narrow but open with pigmented grade 1 angle on gonioscopy. She was admitted and treated with topical apraclonidine, levobunolol, dexamethasone, and intravenous Diamox 500 mg. Pilocarpine 4% every 15 minutes for 1 hour was used in the left eye and a single dose of 4% pilocarpine was instilled in the right eye.

On review 2 hours after admission IOP was 45 mm Hg in the right eye and 26 mm Hg in the left. The right cornea remained clear, the anterior chamber appeared shallow, and repeat gonioscopy showed a closed iridocorneal angle on the right. The angle was opened by compression with a Zeiss gonioprism, and she underwent a Nd:YAG laser peripheral iridotomy initially in the right eye and subsequently in the left eye the following day.

COMMENT

The management of the fellow eye in acute glaucoma is controversial. Although Nd:YAG peripheral iridotomy has established itself as the treatment of choice,1 the use of prophylactic pilocarpine until formal iridotomy can occur remains controversial. In a survey of members of the American Glaucoma Society, pilocarpine was used as the treatment of the fellow eye when iridotomy was deferred by more than half the respondents, whereas close observation was the choice of a third.2 Pilocarpine results in miosis thereby pulling the peripheral iris away from the anterior chamber angle, relieving pupillary block and increasing aqueous outflow facility. Of more concern is the possibility of a paradoxical effect of pilocarpine by a dose dependent shallowing of the anterior chamber, potentially precipitating angle closure in compromised eyes.2,3

The above case highlights concerns on the use of prophylactic pilocarpine (especially in higher concentrations) in the fellow eye. In these cases, prophylactic treatment with pilocarpine did not prevent and probably contributed to angle closure.

Early prophylactic peripheral iridotomy without pilocarpine treatment may be the treatment of choice.

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3 Fleck BW, Wright E, Fairley EA. A randomised prospective comparison of operative peripheral iridectomy and Nd:YAG laser iridotomy treat-


Keratolysis in a patient with pemphigus vulgaris

EDITOR,—Pemphigus vulgaris is an auto-

immune, blistering disease of the skin and mucous membranes. The characteristic ocular finding is conjunctivitis, and corneal involvement is rare.1 We present a case with pemphigus vulgaris with severe keratolysis that required a corneal transplantation.

CASE REPORT

A 41 year old man had suffered from pemphigus vulgaris for 2 years, and prednisolone 40 mg/day and cyclosporine 300 mg/day had been prescribed. He was admitted to the Hamamatsu University Hospital on 15 March 1999 with an acute exacerbation of the symp-
toms because of non-compliance with the corticosteroid therapy. He returned on 17 March 1999 because of increased discharge and visual loss in both eyes. His visual acuity was 20/20 right eye and 20/20 left eye, and his intraocular pressure was 24 mm Hg right eye and 20 mm Hg left eye. No remarkable findings were observed in both visual fields and optic discs. Slit lamp examination showed mild erosions of his eyelid and cornea. The treatment with prednisolone 40 mg/day and cyclosporine 300 mg/day was continued.

He returned on 9 April 1999 because of acute deterioration of vision in both eyes. His visual acuity was light perception in the right eye and counting fingers in the left. The right conjunctiva showed marked oedema and the anterior chamber was flat. Slit lamp examination showed that the lower two thirds of the right cornea had eroded leaving only Descemet’s membrane and endothelium (Fig 1).

The left conjunctiva showed mild oedema and slit lamp examination demonstrated anterior stromal opacities in the lower half of the cornea and bulla-like central corneal epithelial changes. Ofloxacin ointment was prescribed for both eyes.

The opacity of the right corneal stroma gradually increased, and scar-like tissue formed in the area of the erosion. Slit lamp examination showed that Descemet's membrane was touching the iris and lens. The corneal bullous degeneration in the left eye formed an erosion. After the removal of corneal erosion and conjunctival oedema resolved, faint stromal opacities were observed in the region of the corneal lesion in the left eye.

