Solitary CD30+ anaplastic large cell lymphoma of the eyelid showing regression

CD30+ anaplastic large cell lymphoma (ALCL) belongs to the group of T cell non-Hodgkin’s lymphomas. The primary cutaneous variant of ALCL usually presents as a solitary, cutaneous, or subcutaneous reddish violet lesion, which can be superficially ulcerated. We present the case of a solitary CD30+ ALCL of the eyelid showing regression.

Case report

A 39 year old man presented with a 4 week history of a progressive painless ulcerating nodule on the right upper eyelid, unresponsive to oral flucloxacillin. He was systemically well and denied recent foreign travel or contact with animals.

A 17 mm diameter ulcer with rolled margins and serosanguinous exudate was evident over the right upper eyelid (fig 1). His cornea, conjunctiva, and anterior chamber were normal. Systemic examination was unremarkable.

Investigations including full blood count, urea and electrolytes, bone and liver profile, immunoglobulins and electrophoresis, autoantibody screening, and Treponema antibody were normal or negative. Tissue culture failed to demonstrate a bacterial, viral, or fungal pathogen. There was no clinical, radiological, or bone marrow evidence of extracutaneous disease.

Histology of the biopsy taken from the lid ulcer margin showed epidermal necrosis associated with ulceration (fig 2). The ulcer base showed haemorrhagic granulation tissue infiltrated by a mixture of lymphocytes, plasma cells, neutrophils, and eosinophils. There were also ill defined groups of large blast cells showing enlarged and pleomorphic nuclei and high mitotic activity. The immuno-histochemical staining showed these cells to be of T cell lymphoid lineage. Many of the large blast cells were CD30 positive but negative for ALK-1 protein. The features were of a CD30 positive anaplastic large cell lymphoma (ALK negative).

Treatment options such as surgery and radiotherapy were discussed with the patient but as the lesion remained stable over a 10 day period, a conservative approach was agreed. A moderately potent topical corticosteroid (mometasone furoate 0.1% cream) was applied to the lesion once daily. When followed up 8 weeks later the ulcer was completely healed without scarring (fig 3).

Ocular ALCL represents a group of large cell lymphomas. They consist of a proliferation of predominantly large lymphoid cells with strong expression of the cytokine receptor CD30 (>75%). Using molecular and clinical criteria, three entities have been identified: primary systemic anaplastic lymphoma kinase (ALK) + ALCL, primary systemic ALK – ALCL, and primary cutaneous ALCL.¹

Primary cutaneous ALCL arise de novo in the skin, commonly on the head and neck of older patients with a median age of 60 years and a male/female ratio of 3:2. Most patients present with solitary, asymptomatic nodules, which can be superficially ulcerated. Primary cutaneous ALCL has a more favourable prognosis than systemic ALCL, with an 5 year survival of approximately 90%. Partial or complete spontaneous regression can be observed in up to 25% of patients with primary cutaneous ALCL, accounting for the previous designation of “regressing atypical histiocytosis.”² Treatment of localised lesions usually includes excision with or without radiation. However, patients with disseminated skin disease may benefit from systemic polychemotherapy.³ In our patient the lesion had resolved within 3 months of initial appearance. The application of a moderate potent topical steroid might have contributed to the regression of the ulcer.

We present a case of a primary cutaneous ALCL of the eyelid showing regression. Ophthalmologists should be aware of this sometimes self regressing entity and an expectant policy might be indicated in non-progressing tumours, thus avoiding potentially mutilating surgery or radiotherapy.

References

Choroidal translocation with a pedicle following excision of a type 1 choroidal neovascular membrane

Excision of type 1 choroidal neovascular membranes (CNVM) in age related macular degeneration (AMD) have a poor visual outcome because of loss of retinal pigment epithelium (RPE). Simple replacement of the RPE may not work because the relation with Bruch's membrane and choroidal complex is disturbed. Creating a free graft of these three layers detaches the choroid from its blood supply. Hence, restoration of these three layers to the subfoveal position while maintaining a connection to the adjacent choroidal blood supply is desirable.

Case report

A 74 year old woman presented with a 3 month history of a left central scotoma and visual acuity (VA) of counting fingers (CF). Clinical examination and fluorescein fundus angiography (FFA) confirmed a type 1 subfoveal CNVM. The fellow eye was 20/30 with scattered soft drusen. Plans para vitrectomy (PPV) and excision of the CNVM were performed as described previously. Atrophic choroidal vessels underlying the CNVM were not removed. A retinotomy was formed temporal to the fovea and vertical scissors inserted into the subretinal space. The RPE, Bruch's membrane, and choroid were incised en bloc in the area temporal to the site of the CNVM to create a graft on a pedicle. The graft was manipulated to a subfoveal position. The pedicle and graft were equally sized to maximally exploit the rich choroidal vasculature and maintain continuity to the choroidal circulation. We were unable to predetermine the position of choroidal vessels as indocyanine green angiography (ICG) was unavailable to us at the time of surgery. Surprisingly, little bleeding occurred and was easily controlled by increasing the infusion height. The patient required two subsequent operations for a rhegmatogenous retinal detachment with grade B proliferative vitreoretinopathy. The retina was flattened after inferior retinectomy and silicone oil insertion.

