Conjunctival changes associated with yellow nail syndrome

The yellow nail syndrome (YNS) is a rare clinical entity characterised by slow growing yellow discoulored nails in association with peripheral lymphoedema, pulmonary manifestations (bronchiectasis, pleural effusions), and chronic sinusitis. About 100 cases of YNS have been described in the literature but it has never been associated with ocular manifestations. We report one patient with YNS in whom chronic chemosis and conjunctival degenerative lesion was observed.

Case report

A 61 year old man was referred with a 2 month history of ocular irritation in the left eye. His past medical history was significant for yellowish slow growing nails, chronic maxillary sinusitis, and bronchiectasis for 5 years diagnosed as YNS. On examination, nails of both hands and feet showed yellow discoloration and thickening (Fig 1). He also had oedema over the lower limbs and subacute bronchial infection for 2 months. The underlying connective tissue was normal (Fig 2 bottom). Ocular symptoms improved rapidly and postoperative antibiotic and steroid eye drops where discontinued and improved rapidly and postoperative antibiotic and steroid eye drops where discontinued.

Figure 1 Yellow nail syndrome. Note yellow discoloration, increased curvature, and thickening of the nail plates in fingernails.

Comment

YNS was first described by Samman and White in 1964. The syndrome has been defined as the complete triad of slow growing yellow nails, lymphoedema, and pleural effusions but today it is accepted that the presence of two of the three symptoms is sufficient to establish the diagnosis, even though the abnormalities may appear separately with intervals of several years.

The aetiology of YNS is obscure, while pathogenesis seems to involve impaired lymphatic drainage. Ocular involvement has never been described in the YNS. However, conjunctival chemosis observed in our patient could be related to YNS which may involve the pleura, the lungs, but also other serosal or mucosal membranes such as the conjunctiva. Moreover, ocular symptoms and corneal microcyst passed at the same time of a rise of pulmonary symptoms. An inflammatory component, which could alter blood flow and capillary permeability, has not been excluded and, interestingly, topical dexamethasone used to treat chemosis is efficient.

There is very little information on histopathological findings observed in the YNS. Nail biopsies demonstrated features of maturation disarray in the nail bed epithelium associated with dense, fibrous tissue replacing subungual stroma. The changes that we observed in the conjunctival epithelium are similar to that in the nail bed in YNS. Since it appears that there is a pathogenic association between the YNS and ocular findings, this association is probably not coincidental and could be the first report of ocular manifestation of YNS.

References


Bilateral anterior uveitis as a presenting manifestation of sarcoidosis and syphilis

We report an unusual case of bilateral acute anterior uveitis in an asymptomatic patient in which ophthalmic examination and laboratory tests showed the diagnosis of syphilis and sarcoidosis.

Case report

A previously healthy 34 year old Hispanic woman with a 2 week history of blurred vision was referred for evaluation. The patient had no systemic complaints. On examination her best corrected visual acuity was 20/30 in both eyes.
the right eye and 20/40 in the left. Slit lamp examination showed bilateral findings of moderate (2+) anterior chamber cells and non-granulomatous keratic precipitates (Fig 1). Fundus examination did not show any signs of vascular, retinal, or choroidal disease and a diagnosis of bilateral acute anterior uveitis was made. Treatment with topical prednisolone acetate and cyclopentolate drops resulted in mild improvement. Routine laboratory evaluation including complete blood count (CBC), blood chemistry, urinalysis, PPD, chest x-ray, and syphilis testing for syphilis. Sarcoidosis is another condition that can imitate any form of ocular inflammation. We are unaware of any previous reports of the association of sarcoidosis and syphilis in an asymptomatic patient with uveitis, and could find no reference to it in a computerised search using Medline.

The diagnosis of syphilitic uveitis requires a high index of suspicion and the patient’s clinical picture should be taken into consideration. The polymerase chain reaction (PCR) assay has been used to detect Treponema pallidum in CSF and serum but has not been used in routine diagnosis. In this case, syphilis was presumed to be the cause of the ocular inflammation because there was an associated inflammatory pleocytosis in CSF and this structure is embryologically related to the aqueous humour and, additionally, there was a marked improvement in the anterior segment inflammation with the syphilitic treatment. Moreover, the association of both diseases in this patient could be coincidental; however, it might be possible that the anergy induced by sarcoidosis could help the development of syphilis. We suggest a routine investigation in all cases of unexplained ocular inflammation including chest x-ray and syphilitic serology to screen for sarcoidosis and syphilis, owing to their great mimicry. Other laboratory tests should be performed following a tailored approach.

**References**


**Tangent screens are still useful in the assessment of vigabatrin induced visual field defects**

Vigabatrin induced constriction of peripheral visual fields was first reported in 1997. The potential mechanisms of vigabatrin induced peripheral field constriction are many, but in our opinion, not enough attention has been
paid to the method and appropriateness of recording this constriction. Current recommendations for patients prescribed vigabatrin are that they are screened at regular intervals by automated perimetry. Automated perimetry cannot, however, differentiate between pathological and functional (non-physiological) constriction of the visual field. Furthermore, automated perimetry, although deceptively simple for the operator to perform, is notoriously laborious and fatiguing to the unwary. This is clearly demonstrated in the recent case report where a 10 year old girl’s visual field constriction apparently reversed on cessation of vigabatrin. Baseline visual fields, performed with automated perimetry, showed a classic artefact cloverleaf-shaped pattern that was not recognised by the authors. Automated threshold perimetry involves checking the visual threshold of the retina at set intervals. To reduce the number of presented stimuli starting points for threshold determinations are made at four quadrants 9 degrees from the horizontal and vertical meridians. Not infrequently, poorly cooperative patients are only attentive during this initial stage resulting in a cloverleaf-shaped field. In this situation the reliability indices are of little help as the suprathreshold false negative reliability indices are based on already fatigued thresholded locations.

Case report
A 30 year old woman was referred to the neuro-ophthalmology clinic in October 1998 for confirmation of vigabatrin induced constriction of visual fields. Her seizures had started at the age of 12 and consisted of sudden tonic posturing of the limbs preceded by left sided sensory symptoms. Initially she was having 12 seizures a week but by 1998 she was having 18 seizures a day. She was unable to tolerate phenytoin, valproate, carbamazepine, clonazepam, lamotrigine, gabapentin, or topiramate and in 1990 had been started on vigabatrin. In 1998 she was referred for consideration of epilepsy surgery. At that time she complained of bumping into objects and in 1999; she was noted on simple confrontation testing to have constricted visual fields. Automated perimetry was recommended and this was subsequently performed (Fig 1). Gross peripheral field constriction was noted but tangent screen examination at 1 and 2 metres revealed this to be non-physiological tubular visual field constriction (Fig 2).

Comment
The best way to ascertain whether visual constriction is pathological or not is to test the patient at 1 and 2 metres using a wall mounted tangent screen. The visual field, whether constricted or not should be conical in shape and expand geometrically with increasing distances. Patients with functional visual field constriction can often be detected by the fact that on repeated testing of the visual field at an increased distance from the tangent screen they will not report this change in field diameter in an attempt to be consistent with their first field (tunnel visual field). This is not physiologically possible and is clear evidence of functional visual impairment.