Because of the overall improvement of the cornea of the right eye, an 11 mm right penetrating keratoplasty was performed on 22 September 1999. The dislocated lens was extracted and anterior vitrectomy was also performed. His visual acuity on 11 November 1999 was improved to 8/200 with +9.0 −2.5 × 65° right eye, and 20/25 with −3.0 D −3.75 × 180° left eye.

COMMENT

Corneal involvement is a rare complication in patients with pemphigus vulgaris. Severe corneal involvement has never been reported except in the case of a 56 year old man with severe ocular involvement including conjunctivitis, corneal ulceration, and perforation despite immunosuppressive therapy. Although a causative organism was not isolated, the authors suggested that the complications were due to an infectious agent.

Two mechanisms have been suggested to cause the corneal erosion—bacteria or other pathogenic organisms that infect the cornea because of the epithelial defect and tear film disorder brought on by the corticosteroid and immunosuppressive therapy. Although the culture obtained from right ocular discharge before starting ofloxacin ointment showed a negative result, we could not deny the bacterial infection. We did not perform a bacterial or viral culture or polymerase chain reaction examinations using a corneal sample.

The second mechanism is an autoimmune mechanism against one of the intercellular adhesion molecule—for example, desmoglein (Dsg). The patient was diagnosed as pemphi-
gus vulgaris by histological examination, direct immunofluorescent staining of the skin

Figure 1 Photograph of the anterior segment of the right eye on 11 April 1999.
Because the cornea usually does not have Dsg
1140nogloblin G and high titres of circulating anti-
1337showing intracellular deposition of immu-
1264noglobulin G (right).

Figure 2  Histological examination by haematoxylin and eosin staining of lesional skin disclosed intraepidermal clefts which contained several acantholytic cells (left). The direct immunofluorescent staining of the skin showed intercellular deposition of immunoglobulin G (right).

Isolated episcleral plasmacytoma mimicking episcleritis in a patient with benign monoclonal gammopathy

EDITOR,—We present the unique case of a patient with an isolated plasmacytoma of the episclera mimicking a painful episcleritis. Plasmacytomas usually grow in the bone mar-
row probably because of their special homing receptors—e.g., αβ, integrin. Soli-
ary plasmacytic tumours outside the bone marrow are rare. They mostly involve the oropharynx and the upper respiratory tract, but have also been encountered in the lids, the orbit, and the palpebral conjunctiva. Only one case of a solitary epibulbar plasmacytoma with intraocular invasion has been reported yet.

CASE REPORT
A 61 year old patient presented with an "inflammatory" episcleral nodule within the lower temporal quadrant and mild pain in his left eye (Fig 1), which had already lasted 5 months and had been diagnosed as episcleritis. There was no evidence of rheumatic disease; ANA and ANCA were negative. Neither dexamethasone nor oral fluo-
tacortolone (60 mg) were helpful, thus an exci-
therapy a biopsy was performed which finally allowed for the correct diagnosis. Thus soli-
tary plasmacytoma has to be included in the spectrum of ocular masquerade syn-
drome.

Another interesting aspect is that our patient developed a monoclonal gammopathy, apparently not related to the isolated episcler-
al plasmacytoma. The latter showed a kappa light chain restriction, whereas in the serum the level of IgA lambda was increased. As a thorough general examination did not reveal any signs of systemic disease or isolated plasmacytoma elsewhere, the monoclonal component was attributed to a monoclonal gammopathy of unknown significance (MGUS) which is considered as a benign or premalignant disorder.

Lymphocytes and plasma cells of the MALT, especially the GALT, are character-
ised by integrin αβ, and another integrin αβ, which is displayed by plasma cells homing to the bone marrow. According to this extramedullary plasmacytoma tend to occur more often in the MALT or GALT than in other locations except for the solitary plas-
ctyma of the bone. Ninety per cent of the isolated plasmacytomas grow in the head and neck area, especially in the upper respiratory tract, but they are surprisingly rare in the gastrointestinal tract, though 80% of all immunoglobulines producing cells of the body are located here. The atypical location of the plasmacytoma presented here may be medi-
atated through a specific repertoire of adhesion
molecules. Since antibodies for detection of the above mentioned homing receptors in paraffin sections are not available until now, we were not able to find out whether specific integrins or a total loss of them was responsible for the peculiar location of the tumour in our patient. Additional only be speculated that the visual field defects and atrophy of the atrophic arcus were due to involvement of the atrophic arcus. Whether there was a corresponding homing mechanism for the atrophic arcus, we were not able to find out whether special tissue specific immunoregulatory mechanisms involving accessory cells are also implicated in the localised episcleral tumour growth remain to be elucidated.