At review 4 years following initial surgery her vision was CF with a central scotoma on Goldman field testing. The area of translocated RPE, Bruch's membrane, and choroid was visible beneath the fovea with bare sclera demarcating its original site (fig 1A). At 4 years following surgery there was no recurrence of the CNVM on FFA (fig 1B) and ICG angiography demonstrated that the graft and pedicle were vascularised (fig 2).

Comment

Excision of type 1 CNVMs has a poor prognosis because of loss of RPE and atrophy of the choroid. Restoration of the normal anatomical relation between the retinal receptors and the underlying structures is essential for visual recovery. Retinal translocation with strabismic surgery for the movement of the retina to healthy RPE is prolonged and hazardous. Transplantation of homologous RPE cells alone to a subfoveal position has met with varied success. Aylward et al reported no visual improvement after transplantation of an autologous free graft, with fibrosis of the grafts at 10 months, perhaps because of loss of blood supply. Late rich and redundant blood supply of the choroid allowed some freedom in the choice of graft harvest site. As proof of principle we have demonstrated that a choroidal/RPE graft with a pedicle is a feasible surgical technique, resulting in a sustained and vascularised graft. This technique is simpler than time consuming retinal translocation and does therefore merit further investigation.

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References


Intravitreal triamcinolone acetonide and central serous chorioretinopathy

Intravitreal injections of triamcinolone acetonide have increasingly been performed as treatment for intraocular diseases with intraretinal oedema and with subfoveal fluid accumulation, such as diffuse diabetic macular oedema, persistent pseudophakic cystoid macular oedema, central retinal vein occlusion, and exudative age related macular degeneration. In view of the widening spectrum of indications for intravitreal triamcinolone acetonide injections, it was the purpose of this study to evaluate whether intravitreal triamcinolone acetonide injections may be useful as treatment of long-standing central serous chorioretinopathy.

Case report

A 50 year old patient presented with a decrease in visual acuity to 1/20 in his right eye because of longstanding central serous chorioretinopathy. Six years earlier, visual acuity had started to deteriorate, and had remained at 1/20 for the past 2 years. Fluorescein angiograms showed a mottled appearance of the retinal pigment epithelium close to the foveola, and a leakage of dye in the late phase of the angiogram. There was
was stopped. Consent after the injection, all topical and informed about the experimental character of detachment of the fovea.

5 months after the intravitreal injection of 20–25 mg of triamcinolone acetonide, which unchanged. Under topical anaesthesia, the retina was detached. Despite intensive topical optical coherence tomography, the central marked changes in the macula (fig 2). Correspondingly, visual acuity remained at 1/20. Intraocular pressure increased up to levels of 30 mm Hg and was reduced to the normal values by topical application of a carbonic anhydride inhibitor. Thirteen months after the injection, the fovea was still slightly detached. Visual acuity remained at 1/20.

The clinical course suggests that in this eye with longstanding central serous chorioretinopathy an intravitreal injection of a high dosages of triamcinolone acetonide was not accompanied by a fast resolution of the subfoveal fluid and an increase in visual acuity. For more than 5 months after the injection, the fovea remained clearly detached. The partial resorption of the subfoveal fluid 13 months after the injection may not have necessarily been caused by intravitreal triamcinolone but may be explained by the natural course of the disease. The report agrees with other investigations in which patients with central serous chorioretinopathy did not markedly benefit from systemic steroid treatment. This single case report, therefore, does not favour the use of intravitreal triamcinolone acetonide for this treatment.

J B Jonas, B A Kampf...
A regression of the inflammation in the right eye was noted as well as a “cicatrisation” of the choroidal lesions, which appeared as multiple geographical areas of atrophy of both the retina and pigmented epithelium between areas of normal retina. Our suspected diagnosis was a SC.

After 3 weeks, the patient developed the same lesions in the left eye with an occlusion of the superior temporal vein (fig 2). At that time the patient was on prednisone 40 mg/day and anti-TB treatment. A complete clinical examination was done again, but still all results were within the normal limits. The same treatment was introduced (methylprednisolone, intravenous aciclovir).

As the relapse occurred under steroid therapy (prednisone 40 mg/day), the administration of an immunosuppressive drug was discussed. The patient was given mycopenolate mofetil (Gelcept, 2 g/day) and oral prednisone for 1 year, at tapering doses. Anti-TB treatment was continued too. No secondary effects were noted.