Vigabatrin may well induce visual field constriction as a result of retinal toxicity but until studies are reported using tests of patients on vigabatrin at two viewing distances then this issue will remain open to debate.

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Successful treatment of squamous cell carcinoma of the lower eyelid with intralidial cidofovir
Cidofovir (HPMPC), an acyclic nucleoside phosphonate analogue, is a promising drug that acts against a wide number of DNA viruses.1 In 1997, the US Federal Food and Drug Administration approved cidofovir (for intravenous use only) for the treatment of cytomegalovirus retinitis in patients with AIDS.2 Over the last few years, cidofovir in a 1–3% gel or cream vehicle has been found to be effective against unmanageable viral cutaneous lesions induced by herpes, pox, and papilloma families.3 Recent studies have explored intralidial administration of cidofovir for the treatment of HPV related tumours, such as cervical epithelial neoplasia, oesophageal carcinomas, and HSV-8 induced Kaposi’s sarcoma.4 5 We report a squamous cell carcinoma (SCC) of the eyelid which was successfully treated with intralidial cidofovir.

Case report
A 70 year old man presented with a nodular lesion 10 × 14 mm in size on his right lower eyelid, which had appeared 3 months earlier (Fig 1). The patient, who was otherwise in good general condition, reported a history of chronic, intense solar radiation exposure because he had lived in Somalia for many years. In the past 3 years he had been repeatedly treated with liquid nitrogen for multiple actinic keratosis of his forehead and upper eyelids. The clinical diagnosis of cutaneous squamous cell carcinoma was confirmed by biopsy. He was referred to the eye clinic for consideration of treatment. The tumour was situated on the lateral aspect of the lower eyelid. It was a broad based, distinct tumour, shaped like a mushroom (Fig 2). On palpation it was described as firm and mobile. The left lower eyelid was edematous and red from chronic inflammatory processes. The patient was otherwise fit and well, and was taking no medications. A clinical diagnosis of malignant SCC was made.

Successful treatment of squamous cell carcinoma of the lower eyelid with intralidial cidofovir
Kidney toxicity is the most common but less frequently uveitis, macular oedema, neutropaenia, thrombocytopenia, nausea, fever, hair loss, and muscle pain have also been observed. When administered topically or intravenously cidofovir has not, to date, shown systemic toxicity.

Surgical excision remains the best possible treatment for SCC, as it is usually curative and permits the histopathological evaluation of margins. However, a successful outcome with intralesional cidofovir in this case might be worth considering.

SCC was confirmed by the histological examination of a punch biopsy. As the patient refused conventional surgery, after obtaining written consent the lesion was treated with a dose of cidofovir 0.1 ml (7.5 mg of active principle). The drug was injected both intralesionally and perilesionally with a fine needle (26 gauge). Care was taken to avoid intravascular inoculation. Erythema and ulceration were evident after 3 days, then the lesion became progressively smaller and flatter until it disappeared within the month. A skin punch biopsy was performed after 12 months on the previous lesion area, but revealed no presence of neoplastic cells. No systemic side effects were noted and the cosmetic result was excellent (Fig 2). The patient is free from recurrences after a 24 month follow up.

**Comment**

Surgical excision is the treatment of choice for SCC. Alternatively, liquid nitrogen, electrocautery, radiotherapy, or laser photoocoagulation may be used. Decisions regarding treatment depend on the age of the patient, the location, extension, and severity of the neoplasm. For cases in which surgery or alternative cytodestructive techniques are not practical, local treatment with 5-fluorouracil, nitrogen mustard, bleomycin, mitomycin C, photodynamic therapy, or inquisimod may be considered as an alternative therapeutic option. Recent studies have showed that cidofovir exerts tumoricidal activity towards HPV related cervical intraepithelial neoplasia, oesophageal and respiratory papillomatous tumours, or HSV-8 related Kaposi's sarcoma. To our knowledge, the regression of SCC after the intralesional injection of cidofovir has not previously been reported.

The mechanism of cidofovir as an anti-neoplastic agent is unknown. The involution of the neoplastic tissue could be due to the inhibition of rapidly proliferating cells through a decrease in DNA thymidine incorporation, the activation of tumour suppressor genes, the induction of apoptosis, and the inhibition of angiogenesis.**

Systemic administration of cidofovir is burdened with serious, dose related side effects.

**Multiple idiocrinal cysts in patients with mucopolysaccharidosis**

The mucopolysaccharidoses (MPSs) are rare hereditary diseases. They are classified into six types by the distinct lysosomal accumulations of glycosaminoglycans, which give rise to the progressive clinical features with involvement of multisystems. Ophthalmic complications, such as corneal stromal opacity, pigmentary retinal degeneration, optic nerve atrophy, and glaucoma, are common in patients with MPSs. Cysts in various organs have been reported in patients with MPSs—for example, multiple denticigerous cysts, multilocular large cysts in the white matter and arachnoid of the brain, and bone cysts. In the eye, membranebound vacuoles in the non-pigmented epithelium of the ciliary processes have been observed by electron microscopy. However, idiocrinal cysts have never been reported in patients with MPSs.

We present two cases of multiple idiocrinal cysts in two patients with MPSs, one with Scheie syndrome and the other with Maroteaux-Lamy syndrome.

**Case reports**

**Case 1**

A 23 year old woman, who was diagnosed with Scheie syndrome (MPS type IS) by enzyme assay. The activity of α-L-iduronidase in peripheral blood lymphocytes was not detectable. She had bilateral corneal stromal opacities, shallow anterior chambers, and high intraocular pressures. On 6 April 1998 her corrected visual acuity was 20/50 in both eyes. Her right intraocular pressure was 24 mm Hg and the left was 20 mm Hg with topical medication. Ultrasound biomicroscopy revealed multiple round cystic lesions with uniformly low echo density similar to anterior chamber fluid in all quadrants of the posterior iris, idiocrinal sulcus, and pars plicata of both eyes (Fig 1).

**Case 2**

A 23 year old woman, who was diagnosed with Maroteaux-Lamy syndrome (MPS type VI). The activity of arylsulphatase B in the peripheral blood lymphocytes was significantly low. At the age of 13 years, she underwent penetrating keratoplasty on her right eye because of corneal stromal opacity. At the age of 23 years, she underwent deep lamellar keratoplasty on her left eye. On 10 December 1997, slit lamp examination disclosed a clear graft and the shallow anterior chamber in both eyes. The corrected visual acuity in her right eye was 20/30 and left was 20/400. Her right intraocular pressure was 12 mm Hg and left was 18 mm Hg without medication. Ultrasound biomicroscopy revealed multiple round echo lesions in the posterior iris and ciliary body similar to case 1 in both eyes (Fig 2).

We examined an additional two patients with Scheie syndrome; however, no idiocrinal cysts were found in either patient.