We thank Mrs Renate Buchen for technical assistance, Dr Sarah Coupland for providing the VS38 immunostaining, and Dr Fleming Steurbach for reading the manuscript.

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Ocular involvement caused by the accumulation of porphyrins in a patient with congenital erythropoietic porphyria

EDITOR,—Congenital erythropoietic porphyria (CEP; MIM No 263700) is an extremely rare disorder inherited as an autosomal recessive trait, which is characterised by an 80–98% reduction in the activity of uroporphyrinogen III synthase (UROS: EC 4.2.1.75). Clinically, CEP is characterised by severe cutaneous photosensitivity, chronic haemolysis, and massive porphyrinuria resulting from the accumulation in the bone marrow, peripheral blood, and other organs of the non-physiological and pathogenic porphyrin isomers, uroporphyrin I and coproporphyrin I. Red urine may be observed from infancy, and the teeth become stained red. Haemolytic anaemia, an additional complication, may be helped by splenectomy. Besides such manifestations, we reported a scleral change in the patient with CEP, who had a remarkable increase of porphyrins in tear drops. Our case report strongly suggests that the accumulation of porphyrins in tear drops may directly cause the scleral changes in the patients with CEP.

CASE REPORT
A 24 year old man presented typical manifestations of CEP such as skin ulcer and scarring. He was diagnosed with CEP in childhood, because of the elevation of porphyrins in urine. At the time of visit, slit lamp examination of bulbar conjunctiva revealed irregular hypertrophy between palpebral fissures in both eyes. A 3 × 4 mm area of scleral necrosis was observed at the limbus in the right eye (Fig. 1). Hypertrophy of the temporal limbus and pigmentation of eyelids were also observed, but lid closure was normal. Corneal changes were not observed. Visual activity was right eye: 20/50; left eye: 20/20.

In order to cover the region of scleral necrosis, anmmic membrane grafting was performed, but postoperative wound healing was slow and the graft failed to be attached. Histological finding with a tissue taken during this operation showed an inflammatory infiltration of neutrophils and plasma cells in connective tissue under conjunctival layer (data not shown).

To confirm whether this scleral necrosis is caused by the direct effect of the accumulation of porphyrins in tear drops, we provisionally treated the patient with a cumulative sulphadiazine dose of 40 g. Microscopy of freshly voided urine commonly shows characteristic “sheaves of wheat” crystalluria and haematuria. Ultrasonography can reveal echogenic foci in the renal parenchyma as well as in the collecting systems, and hydroureteroscopy. x-ray examination has a low diagnostic sensitivity.

Management can be conservative with prompt analgesia, intravenous fluids, plus or minus diuretics, and alkalisation of urine with sodium bicarbonate to above a pH of 7.5. This usually achieves prompt dissolution of even large calculi. It is not always necessary to stop sulphadiazine.

Letters, Mailbox, Notices


COMMENT
The major reports of sulphadiazine crystalluria occur in patients with AIDS under treatment for toxoplasmosis encephalitis. These patients have various factors predisposing them to the development of crystalluria such as poor fluid intake, fever, diarrhoea, hypoalbuminuria, and acidification of the urine. The associated polypharmacy of many AIDS patients (i.e. drugs such as aciclovir, triamterene, primidone, or other sulphonamides.

Historically, sulphadiazine crystalluria has been reported in non-AIDS patients and may cause renal impairment in 1–4% of HIV negative patients. To our knowledge, this observation has not been reported in the ophthalmologic literature. Dermatologists who we surveyed were not aware of this potential complication, nor is it documented in the British National Formulary.