We followed the patient during 12 months; his visual acuity returned to 20/20, without a correction, in both eyes. The anterior segment was normal. The posterior segment examination disclosed permanent geographic choroidal atrophic lesions along the vessels in both eyes, confirming the diagnosis of SC.

Comment
SC induces a progressive loss of the retinal pigment epithelium and choriocapillaris. The cause of this disorder is still under investigation but some studies suggest that an inflammatory or a vascular factor are involved in the pathogenesis.1,2 Histopathological studies have shown the presence of extensive lymphocyte choroidal infiltrates but in eyes chronically affected,4 the distribution of the lesions and their angiographic features (fluorescein and indocyanine) may suggest a choroidal occlusion.3 Genetic studies demonstrated an increased frequency of HLA-B7 in this affection,5 our patient was positive. SC affects not only white people but also oriental and blacks people. There is no sex predilection and the patients are middle aged when the diagnosis is made. The patient described in this report was 30 year old which is uncharacteristic. The aetiology remains unknown although recently tubercular serpiginous-like choroiditis has been reported.6

Clinically, inflammatory signs may be noted both in segments (anterior uveitis, vitritis) along with the classic whitish choroidal lesions in the acute stage. The most frequent ocular complication of SC is subretinal neovascularisation which affects 13% to 20% of the eyes. Retinal vasculitis is also observed.

Gupta et al reported a case of SC with a branch vein occlusion in the acute phase.6 Our patient developed a vein occlusion while under treatment (steroids, aciclovir, and anti-TB therapy) which is rare and was never reported to our knowledge. Haemorrhages are sometimes observed in inflammatory diseases (Behcet’s syndrome, sarcoidosis) or in infectious posterior uveitis (syphils, viral infections, toxoplasmosis).

This case demonstrates that SC can affect young patients and that HLA-B7 can be found in Indian patients with SC. The clinical features (vascular occlusion in this case) and the development of new lesions while under treatment let us suspect that the cause of this disease is still not clear.

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Persistent acanthamoeba keratitis in a non-contact lens wearer following exposure to bird seed dust
Acanthamoeba keratitis is a serious and vision threatening disease. It is commonly associated with contact lens wear (up to 93%).1 Early diagnosis and treatment are essential to improve the visual outcome.2 Devastating ocular damage can be attributed to various factors such as misdiagnosis,3 incorrect treatment, excessive topical steroid before diagnosis,4 and resistance.5

Acanthamoeba keratitis in non-contact lens wearers is rare and poses a diagnostic challenge. We present a case of acantha-moeba keratitis in a non-contact lens wearer following accidental exposure to bird seed dust. The strain of acanthamoeba obtained from this patient appeared to show in vivo and in vitro resistance to poly(hexamethylen biguanide), PHMB and chlorhexidine after a good clinical response initially.

Case report
A 57 year male patient presented with pain, blurring of vision, and photophobia of his left eye. Two weeks before the presentation he had an accidental exposure to bird seed dust (brand name Trill, manufactured by Master Foods, Hungary) for his budgies while cleaning the seed pot. It was a seemingly trivial injury not likely to have caused a breach of the epithelium. Examination revealed a visual acuity of 6/6 of the right eye and 6/18 for the left eye. The left eye showed multiple punctate epidermal erosions with epithelial and stromal infiltrates. There was no retained debris at the time of presentation. Initially he was treated as a case of viral keratitis with topical aciclovir and steroid. Although there was an early improvement, the keratitis relapsed after 2 weeks. At that stage a typical ring infiltrate suggestive of acanthamoeba keratitis developed and epithelial culture grew Acanthamoeba polyphaga. He was started on intensive treatment with PHMB, Brolene, and neomycin. His symptoms improved and his visual acuity recovered to 6/9 over a period of 3 weeks. Topical steroids were then added. The antimicrobial treatment was given for 2–3 months and withdrawn gradually over next 4–6 weeks after complete resolution. But following complete cessation of all drops he developed a recurrence with positive cultures. We restarted the intensive treatment with PHMB B and chlorhexidine but the clinical response was poor. Pain was severe with intense limbal inflammation and signs of scarring.

A corneal biopsy was performed which showed persistence of infection. Resistance to PHMB (minimum inhibitory concentration (MIC), 3.125 μg/ml) and chlorhexidine (MIC, 6.25 μg/ml) was demonstrated in the culture obtained from the biopsy. The strain showed in vitro sensitivity to propamidine (MIC, 3.9 μg/ml). A change of treatment to topical propamidine isethionate 0.1% (Brolene) and neomycin led to a rapid response with a decrease in symptoms. Six months after initial diagnosis he is comfortable on maintenance treatment with propamidine isethionate 0.1% and neomycin, but unfortunately has developed a dense central corneal scar (fig 1) and vision of hand movements.