**Comment**

We have demonstrated the presence of multiple round cystic lesions. From this ophthalographic finding, we interpret these lesions as multiple idiocrinal cysts in both patients. There is a wide gap in the incidence of ciliary body cysts on the posterior ciliary body because of the difficulty in detecting them by conventional methods. Marigo et al retrospectively reported that cystic lesions were identified in 108 eyes of 86 out of 4632 patients by ultrasound biomicroscopy and the incidence of the multiple cysts occupying more than 180° was 13.3%. Kunihara et al studied ciliary body in 232 eyes of 116 healthy people by ultrasound biomicroscopy. They reported that ciliary body cysts were detected in 54.3%, and all the cysts were located at the idiocrinal sulcus or pars plicata. The cysts in our patients were located at the posterior iris as
well as in the iridociliary sulcus and pars plicata, and the number of cysts was much larger than that of healthy people in the previous reports.

Also, the reports concerning cysts in other organs in MPSs patients support the notion that iridociliary cysts in MPSs patients were different from usual cysts in normal patients. Because no evidence of the progression of the iris cysts was obtained, neither pathological examination nor the analysis of contents of cysts was performed in our cases.

All of our patients were diagnosed with glaucomas or ocular hypertension. It has been suggested that the high intraocular pressure was caused by a blockage of the trabecular meshwork by the glycosaminoglycan, or a false pressure due to the rigidity of the cornea in the MPSs patients. On the other hand, angle closure that is caused by multiple bilateral iridociliary cysts was performed in our cases.

A case of encephalocraniocutaneous lipomatosis

Encephalocraniocutaneous lipomatosis (ECCL) is a rare neurocutaneous syndrome characterised by cranial and facial asymmetry, cutaneous lesions, central nervous system abnormalities, and ocular abnormalities. A case is described of a young man who presented with limbal dermoids, subcutaneous lipomas, and scalp alopecia. Further investigation revealed cranial and facial asymmetry, intracranial lipomas, and calcification and an arachnoid cyst, supporting a diagnosis of ECCL. This patient also had the additional ocular abnormality of bilateral optic disc colobomas, an association with ECCL not previously reported in the literature.

Case report

A 23 year old Asian man first presented to the eye clinic aged 16 with a left conjunctival lesion. On examination he had bilateral conjunctival dermopomas, preauricular lipomas, and bilateral optic disc colobomas. Surgery was not advised at this time and no follow up was arranged, although photographs were taken (Fig 1). Aged 20, he presented to the eye clinic again. Before excision biopsy of the left conjunctival dermopoma, a computed tomograph (CT) scan of the head was requested to delineate the posterior extent of the lesion. This revealed some asymmetry of the skull vault, intracranial calcification within the right cerebellar hemisphere, and a possible cyst in the left parietal area (Fig 2 (left)). Subsequent examination by a neurologist was entirely normal with no stigmata of the phacomatoses. Histology of the conjunctival lesion confirmed a complex compound choristoma comprising collagenous tissue with fat and a focus of cartilage.

Three years later the patient was seen by a dermatologist complaining of a lesion on his left eyebrow, which was clinically a lipoma. Skull and facial X rays revealed asymmetry of the skull vault and facial bones including the zygomatic arches. Further investigation in the form of an magnetic resonance imaging (MRI) scan of the brain showed lipomas within the subcutaneous fat of the scalp and also intracranial lipomas. In addition, there was an arachnoid cyst anterior and inferior to the left temporal lobe (Fig 2 (right)). There was no connection between the soft tissue tumour on the left eyebrow and the intracranial cavity. Subsequent histology of the excised lesion revealed a lipoma.

Review of the patient’s childhood medical records revealed that since birth he had had large patches of scalp alopecia and aged 7 he was noted to have a large suprapubic fat pad. A final diagnosis of ECCL was made based on the findings of limbal dermoids, subcutaneous lipomas, scalp alopecia, cranial and facial asymmetry, intracranial lipomas and calcification, and an arachnoid cyst.

Comment

ECCL is a rare neurocutaneous syndrome of unknown aetiology, first described by Haberland and Perou in 1970. All cases described in the literature have been sporadic and there does not appear to be any geographic, racial,
or sex predilection. The syndrome is characterised by cranial and facial asymmetry, cutaneous lesions, central nervous system abnormalities, ocular abnormalities, and occasionally visceral lipomas. The abnormalities tend to be unilateral, although bilateral involvement has been described.\(^1\)\(^2\)

Cutaneous lesions consist mainly of subcutaneous scalp lipomas with overlying alopecia but lipomas involving the limbs and paravertebral areas have also been reported.\(^3\)\(^4\) Poplar skin lesions are also common and histologically have been found to be lipomas, fibrolipomas, and angiolipomas.\(^5\) Central nervous system abnormalities are numerous and include cerebral lipomas, cerebral calcifications, ventricular dilatation, cerebral atrophy, arachnoid cysts, seizures, spasticity, and mental retardation.\(^6\) The most common ocular lesions in ECCL are epibulbar choristomas and small skin nodules around the eyelids, which histologically represent connective tissue naevi.\(^7\) Other reported ocular abnormalities include a subcutaneous choristoma of the eyelid,\(^8\) a small tag of tissue in the anterior chamber,\(^9\) a persistent posterior hyaloid system,\(^10\) a dysplastic iris,\(^11\) papillodema, and epicanthus inversus plus hypertelorism.\(^12\)

The patient in this case report demonstrates the main features of ECCL: cranial and facial asymmetry, subcutaneous lipomas and scalp alopecia, intracranial calcification and lipomas, an arachnoid cyst, and limbal dermoids. There was also the additional finding of optic disc colobomas, an association with ECCL not previously reported in the literature. Other common findings in ECCL are seizures and mental retardation, but neither were apparent in this case and may explain the comparatively late presentation of this patient; most patients present in early childhood.\(^13\)\(^14\) The abnormalities in ECCL are usually unilateral but bilateral involvement does occur\(^15\) and was evident in this patient. Although epibular choristomas and limbal dermoids can occur sporadically in isolation or in a Mendelian inherited pattern\(^16\) there may be systemic associations such Goldenhar’s syndrome, the linear naevus sebaceous syndrome, or ECCL and the ophthalmologist should consider these diagnoses when a patient presents with an epibular choristoma or limbal dermoid.

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Figure 1  [Top] Photograph of left conjunctival dermolipoma. [Bottom] Photographs of right and left optic disc colobomas.
Bilateral non-specific orbital inflammation (orbital “pseudotumour”), posterior scleritis, and anterior uveitis associated with hypothyroidism in a child

Posterior scleritis and non-specific orbital inflammation (NSOI), also known as orbital “pseudotumour,” are rarely seen in children. Paediatric posterior scleritis and NSOI seldom have an underlying systemic association and, to our knowledge, hypothyroidism has not been reported as an association, although thyroid abnormalities are recognised in adults.

We present a case of a child with bilateral anterior NSOI, posterior scleritis, and anterior uveitis who was also found to be hypothyroid.