Although it occurred quickly in our patient, the complication usually occurs after a median of 10 days in HIV negative subjects at a cumulative sulphadiazine dose of 40 g. Microscopy of freshly voided urine commonly shows characteristic “sheaves of wheat” crystalluria and haematuria. Ultrasonography can reveal echogenic foci in the renal parenchyma as well as in the collecting systems, and hydroureteroscopy. x-ray examination has a low diagnostic sensitivity.

Management can be conservative with prompt analgesia, intravenous fluids, plus or minus diuretics, and alkalisation of urine with sodium bicarbonate to above a pH of 7.5. This usually achieves prompt dissolution of even large calculi. It is not always necessary to stop sulphadiazine.

We report a case of acute ureteric obstruction in a young female with her first presentation of recurrent ocular toxoplasmosis. We would like to bring to the attention of ophthalmologists the risk of crystalluria in patients being treated with sulphadiazine.

CASE REPORT
A 22 year old, otherwise fit woman presented with floaters in the left eye. She had had poor vision since childhood when she had been diagnosed as “amblyopic” and undergone strabismus surgery for esotropia. A pigmented and atrophic scar was present at the left macula, and involving the fovea. At the inferonasal edge of the scar was a raised creamy area of activity with overlying vitritis. A diagnosis of recurrent toxoplasmosis was made.

Despite the poor visual prognosis of this eye, the symptomatic nature of this lesion and the intensity of the inflammatory response prompted treatment. Pyrimethamine (75 mg immediately then 25 mg twice daily) and sulphadiazine (1 g four times daily) were started with folic acid (5 mg twice weekly). Topical dexamethasonc, cyclopentolate, and oral prednisolone (60 mg reducing course) were added later.

Within 24 hours of starting treatment the patient felt unwell, with nausea, anorexia, and oligidipsia. She developed pink discoloration of the urine in which she noted sediment, and intense loin pain. Hospitalisation followed. Urinalysis demonstrated a pH of 5.0, urinary blood and protein. An intravenous urogram suggested an obstruction at the right vesi- coureteric junction and retrograde ureteroscopy demonstrated crystalluria, and insertion of a temporary ureteric stent at this time, with administration of intravenous fluids, effected symptomatic relief. Sulphadi- azine was suspended.

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enuously reported by Xu et al. These mutations have been predicted to cause a glutamine to alanine substitution at residue 62 (T62A), and a C to T transition of nucleotide 184 that predicted a threonine to alanine substitution at residue 64 (T64A). These mutations have been previously reported by Xu et al. Furthermore, sequence analysis of UROS was performed and an A to G transition of nucleotide 184 that predicted a threonine to alanine substitution at residue 62 (T62A), and a C to T transition of nucleotide 745 that predicted a glutamine to premature stop codon (Q249X). These mutations have been previously reported by Xu et al.6

COMMENT

This patient was confirmed to have compound heterozygous mutations, T62A/Q249X. These mutations had been described by Xu et al. in a Japanese patient with CEP. They performed in vivo expression study for each mutation, and confirmed that each of them had no residual activity. We can expect that both mutations in this case are "disease causative." Scirial changes at the body surface lesions in CEP are mainly caused by the accumulation of porphyrins.4 Here we proved the accumulation of porphyrins in tear drops with a single case of CEP. Additional cases are needed to confirm the presence of porphyrins in tear drops although they are asymmetrical for eye involvement. Our finding demonstrates the likelihood that accumulated porphyrins in tear drops directly exerted a toxic effect in scirial lesions, the protection of sunlight by ultraviolet cut glasses is strongly recommended for prevention against the initiation and progression of scirial lesions in the patients with CEP.

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Figure 1 Four direction images of right eye. Irregular hypertrophy at the temporal limbus and scleral necrosis at the limbus were observed.

Figure 2 Tear drop porphyrin analysis by high performance liquid chromatography (HPLC). In a normal sample, no porphyrin isomers were observed (data not shown), whereas in a patient sample remarkable elevation of uroporphyrin I + III (UROI + III), coproporphyrin I (CPI), and protoporphyrin IX (PP) were observed (8.48, 1.46, and 22.96 µg/g creatinine, respectively).


Bilateral facial nerve palsy associated with p-ANCA positive vasculitis in a patient with rheumatoid arthritis

EDUCR.—Rheumatoid arthritis is a chronic, generalised, symmetrical, inflammatory polyarthritis. Extra-articular associations may involve the eyes, heart, lung, skin, and more rarely, the central and peripheral nervous system. We describe a case of bilateral facial paresis associated with a p-ANCA positive vasculitis in a patient with rheumatoid arthritis.

CASE REPORT

A 67-year-old woman presented with 2 days of left sided facial weakness. She was known to suffer from rheumatoid arthritis, and displayed the characteristic hand and finger deformities of this condition. Additional features of vitiligo, hypothyroidism, and splenomegaly were present. Her medication consisted of methotrexate 5 mg weekly, thyroxine 100 µg once daily, and folic acid 5 mg once daily. Examination revealed isolated left sided lower motor neuron facial palsy, and a left Bell’s palsy was diagnosed. One week later, she returned with right sided facial weakness. No improvement on the left side had occurred and bilateral lower lid paralytic ectropion was noted. A provisional diagnosis of rheumatoid associated mononeuropathy multiplex was made, and a rheumatological consultation was obtained. Haematological investigations revealed a positive rheumatoid factor (RF) and p-ANCA, and a raised plasma viscosity of 1.80. Other autoimmune studies including ANA, anti-Ro and La antibodies, and c-ANCA were negative, and renal function was normal. Chest radiography and magnetic resonance imaging of the brain were unremarkable.

Three pulses of intravenous methylprednisolone 500 mg were given over 3 days, with commencement of oral prednisolone 1 mg/kg. Despite intensive topical lubrication, developing exposure keratopathy necessitated the surgical correction of the bilateral paralytic ectropion. The oral prednisolone was rapidly tapered down to 5 mg/day, and then discontinued after 3 months. p-ANCA levels subsequently became undetectable.

Full orbicularis function gradually recovered, but only partial recovery of the lower facial muscles occurred. Renal function remained normal throughout and there was no significant exacerbation of the polyarthritis.

COMMENT

Facial nerve weakness may be the result of a number of underlying disorders including vasculitis. The development of bilateral signs in rapid succession, in association with rheumatoid arthritis, highlighted a potential vasculitic process in this case. Other causes of facial paresis, such as pontine disease—for example, demyelination, or primary muscular disorders—for example, myasthenia gravis, and post-infective polymyopathy were excluded on clinical grounds and after investigation.

Rheumatoid factor consists of IgM antibodies against the patients’ own IgG, and is an important diagnostic feature in rheumatoid arthritis. However, RF may also be seen in

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polyarteritis nodosa, scleroderma, Wegener’s granulomatosis, systemic lupus erythematosus, and sarcoidosis. No clinical or other investiga-tive features of these conditions were demonstrated in the case described here, and the patient displayed typical erosive joint features of rheumatoid arthritis. RF may lead to immune complex (IC) mediated vasculitis due to IC formation and deposition in the joints and vessels causing endothelial damage, perivascular cellular infiltration, and thrombosis formation.

Another mechanism of a vasculitic process is through leucocyte mediated cytotoxicity caused by ANCA. ANCA may promote neutrophil activation and endothelial injury,5,6 by targeting the neutrophil granule enzymes. ANCA and myeloperoxidase (p-ANCA) are useful diagnostic sero-logical markers in a number of vasculitic conditions such as Wegener’s granulomatosis, microscopic polyangiitis, and Churg-Strauss syndrome. They may be found less commonly in rheumatoid arthritis, systemic lupus ery-thematosi,7 inflammatory bowel disease, and autoimmune haematopoietic diseases.8 In one study, the incidence of p-ANCA in patients with rheumatoid arthritis was 21%, and was strongly associated with nephropathy, more severe disease, and increased inflammation.8