Case report
A previously fit and well 13 year old girl presented with a 3 week history of bilateral red eyes and painless puffiness in her upper and lower lids for 1 week. She had had a headache for 10 days which had failed to respond to oral antibiotics. Systemic inquiry revealed a sore throat for 10 days but no other symptoms, in particular no thyroid related symptoms. Her visual acuity was 6/9 right, and 6/18 unaided, improving to 6/12 with pinhole in the left eye. She read all the Ishihara plates with the right eye, but missed three out of 17 plates with the left. No relative afferent pupillary defect (RAPD) was present. She had mild left peri-orbital swelling which was not erythematous or tender. There was no proptosis. She had full extraocular movements with no diplopia. The right eye had signs of mild anterior uveitis with 1+ of cells while the left eye had 2+ of cells in the anterior chamber. There was no evidence of posterior segment involvement, with normal vitreous and fundi.

She was commenced with 2 hourly dexamethasone eye drops to both eyes and on review 3 days later her vision improved to 6/6 in both eyes. A week later, she returned with pain and increasing periorbital swelling, left eye greater than right. Her visual acuity was reduced to 6/12 (right eye) and 6/60 improving to 6/18 with pinhole in the left eye. She had mild diffuse anterior uveitis. The left disc was more swollen than the right and retinal striae were present in both eyes (Fig 1). There was no evidence of vitreous or chorioteratal inflammation. An orbital computed tomograph (CT) scan suggested a diagnosis of bilateral anterior NSOI without enlargement of the extraocular muscles (Fig 2). A-mode ultrasonography showed bilateral diffuse thickening of the posterior coats of the eyes (maximum left thickness of 4.4 mm) and fluid in Tenon’s capsule consistent with scleritis.

Full blood count, urea, and electrolytes, liver function tests, serum angiotensin converting enzyme, serum calcium, autoantibodies (including ANA, ANCA, and rheumatoid factor), C reactive protein (CRP), and chest x ray were normal. She had negative serology for Borrelia, HTLV-1, and HTLV-2. Her erythrocyte sedimentation rate (ESR) was 32 mm in the first hour and a mildly raised Ig M was found. She was biochemically hypothyroid (raised TSH of 25.5 mU/l (0.4–4.0) and T4 of 10.6 pmol/l (9–20)) with positive anti-thyroid M antibodies. Thyrotrophin releasing hormone stimulation test confirmed primary hypothyroidism.

Treatment was commenced with prednisolone 50 mg which was reduced gradually over 3 months with resolution of her symptoms and signs. She had no relapse at 1 year follow up. She was also treated with thyroxine for her hypothyroidism.

Comment
The distinction between posterior scleritis and diffuse anterior NSOI is not always clear. As in this case, they commonly have evidence of inflammatory changes of the posterior coats of the eye and pericocular tissues.

In a series of 29 paediatric NSOI cases, there was an association with peripheral blood eosinophilia, raised ESR, and positive ANA. Normal thyroid function tests (TFTs) were present in all of the nine children tested.

Other reports of paediatric NSOI do not state whether TFTs were performed. Similarly, children with posterior scleritis tend not to have any clinical or laboratory evidence of associated systemic disease, but TFTs are not specifically mentioned.

Our patient responded well to steroid treatment with no relapse at 1 year.

Scleritis with uveitis, although rare in children, should be recognised as part of the differential diagnosis of acute paediatric orbital inflammation. Hypothyroidism may be an incidental finding in our case, but the role of thyroid autoimmunity in children with NSOI and scleritis needs to be further investigated.

References

Unilateral enlargement of the blind spot: a diagnostic dilemma

Unilateral blind spot enlargement occurs as an isolated entity (acute idiopathic blind spot enlargement) or in association with other conditions such as multiple evanescent white dot syndrome, multifocal choroiditis with panuveitis, or punctate inner chorioidopathy. It remains controversial whether blind spot enlargement in these conditions serves to unify them as a diagnostic group. The patient presented here had clinical features suggesting “diagnostic overlap” with some of these conditions, suggesting that diagnostic “lumping” of these diseases may have more logic than “splitting” them.

Case report
A 30 year old female patient presented to us in September 2000 with a blind spot close to the centre of vision in her left eye. She was uncertain as to how long it had been present, having noticed it only when the other eye was temporarily covered by chance. She was fit and well, with no recent viral illness or previous eye problems. Her acuity with myopic correction (−3.00 dioptre sphere right, −3.25 dioptre sphere left) was recorded at 6/6 right and left, and discrete foci of chorioretinal scarring were noted above and nasal to the optic disc in the left eye (Fig 1). There was no evidence of vitreous inflammatory activity in
Fletcher Comment has gradually decreased over 12 months. and late leakage at the optic disc margin. Fluorescein angiography demonstrated window and masking on the right it was normal. 

either eye. Humphrey C24-2 testing revealed an enlarged blind spot on the left (Fig 2), while on the right it was normal. Fluorescein angiography demonstrated window and masking leaks. Fluorescein angiography demonstrated window and masking leaks.

Comment

Fletcher et al. were the first to describe a syndrome of acute idiopathic blind spot enlargement (AIBSE) without optic disc oedema in a series of seven patients. This phenomenon has since become well recognised both as an isolated finding, and in association with various forms of chorioretinitis including multiple evanescent white dot syndrome (MEWDS), multifocal choroiditis with panuveitis (MCP), acute macular neuroretinitis (AMN), diffuse subretinal fibrosis (DSF), and punctate inner choroidopathy (PIC). Together, these diagnoses span a wide spectrum of clinical disease expression, and it remains controversial whether blind spot enlargement serves to unify the group or whether other clinical features are sufficiently distinctive for them to be regarded as separate disorders.

This patient is young, myopic and female, and presented with unilateral blind spot enlargement and chorioretinal scarring in the absence of acute symptoms such as photopsias. These features make it difficult to assign a specific diagnosis, as discussed below.

AIBSE and MEWDS share many common features. Both tend to present acutely with visual loss and photopsias in young myopic females, and follow a prodromal viral illness. They occasionally recur and can both be bilateral. The visual prognosis is good, with early disappearance of the white spots and later resolution of blind spot enlargement in most, though not all, cases. The principal distinguishing feature of MEWDS is the presence of white spots at the level of the outer retina or retinal pigment epithelium (RPE), and the variable presence of vitreous cells, retinal vascular sheathing, and optic disc swelling. Since the white spots can be fleeting and hard to see, it has led some to believe that AIBSE is really a subset of MEWDS patients first seen after resolution of the white spots. However, this has been strongly refuted by Hoyt and Imes, who argued that, in contrast with MEWDS, the peripapillary visual loss in AIBSE is absolute in density and has steep edged margins. The presence of chorioretinal scarring in our patient would not be in keeping with either of these conditions.

In multifocal choroiditis with panuveitis (MCP), patients again present acutely with visual loss, scotomata, and/or photopsias.

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Figure 1 Fundus photograph, left eye. Chorioretinal scarring is present superonasally. Anterior and/or posterior uveitis is present and focal of choriorretinitis become apparent, most commonly in the peripapillary region. Inflammation leads to a variable degree of chorioretinal scarring, which can gradually enlarge and develop a subretinal component. Blind spot enlargement can occur, and does not always correlate with disc swelling or peripapillary choroidal scarring. MCP tends to be recurrent, with asymmetric bilaterality. Subfoveal choroidal neovascularisation (CNV) is the commonest cause of permanent vision loss, with resolution of blind spot enlargement occurring in most patients. Presumed ocular histoplasmosis syndrome (POHS) can cause similar choroidal scarring, but is not usually associated with uveitis or blind spot enlargement, and has no female preponderance. So called "pseudo POHS" has been linked with MEWDS, AMN, and AIBSE, though the absence of acute symptoms or vitreous inflammatory activity in our patient is at variance with most previous reports of patients with MCP or POHS.

Punctate inner choroidopathy (PIC) is similar to MCP in many ways, including the presence of an enlarged blind spot in some cases. It is rarer than MCP and no cells or other signs of inflammation are seen in the vitreous or anterior chamber. Our patient would perhaps be closest to PIC in clinical findings, though again the lack of acute symptoms or bilaterality would be atypical.

AMN is less well associated with blind spot enlargement. It occurs predominantly in young adult females, and presents with rapid onset of dense paracentral scotoma. Reddish brown retinal lesions corresponding to the dense scotoma become evident, and are best seen with red free light. Uveitis is not present, and the scotoma diminish over months or years. Diffuse subretinal fibrosis (DSF) is very rare and regarded by some as a variant of MCP. In addition to many of the clinical features of MCP already discussed, this condition is distinguished by widespread and progressive subretinal fibrosis not preceded by CNV. The blind spot enlargement in AIBSE, MEWDS, MCP, PIC, AMN, and DSF, coupled with their tendency to present in young adult females, has led to a proposal that they be
grouped under the term “acute zonal occult outer retinopathy” (AZOOR).1 Added to the clinical similarities already described, Jacobson et al1 demonstrated electroretinographic (ERG) abnormalities in a group of 24 AZOOR patients, though in some cases there were only subtle intereye differences detected. It was concluded that ERG findings help to unify this diagnostic group, as well as indicating that the primary pathophysiology lies at the level of the photoreceptor outer segment. This view was not supported by Jampol and Wiredu, who argued that the above entities were sufficiently distinctive to warrant “splitting” rather than “lumping.”

Our patient does not fit neatly into any of the diagnoses discussed above, and the principal clinical features of blind spot enlargement with chorioretinal scarring in the absence of acute symptoms or evidence of vitritis suggest that there is a degree of diagnostic overlap in her case. To the extent that a single case report can inform this debate, it does indicate that some patients do not fit neatly into diagnostic groups, strengthening the case for those who would “lump” these diagnoses rather than “split” them. Perhaps there are other cases which remain unreported because of this diagnostic uncertainty.

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Acquired ocular toxoplasmosis in pregnancy

We describe the management of a case of acquired ocular toxoplasmosis that occurred in the first trimester of pregnancy.

Case report

A 27 year old apparently healthy Pakistani woman, at 9 weeks’ gestation, presented to the Birmingham and Midland Eye Centre with a 1 week history of blurred vision in the left eye. She had no past ocular problems. Her first uncomplicated pregnancy was 2 years previously. On examination, the best corrected visual acuities were 6/6 in each eye. The anterior segments were normal and the intraocular pressures were 16 mm Hg right eye, 14 mm Hg left eye. Funduscopy showed a diffuse, elevated white lesion in the left retina half a disc diameter in size located one disc diameter superior to the fovea (Fig 1). There was an area of associated perivascular sheathing and minimal vitreous activity. An active left retinochoroiditis with associated retinal vasculitis was diagnosed. The right fundus was normal.

Sero logical testing revealed antibodies to Toxoplasma gondii at a titre of 1:4096 (2000 IU/mL) using the dye test and a toxoplasma IgM enzyme linked immunosorbent assay (ELISA) on the patient’s peripheral blood was positive. Subsequent serological tests at the PHLS Toxoplasma Reference Unit, Swansea, confirmed these results and IgG avidity testing demonstrated that the infection was acute (Table 1). The polymerase chain reaction (PCR) on peripheral blood for T gondii was negative. At 3 weeks after presentation (12 weeks’ gestation), the patient’s visual acuities were unchanged. There was now occlusion of a small retinal arteriole crossing the lesion. After consultation with the obstetrician the patient decided to proceed with the pregnancy. She was commenced on oral spiramycin 1 g three times daily.

Eight weeks after presentation (17 weeks’ gestation) the vision remained the same and the focus of retinochoroiditis was beginning to scar. The patient underwent an amniocentesis for PCR against T gondii. This was negative suggesting the fetus was unaffected. Spiramycin was continued until the end of pregnancy. Sequential fetal ultrasounds were normal.

At term the patient gave birth to a healthy baby girl. Polymerase chain reaction (PCR) and culture for T gondii were negative on placental tissue. Nevertheless, the dye test on cord blood was positive at 500 IU/mL. As toxoplasma IgM ELISA, IgM, and IgA ISAGA on cord blood were negative, the positive dye test was probably detecting passively transferred maternal IgG. The mother’s vision remained normal, and only a small, pigmented scar was present (Fig 2).

Table 1 Serum dye test titres, IgM levels, and IgG avidity levels

<table>
<thead>
<tr>
<th>Time (days) after onset of symptoms</th>
<th>Dye test titre</th>
<th>Dye test (IU/mL)</th>
<th>ELISA IgM</th>
<th>IgG avidity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>1/4096</td>
<td>2000</td>
<td>Positive</td>
<td>20</td>
</tr>
<tr>
<td>20</td>
<td>1/2048</td>
<td>1000</td>
<td>Positive</td>
<td>19</td>
</tr>
<tr>
<td>103</td>
<td>1/2048</td>
<td>1000</td>
<td>Positive</td>
<td>--</td>
</tr>
</tbody>
</table>

Figure 1 Left fundus showing active retinochoroiditis with retinal vasculitis.

Figure 2 Left fundus showing healed area of previous retinochoroiditis.

Comment

In the United Kingdom ocular toxoplasmosis is normally thought to occur through the congenital route, although recent evidence suggests that we may be underestimating the amount of acquired disease.2

Overall, about 40% of primary maternal infections lead to congenital infection of children, with a transplacental transmission rate of T gondii reported to be 3% in the first trimester, 22% in the second, and 63% in the third.3

In our case the features supporting an acquired aetiology included lack retinal scars from previous infection, and the positive IgM serology and IgG avidity results.4 Sera taken early in infection (<3 months) usually have avidity levels of less than 30%. Most sera taken later in infection (>6 months) have avidity levels of greater than 40%.

There are conflicting reports on the value of the PCR to detect toxoplasma DNA.5–11 This may represent the different target DNA strands used in the studies. The negative PCR result on the mother’s serum in our report is therefore not surprising. A negative PCR on amniotic fluid suggested that the fetus was not infected by toxoplasma, which was supported by the normal fetal ultrasounds.

Spiramycin is effective in reducing the risk of transmission to the fetus and therefore was given throughout pregnancy. This treatment may have contributed to the resultant good outcome for both mother and fetus.