In this case, other conditions more commonly associated with positive ANCA titres were excluded on clinical grounds and follow-up investigations. Magnetic resonance imaging is sensitive for cerebral vasculitis,9 and excludes CNS involvement. The optimum treatment of ANCA associated vasculitis is generally considered to consist of a combination of corticosteroids and immunosuppressive agents. Azathioprine, cyclosporin, azathioprine, or cyclophosphamide may be used although the most effective treatment protocols are yet to be determined. Evidence of renal or CNS involvement should prompt aggressive therapy because of potentially life threatening complications. In this case, therapy consisted of pulsed intravenous methylprednisolone in the initial phase, followed by oral pred-nisone. Additional immunosuppression was not required as widespread evidence of disease activity was absent. Gradual improvement of the facial paresis occurred and vigorous treatment of the exposure keratopathy prevented visual loss in this case.

Bilateral facial nerve palsy is rarely seen in vasculitic conditions. Isolated reports of bilateral facial nerve paralysis associated with Sjogren’s syndrome and polyarteritis nodosa exist.10

Rheumatoid arthritis is a common condition, and life threatening complications, although rare, are well recognised. Initial presentation may be to the ophthalmologist and awareness of such situations, will improve the prognosis for these patients.


Bilateral conjunctival lesions in Melkersson-Rosenthal syndrome

**EDITOR,—**The Melkersson syndrome is a rare granulomatous disease of unknown origin. The typical clinical picture consists of recurrent facial oedema associated with peripheral facial palsy and was first described by Melkersson in 1928.1 Three years later a fissured tongue called lingua pilcata was added to the classic features by Rosenthal.2 This clinical was in patients with granulomatous cheilitis, facial palsy, and fissured tongue was first called Melkersson-Rosenthal syndro-mome (MRS) by Lüscher in 1949.3 We report on a patient with the typical clinical signs who had chronic bilateral conjunc-tival lesions. To our knowledge an association of conjunctival lesions with MRS has not been described previously.

CASE REPORT

A 64 year old man presented with a fissured tongue, recurrent painless facial oedema, especially of the eyelids, and facial flush for 6 years. A review of the other systemic diseases was unremarkable. He required blepharoplasty for the correction of lid malformation.

CASE REPORT

The histopathological findings of the skin biopsy confirmed the clinical suspicion of MRS by the typical granulomatous infiltration (Fig 1A).

Furthermore, he complained bilateral conjunctival swelling had been present for 6 months. Visual acuity was 20/30. Slit lamp examination revealed a bilateral flashy mass extending from the upper fornices to the limbus and conjunctival injection (Fig 1B). Motility of the eyeball was normal and an exophthalmus has not been present. In the magnetic resonance image (MRI) of the orbit a bilateral enlargement of lacrimal glands and a swelling of the lateral rectus and superior rectus muscle of the right eye were observed. Staging examinations for lymphoma or other malignancies were uneventful. Since differential diagnosis included a bilateral orbital lymphoma a conjunctival biopsy was performed. The histopathological examination of the conjunctival specimen by light microscopy revealed a subepithelial process. An infiltrate of small lymphocytes without any differentiation and with septate orientation was found extending from the upper fornices to the limbus (Fig 1A).


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A satisfactory conservative therapy has not been established so far. The results of various modes of symptomatic treatment, including systemic or topical glucocorticosteroids, are questionable. They may reduce the patients’ complaints, at least temporarily. Another treatment consists of clofazimine, which is an oral phenazine. This drug had been useful in other conditions with granulomatous inflammation. Finally, a surgical excision of the mass has been suggested for granulomatous chelitis or blepharitis, in order to improve the motility of the eyeball when exophthalmus occurred. Conjunctival biopsy with histopathological and immunohistochemical examination may be helpful to differentiate it from other lymphoid lesions.

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1 Melkerson E. Case of recurrent facial paralyses with angioneurotic edema. Hygiea 1928;90:737-41.