Determining an aetiology of acquired T gondii infection was important in this patient as it allowed the most appropriate management plan to be initiated resulting in an excellent outcome. Acquired ocular toxoplasmosis occurring in pregnancy is rare and we hope this case report will raise the awareness of this unusual presentation.

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which she had made a full recovery. The infection (chickenpox) 3 weeks earlier, from an uneventful episode of primary varicella zoster practitioner with a red and painful right eye

This 11 year old girl presented to her general Case report

We present the case of an 11 year old myopic girl who developed significant refractive correction following varicella disciform stromal keratitis

Spontaneous reduction in myopic correction following varicella disciform stromal keratitis

We present the case of an 11 year old myopic girl who developed significant refractive changes due to corneal scarring following varicella disciform stromal keratitis in her right eye. This has markedly reduced the myopia in her right eye and resulted in significant anisometropia.

Case report

This 11 year old girl presented to her general practitioner with a red and painful right eye with reduced vision. She had had an uneventful episode of primary varicella zoster infection (chickenpox) 3 weeks earlier, from which she had made a full recovery. The general practitioner diagnosed her as having conjunctivitis and she was treated with topical chloramphenicol. One week later, the redness and pain had settled but her vision remained hazy. As a result she was referred to the eye department. Examination confirmed a remnant of visual acuity to 6/24 (Snellen) in the eye department. As there was no change in her symptoms or in the eye for distant vision rather than wearing the

Figure 2. Corneal topography of the affected right eye (top) shows a flattening of the corneal surface secondary to disciform corneal scar. Compare this with the unaffected myopic left eye, which has a steeper corneal surface (bottom).

Figure 1. Anterior segment photograph of the right eye showing mild corneal scarring.

References


Comment
This is an unusual case of spontaneous myopia correction following corneal scarring secondary to varicella zoster disciform stromal keratitis. Primary varicella zoster infection (chickenpox) is a diffuse vesicular skin rash mainly affecting children and is usually self-limited. Common ocular findings are eyelid vesicles or marginal erosions and acute conjunctivitis. Corneal changes are infrequent but can occur during the first week or two after the onset of chickenpox. Disciform stromal keratitis is an unusual but well-recognized complication of primary varicella zoster infection. Varicella viral antigen and intracellular viral inclusions have been found in the corneal epithelium of affected eyes. Wilhelmus et al reported five cases and reviewed the literature and found that this condition is typically unilateral, has a delayed onset, typically several weeks after the onset of skin rash. The complications following disciform stromal keratitis identified in this review are corneal scarring, neurotrophic keratopathy, iridocyclitis with secondary glaucoma, and iris stromal atrophy. The principal cause of loss of vision is corneal scarring. In our case, the corneal scarring had resulted in the flattening of the corneal surface thus reducing the degree of myopia significantly. This was highly unusual and had contributed to improving theunal visual acuity in her affected eye. The effect on the cornea was similar to post LASIK (laser assisted in situ keratomileusis) and PRK (photorefractive keratectomy) in that the cornea appearance was similar as was the topography. In addition, the degree of corneal haze was limited and there had been a degree of stability for a period of over 1 year.

She was unable to tolerate the full refractive corrections because of the significant anisometropia and aniseikonia. Despite this she was pleased as she can see 6/9 unaided and considered herself myopic and aniseikonia. Despite this she was receiving HDSB substitution therapy. In view of her young age, she would rather not wear her rather high myopic spectacles correction. In our patient, the keratitis settled despite her refractive state and her lack of visual improvement for a period of over 1 year. The bacterial and fungal cultures from both blood and anterior chamber tap were negative. Treatment was begun with intravenous fluconazole associated with three intravitreal amphotericin B injections (IVT). After 15 days of therapy, he was discharged on oral fluconazole. Ten days later, the endophthalmitis relapsed with development of a second paramacular necrotising lesion. A posterior vitrectomy was performed. The vitreous cultures were negative for Candida albicans. He was treated again with intravenous amphotericin B, along with amphotericin B IVT with success.

Case 2
A 27 year old man, a former heroin misuser, was receiving HDSB substitution therapy. A few weeks following such an injection, he developed a skin abscess in which cultures revealed C albicans, posterior cervical lymphadenopathy, nodules of the scalp, and arthritis of the left wrist. Three weeks later he complained of decreased vision in his right eye. Funduscopy revealed a parapapillary white lesion and few white snowballs (Fig 2) Treatment was instituted with intravenous amphotericin B and fluconazole but the patient left the hospital against medical advice 4 days later.

Case 3
A 25 year old man, with history of heroin misuse, was referred for blurry vision and floaters in the right eye. His ocular symptoms started following an intravenous HDSB injection prepared with rotten lemon juice. Funduscopy revealed a parapapillary white lesion and few white vitreous snowballs (Fig 3) Improvement was obtained after 14 days of intravenous fluconazole and amphotericin B IVT.

Case 4
A 30 year old man, a former heroin misuser, was referred for decreased vision in the left eye. He had been using intravenous HDSB by dissolving the tablets in preserved lemon juice. Ten days earlier, he had a disseminated pustular rash with folliculitis over the chest, shoulders, and back. On funduscopy, there was a 2 + vitritis and a white chorioretinal lesion below the inferotemporal arcade (Fig 4). He improved after a treatment with intravenous amphotericin B rapidly switched to intravenous fluconazole combined with two amphotericin B IVT.

Comment
Endogenous candida endophthalmitis diagnosis is usually based on the combination of the
and typical fundus lesions. In our cases, the diagnosis of presumed candida endophthalmitis was based on (1) the characteristic clinical setting, (2) the typical ocular involvement that was characterised by creamy-white chorioretinal lesions with white balls and vitritis, and (3) the response to antifungal therapy. Vitrectomy was performed only in one case. It was not performed in three patients because they presented an isolated chorioretinitis or associated with a mild vitritis and a characteristic clinical presentation. Patients were treated with intravenous amphotericin B or intravenous fluconazole and intravitreal injections of amphotericin B, except in one patient who refused intravitreal injections.

In the mid-1980s, in France, an outbreak of candidiasis followed the introduction of the drug market of a new brown heroin. The hypothesis that the lemon juice used to dissolve the heroin might have been contaminated with C albicans was demonstrated. Our cases also seem to confirm that the fungi probably come from the lemon juice or the patient himself rather than from the buprenorphine itself. Since March 1995, substitution therapy with HDSB tablets (Subutex) is approved for licence in France. Unfortunately, 8% of the patients enrolled in substitution programmes continued to use the intravenous route. This drug is now widely prescribed in France. Recently, in Europe, this treatment obtained the authorisation for commercialisation in 13 European countries. Our report demonstrates the need to inform general practitioners, pharmacists, and patients of the risks involved with the intravenous use of substitute agents.

### Aetiology of microbial keratitis in northern Tanzania

The incidence of corneal blindness caused by microbial keratitis in the developing world is far higher than that in the developed world. Microbial keratitis has become a more prominent cause of corneal blindness in east Africa as the uptake of measles immunisation improves, reducing measles keratitis scarring, and with improved recognition and treatment of vitamin A deficiency, reducing its associated xerophthalmia and subsequent corneal scarring.

It has been shown that in tropical climates, keratitis of fungal aetiology is much more prevalent than in temperate climates. Little information is available about microbial keratitis in east Africa. The aim of this study was to identify the causative organisms of the condition seen in patients presenting to the Kilimanjaro Christian Medical Centre (KCNC) hospital in northern Tanzania, east Africa. KCNC is one of the largest hospitals in Tanzania, situated on the foothills of Mount Kilimanjaro, serving five regions in northern and central Tanzania—Kilimanjaro, Arusha, Singida, Tanga, and Dodoma with a population of approximately eight million people.

### Clinical cases

Patients referred to, or presenting for the first time, to KCNC with clinical signs of microbial keratitis, were prospectively recruited to the study, between May 1997 and April 1998.

### Table 1

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Range</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [years]</td>
<td>8–97</td>
<td>44</td>
</tr>
<tr>
<td>Time from onset of symptoms to presentation (days)</td>
<td>1–90</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>29</td>
<td>65.9</td>
</tr>
<tr>
<td>Female</td>
<td>15</td>
<td>34.1</td>
</tr>
<tr>
<td>Occupation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Agricultural</td>
<td>22</td>
<td>50</td>
</tr>
<tr>
<td>Student/school</td>
<td>6</td>
<td>13.6</td>
</tr>
<tr>
<td>Driver/conductor</td>
<td>4</td>
<td>9.1</td>
</tr>
<tr>
<td>Maasai*</td>
<td>4</td>
<td>9.1</td>
</tr>
<tr>
<td>Retired</td>
<td>4</td>
<td>9.1</td>
</tr>
<tr>
<td>Soldier/guard</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Miner</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Teacher</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Businessman</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Treatment before presentation at KCNC of all cases (culture negative cases only)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nil</td>
<td>11 (6)</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>*Local remedy</td>
<td>4 (2)</td>
</tr>
<tr>
<td></td>
<td>Inappropriate treatment</td>
<td>3 (1)</td>
</tr>
<tr>
<td></td>
<td><em>Appropriate</em>† treatment, inadequate intensity</td>
<td>18 (5)</td>
</tr>
<tr>
<td></td>
<td><em>Appropriate</em>† treatment</td>
<td>4 (2)</td>
</tr>
<tr>
<td></td>
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<td>4 (2)</td>
</tr>
<tr>
<td>History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vegetative trauma</td>
<td>14</td>
<td>32</td>
</tr>
<tr>
<td>All trauma</td>
<td>17</td>
<td>38.6</td>
</tr>
<tr>
<td>Previous corneal scar</td>
<td>14</td>
<td>32</td>
</tr>
<tr>
<td>Lid problems</td>
<td>2</td>
<td>4.6</td>
</tr>
<tr>
<td>Nil</td>
<td>11</td>
<td>25</td>
</tr>
<tr>
<td>Organisms cultured</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fungi</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fusarium solani</td>
<td>4</td>
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<tr>
<td>Fusarium sp</td>
<td>5</td>
<td>11.4</td>
</tr>
<tr>
<td>Aspergillus fumigatus</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Cladosporium sp + Bacillus sp</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>Unidentified fungus + Staphy epidermidis</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>All fungi</td>
<td>12</td>
<td>27.3</td>
</tr>
<tr>
<td>Gram positive bacteria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Staphylococcus epidermidis</td>
<td>3</td>
<td>6.8</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>2</td>
<td>4.6</td>
</tr>
<tr>
<td>Gram negative bacteria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>6</td>
<td>13.6</td>
</tr>
<tr>
<td>Proteus sp</td>
<td>1</td>
<td>2.3</td>
</tr>
<tr>
<td>No organism cultured</td>
<td>20</td>
<td>45.5</td>
</tr>
</tbody>
</table>

*Empirical herdsman.
†Empirical treatment with broad spectrum topical antibiotic.

### References


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Patients with corneal ulceration without infiltration were excluded. These consisted of three patients with herpes simplex keratitis, two patients with Mooren’s ulcer, and one patient with a neurotrophic ulcer. Two patients with secondary infection of a recent penetrating injury were excluded. Two children were unable to undergo slit lamp examination and corneal scraping for microbiology specimens and were also excluded from the study.

Forty-four corneal ulcers were seen. Thirty-eight of the patients had visual acuity in the affected eye of 6/60 or worse, and the mean greatest diameter of the infiltrate on presentation was 5.1 mm. Organisms were cultured from 24 of the 44 ulcers (54.6%).

Fifteen of these cultures were fungal. Larger diameter ulcers were more likely to be culture positive and have poorer outcomes: mean ulcer diameter was 6.0 mm in culture positive ulcers and 3.7 mm in culture negative ulcers. Fungal growth had been predicted by positive microscopy for fungal elements in nine of the 12 cases that grew fungi (75%), and there was never positive fungal microscopy without fungal growth. All fungal positive isolates had had Gram-positive cocci identified on Gram staining initially but, in contrast, there were three cases where Gram positive cocci were initially seen on Gram staining, but cultures had grown other organisms (two fungi, one Gram-negative bacillus).

Full details are shown in Table 1.

Comment

In this study fungal keratitis accounted for 50% of culture positive cases of microbial keratitis in northern Tanzania, with the majority of these cases (42%) yielding pure fungal isolates on culture. These figures are similar to those published from west Africa, where 56% of microbial keratitis was caused alone or in part by fungi. As in studies from the United States, west Africa, and southern India, the most common genus of fungus isolated was that of the filamentous fungus Fusarium.

There may have been a bias towards fungal ulcers in this study. KCMD is a referral centre, receiving severe ulcers from primary care centres, where topical antibacterial treatments are generally available and topical antifungal treatments are generally not. If the culture negative ulcers were predominantly bacterial ulcers, the relative frequency of fungal keratitis in this study would be artificially high. However culture negative ulcers had a similar range of treatments to culture positive ulcers before presenting to KCMD, as can be seen from Table 1. There is no evidence to suggest the culture negative ulcers were predominantly of treated bacterial rather than fungal aetiology.

Study design limitations precluded any estimation of prevalence of microbial keratitis in the region, which would have been useful. The findings relate to northern and part of central Tanzania; with a similar climate, risk factors and primary care set up, the aetiology of microbial keratitis is likely to be similar in the wider region. The study set out Gram positive isolates had had Gram-positive cocci as part of the microbiological examination, but this became impossible after logistical difficulties.

The most important factor in outcome was the initial size of the ulcer on examination. Larger ulcers were more likely to be culture positive and to have a poor outcome, such as a blinding corneal scar or requiring excisionation. Culture results were nevertheless useful in ascertaining antibiotic sensitivities, and in adding antifungal treatment where necessary. Any patient undergoing excisionation had usually been an inpatient for several weeks with a painful blind eye, and had tried multiple treatment regimens, and had often requested excisionation himself. The overall rate of excisionation was 25%. Although there is little published material on outcomes of microbial keratitis in the developing world, this figure compares with a series of culture positive fungal ulcers in Madurai, India, where 20% were classified as “severe, with little prospect of recovery”.