Choroidal detachment following extracapsular cataract extraction in a patient treated with latanoprost

EDITOR—Adverse reactions associated with the topical administration of the synthetic prostaglandin F2α analogue latanoprost have been described. We would like to report a case of choroidal detachment following extracapsular cataract extraction in a patient treated with topical latanoprost.

CASE REPORT
A 78 year old man initially presented with primary open angle glaucoma in 1981. This was well controlled on timolol and epithalamic follow up was uneventful except for the development of left age related maculopathy in 1995 reducing the vision to 6/9. In November 1999 the intraocular pressure (IOP) became uncontrolled and a left sided cataract noted. Latanoprost was substituted with subsequent control of the IOP.

He underwent an uneventful left extracap- sular cataract extraction by a traditional, non- phacoemulsification technique at another facility in January 2000 (the operating surgeon did not perform phacoemulsification on an open cataract patient). Postoperative drops were betamethasone, chloramphencil, and latanoprost. Immediately postoperatively he experienced transient nocturnal eye pain and subsequent photophobia. He also noticed a shadow in his left vision. Two weeks postoperatively he still had persistent eye pain and the IOP was recorded as 25 mm Hg. Acetazolamide (orally) and Timolol LA (MSD) were added to the above medications. Three days later examination revealed a visual acuity of 6/24 and IOP 16 mm Hg. Fundoscopy showed the presence of a large temporal choroidal effusion.

An opinion was requested and we first saw the patient 3 days later. Visual acuity was 6/60 at best, and examination revealed corneal folds, a marked anterior uveitis with 3+ cells, and a 360 degree choroidal detachment most marked temporally. The IOP measured 10 mm Hg. The latanoprost, chloramphencil, and acetazolamide were stopped, the Timolol LA continued and dexamethasone 0.1% 2 hourly and cyclopentolate 1% twice daily commenced. Three days later the choroidal detachment was complete and there were no signs of uveitis. The IOP was 22 mm Hg and the visual acuity had improved to 6/12 at best.

COMMENT
The development of choroidal detachment in a patient with primary open angle glaucoma following cataract extraction has been described. However, this patient had previously had a trabeculectomy, undergone phacoemul- sification, and had severe hypotony postopera- tively. In another report choroidal effusion and hypotony were noted in a patient who 8 weeks before commencing latanoprost had undergone a combined cataract extraction and trabeculectomy. It is therefore plausible that, in our case, the choroidal detachment was present from a short time following surgery in view of the subjective shadow in the patient’s vision. It would appear that the detachment developed and persisted in the presence of an elevated IOP. Withdrawal of the latanoprost led to complete resolution of the choroidal detachment but the IOP remained elevated. Uveal effusion has been noted following phacoemul- sification without concurrent use of latanoprost. However, in this study all effusions were small and correlated with the presence of hypotony following surgery.

Latanoprost would appear to lower IOP by conduction from choroidal blood flow cools the less, these zones experience the same tem- perature rise as every other treated area and spots. This physiological phenomenon was established in experiments, theoretical, and clinical studies. Furthermore, it is true that the overlapping zones occur when multiple spots are used for very large treatment areas. None the less, these zones experience the same tem- perature rise as every other treated area and no clinical abnormalities have been noted in the small overlapping zones. Although TTT is mainly used for occult membranes our results indicate that it may have a place in classic

2 Wu G. Severe hypotony following cataract extraction in a patient on latanoprost. Eye 2000; 14:915-16.

T&T and CNV

EDITOR.—We thank Ergun and Stru for their interest in our paper and agree with their comments that it is not possible to directly compare a pilot study with a randomised controlled study. We also pointed out that the angiographic follow up data were not com- plete, as once membrane closure was obtained the patients were followed up clinically.

The issue of the laser spot size in transpupil- lar thermotherapy (T&T) is confusing; how- ever, it is known that more irradiance (W/cm²) is needed for smaller laser spots because heat conduction from choroidal blood flow cools smaller spots more efficiently than larger spots. This physiological phenomenon was established in experiments, theoretical, and clinical studies. Furthermore, it is true that overlapping zones occur when multiple spots are used for very large treatment areas. None the less, these zones experience the same tem- perature rise as every other treated area and no clinical abnormalities have been noted in the small overlapping zones. Although TTT is mainly used for occult membranes our results indicate that it may have a place in classic
membranes and in this study stabilisation of vision was obtained in the majority of these patients and in a minority an improved vision was noted.

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NOTICES

Affordable eye care
The latest issue of Community Eye Health (37) discusses affordable eye care. For further information please contact Community Eye Health, International Centre for Eye Health, Institute of Ophthalmology, 11–43 Bath Street, London EC1V 9EL. (Tel: (+44) (0) 20-7608 6900; fax: (+44) (0) 2750 3207; email: eyeresource@ucl.ac.uk) Annual subscription £25. Free to workers in developing countries.

International Centre for Eye Health
The International Centre for Eye Health has published a new edition of the Standard List of Medicines, Equipment, Instruments and Optical Supplies (2001) for eye care services in developing countries. It is compiled by the Task Force of the International Agency for the Prevention of Blindness. Further details: Sue Stevens, International Centre for Eye Health, 11–43 Bath Street, London EC1V 9EL, UK (Tel: (+44) (0) 20-7608 6910; email: eyeresource@ucl.ac.uk).

22nd Annual Meeting of the Glaucoma Society (UK & Eire)
The 22nd Annual Meeting of the Glaucoma Society (UK & Eire) will take place on 22 November 2001 at the Central Conference Centre, 90 Central Street, London EC1V 8AQ.

The Allergan Guest Lecture will be delivered by Professor Jost Jonas of the University of Erlangen, Germany on the subject of the optic disc.

Further details: Mrs Janet Flowers, Administrator, 29 Quarry Hill, Grays, Essex, RM17 5BT (tel/fax: 01375 383172; email: glaucomasocukeire@talk21.com; website: www.iga.org.uk).

41st St Andrew’s Day Festival
Symposium on Therapeutics
The 41st St Andrew’s Day Festival Symposium on Therapeutics will be held on 6–7 December 2001 at the Royal College of Physicians of Edinburgh. Further details: Ms Eileen Strawn, Symposium Co-ordinator (tel: 0131 225 7324; fax: 0131-220 4393; email: e.strawn@rcpe.ac.uk; website: www.rcpe.ac.uk).

4th International Conference on the Adjuvant Therapy of Malignant Melanoma
The 4th International Conference on the adjuvant therapy of malignant melanoma will be held at The Royal College of Physicians, London on 15–16 March 2002. Further details: Conference Secretariat, CCI Ltd, 2 Palmerston Court, Palmerston Way, London SW8 4AJ, UK (tel: + 44 (0) 20 7720 0600; fax: + 44 (0) 20 7720 7177; email: melanoma@confcomm.co.uk; website: www.confcomm.co.uk/Melanoma).

XXIXth International Congress of Ophthalmology
The XXIXth International Congress of Ophthalmology will be held on 21–25 April 2002 in Sydney, Australia. Further details: Congress Secretariat, C/- ICMS Australia Pty Ltd, GPO Box 2609, Sydney, NSW 2001, Australia (tel: +61 2 9241 1478; fax: +61 2 9251 3552; email: ophthalm@icmsaustr.com.au; website: www.ophthalmology.aust.com).

International Society for Behçet’s Disease
The International Society for Behçet’s Disease was inaugurated at the 9th International Congress on Behçet’s Disease. Professor Shigeaki Ohno represents the ophthalmology division (Department of Ophthalmology and Visual Sciences, Hokkaido University Graduate School of Medicine, Sapporo, Japan: tel: +81-11-716-1161 (ext 5944); fax +81-11-736-0952; email: sohno@med.hokudai.ac.jp). The 10th International Congress on Behçet’s Disease will be held in Berlin 27–29 June 2002. Further details: Professor Ch Zouboulis (email: zoubbere@zedat.fu-berlin.de).