Clearly, awareness of proper treatment regimens in northern Tanzania is paramount, particularly at the place of first contact, the village health centre, to prevent this overall picture of severe corneal ulcers with very poor visual prognosis. Treatment regimens should also take into account the high proportion of fungal keratitis. Microscopy looking specifically for fungal elements is a simple, quick, and useful test that could help direct initial treatment, along with clinical appearances suggestive of fungal infection. This might be more economically viable and practical than blanket coverage of all keratitis with an antifungal topical treatment in the first line therapy. Unfortunately consistently effective antifungal topical treatments are not widely available in this part of Africa.

Future studies could examine the efficacy of current treatments for fungal keratitis and how best to improve awareness of appropriate treatment regimens in the region.

Acknowledgements

This work was supported by grants from the British Council for the Prevention of Blindness and the Uverscroft Foundation.

We are grateful to the hospital director, the staff, and the patients of KCMD for their collaboration in the above study.

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References


This CD video atlas with accompanying text is the first in a series of three such atlases by these authors and is the first such oculoplastic atlas available. The atlas consists of 95 pages of text supplemented with black and white drawings in six chapters. Two CDs contain all the video sequences and these follow the same chapter layout as the text: surgical anatomy of the eyelid, entropion, ectropion, eyelid retraction, ptosis, and eyelid reconstruction.

There are a total of 17 video procedures typically lasting between 3 and 5 minutes and a 10 minute cadaveric anatomy sequence. The video quality is very acceptable for individual viewing but does not project as successfully. All sequences are well narrated and informative, covering basic and some more advanced oculoplastic procedures. The need for free skin grafting in one of the cases demonstrated is perhaps questionable but this does not detract from the educational value of the atlas. A number of associated procedures are covered in the videos—for example, harvesting skin/cartilage; however, these cannot be instantly located from the menus and an additional separate section including such procedures could perhaps have been usefully included.

The text is clearly written and concise but is not comprehensive and provides limited information relating to the particular procedure selection. A good deal of the text is covered in the narration accompanying the video.

The atlas is aimed at all those who have an interest in oculoplastic surgery but is particularly relevant to the ophthalmic surgeon. It will be a valuable asset for teaching residents and fellows and should make interesting viewing for all those routinely practising oculoplastic surgery.

From a personal point of view, I am always fascinated to watch other surgeons at work, as technical approaches to the same problem/operation are often quite diverse. This is one of the best ways to keep up to date and improve your own surgery.

Overall, this atlas will be a valuable asset to all those in training and a useful tool for those practising oculoplastic surgical procedures. I look forward to the next two atlases in the series and to further editions.

Garry Shuttleworth
for Eye Health, Institute of Ophthalmology, 11–43 Bath Street, London EC1V 9EL, UK (tel: +44 (0)20 7608 6910; fax: +44 (0)20 7250 3207; email: eyeresource@ucl.ac.uk; website: www.jceh.co.uk). Annual subscription (4 issues) UK£25/US$40. 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).

**Second Sight**

Second Sight, a UK based charity whose aims are to eliminate the backlog of cataract blind in India by the year 2020 and to establish strong links between Indian and British ophthalmologists, is regularly sending volunteer surgeons to India. Details can be found at the charity website (www.secondsight.org.uk) or by contacting Dr Lucy Mathen (lucymathen@ yahoo.com).

**Specific Eye Conditions (SPECS)**

SPECific Eye Conditions (SPECS) is a not for profit organisation which acts as an umbrella organisation for support groups of any conditions or syndrome with an integral eye disorder. SPECS represents over fifty different organisations related to eye disorders ranging from conditions that are relatively common to very rare syndromes. We also include groups who offer support of a more general nature to visually impaired and blind people. Support groups meet regularly in the Boardroom at Moorfields Eye Hospital to offer support to each other, share experiences and explore new ways of working together. The web site www.eyeconditions.org.uk acts as a portal giving direct access to support groups own sites. The SPECS web page is a valuable resource for professionals and may also be of interest to people with a visual impairment or who are blind. For further details about SPECS contact: Kay Parkinson, SPECS Development Officer (tel: +44 (0)1803 524238; email: k@eyeconditions.org.uk; www.eyeconditions.org.uk).

**The British Retinitis Pigmentosa Society**

The British Retinitis Pigmentosa Society (BRPS) was formed in 1975 to bring together people with retinitis pigmentosa and their families. The principle aims of BRPS are to raise funds to support the programme of medical research into an eventual cure for this hereditary disease, and through the BRPS welfare service, help members and their families cope with the everyday concerns caused by retinitis pigmentosa. Part of the welfare service is the telephone helpline (+44 (0)1280 860 363), which is a useful resource for any queries or worries relating to the problems retinitis pigmentosa can bring. This service is especially valuable for those recently diagnosed with retinitis pigmentosa, and all calls are taken in the strictest confidence. Many people with retinitis pigmentosa have found the Society helpful, providing encouragement, and support through the Helpline, the welfare network and the BRPS branches throughout the UK. (tel: +44 (0)1280 821 334; email: lynda@brps.demon.co.uk; web site: www.brps.demon.co.uk)

**Ophthalmic Anesthesia Society (OAS) 16th Scientific Meeting**

The 16th Scientific Meeting of the OAS will be held on 4–6 October 2002 in The Westin, Michigan Avenue, 909 North Michigan Avenue, Chicago, USA (reservations +1 800 228 3000). Further details: OAS, 793-A Foothill Blvd, PMB 110, San Luis Obispo, CA 93405, USA (tel: +1 805 771 8300; web site: www.eyeanesthesia.org).

**BEAVRS Meeting**

The next BEAVRS meeting will be held in the Dalmahoy Hotel near Edinburgh on 31 October to 1 November 2002. Further details: Susan Campbell, Medical Secretary, Gartnavel General Hospital (email: susan.j.campbell.wg@northglasgow.scot.nhs.uk).

**Cornea 2002—Celebrating 50 Years of Eyebanking**

The Cornea 2002 meeting will be held in Le Meridien Hotel, London, Gatwick on 14–15 November 2002. Subjects to be covered will include eye banking, penetrating and lamellar keratoplasty, stem cell restoration, keratoprossthesis, advanced keratoplasty techniques, paediatric cornea, keratorefractive surgery, and intraocular refractive surgery. Spaces are limited and a beneficial package rate is available prior to 30 September 2002. Further details: CORNEA 2002 organiser at the Corneo Plastic Unit, The Queen Victoria Hospital, Holtye Road, East Grinstead, West Sussex, RH19 3DZ (tel: 01342 410 210 ext 560; fax: 01342 317 181; email: cornea2002@hotmail.com).

**CORRECTION**

An error occurred in the article: Bilateral circumscribed haemangioma of the choroid not associated with systemic vascular syndrome. *Br J Ophthalmol* 2001;85:1260. The authors should have been listed as P Perri, C Incorvaia, C Costagliola, P Parmeggiani, G Lamberti, B Paduano, A Sebastiani.