Comment
Acanthamoeba keratitis not related to contact lens wear has been reported before and risk factors include trauma, dirty water splash, and exposure to leaf juice.7 Exposure to bird seed dust has to our knowledge not been reported previously as a known risk factor. Unfortunately, an attempt to culture acanthamoeba from the actual bird seeds and tray was unsuccessful. The second
uncommon feature in our case is the demonstration of in vitro resistance of this strain of acanthamoeba to two of the modern first line acanthamoeba drugs (PHMB and chlorhexidine) while showing a good sensitivity to propamidine.

This is contrary to what has been reported by other authors.1–4 We are unable to say whether resistance developed during treatment or was pre-existent, as sensitivity profiles of the earlier isolates were not obtained. This patient’s initial good clinical response was achieved with a combination of PHMB and propamidine with the latter tapered early during the course of treatment, indicating at least partial in vivo sensitivity to PHMB in the earlier stages. A poor association between in vivo and in vitro resistance has been described for biguanides,5 but this case shows that in vitro MIC can be useful information in the management of persistent acanthamoeba keratitis.

Other authors have stressed the need for long term treatment6,7 and this case also underscores the importance of prolonged effective antimicrobial treatment in order to prevent recurrences.

Figure 1 Photograph showing diffuse central corneal haziness. This photograph was taken 5 months after the initial visit. The area of biopsy is visible inferonasally.

Figure 2 Case 1. (A) Corneal oedema left eye with Descemet’s folds 10 hours after the last dose of topical brinzolamide 1%. (B) Corneal oedema completely resolved 7 days after discontinuing brinzolamide 1% with minimal guttae noted.

References

Brinzolamide induced reversible corneal decompensation

Topical carbonic anhydrase inhibitors (CAIs) such as brinzolamide 1% (Azopt; Alcon Laboratories, Fort Worth, TX, USA) attenuate bicarbonate efflux, and this may lead to corneal oedema.

To our knowledge, this is the first report of complete resolution of corneal oedema after cessation of topical brinzolamide 1%.

Case reports

A 57 year old African-American man with primary open angle glaucoma (POAG) presented with painless blurry vision left eye 1 hour after instilling brinzolamide 1% in both eyes. He had been on brinzolamide 1% both eyes twice daily, brimonidine tarrate 0.2% (Alphagan) in both eyes twice daily, and latanoprost 0.005% (Xalatan) in both eyes once at night for 2 years. On presentation, best corrected visual acuity (BCVA) was 20/25 right eye and 20/50 left eye. The left eye had mild corneal oedema. Descemet’s folds, and whitish fleck-like debris on the corneal endothelium (fig 1A). Intraocular pressures (IOPs) were 15 mm Hg and 16 mm Hg. The brinzolamide 1% in both eyes was discontinued. Timolol maleate 0.5% (Timoptic) in both eyes twice daily and topical prednisolone acetate 1% (Pred Forte) left eye four times daily were started. By 1 week follow up, the cornea was clear (fig 1B). Specular microscopy revealed endothelial cell counts (ECC) of 1355 cells/mm² right eye and 648 cells/mm² left eye with enlarged pleomorphic endothelial cells left eye (fig 2). Central corneal thickness (CCT) was measured as 512 μm right eye and 505 μm left eye.

A 77 year old white man, who had had cataract extraction 46 years earlier and subsequent aphakia right eye, had been followed for open angle glaucoma in both eyes for 25 years. He was on timolol maleate 0.5% in both eyes twice daily, latanoprost 0.005% in both eyes once at night, and pilocarpine hydrochloride 4% gel (Pilopine Gel HS) in both eyes once at night. His visual acuities were hand movement right eye and counting fingers at 1 foot left eye. An IOP of 19 mm Hg right eye and 10 mm Hg left eye left eye necessitated the addition of brinzolamide 1% twice daily right eye. Both corneas were clear at that time. Fifteen months after starting brinzolamide 1%, there was moderate corneal oedema right eye. Brinzolamide 1% was discontinued. Over 3 months, the corneal oedema in the right eye gradually resolved. The patient later needed trabeculectomy with mitomycin C right eye because of medically uncontrolled IOP.

Comment

The Merck Worldwide Adverse Experience (ECC) of 1355 cells/mm² right eye and 648 cells/mm² with pleomorphic cells left eye.

Topical CAIs can cause corneal oedema in compromised corneas—for example, those corneal effect from a similar medication should indeed be potentially reversible.1 Dorzolamide’s peak concentration in rabbit corneas is also reached 1 hour after dosing, and the half life in the cornea is approximately 2 hours.3 This is consistent with case 1 having blurry vision 1 hour after instilling brinzolamide 1%.

Patients with primary open angle glaucoma or ocular hypertension and a baseline ECC of greater than 1500 cells/mm² have an average 3.6% decrease in ECC after a year of dorzolamide 2% three times daily.7 This endothelial loss is much higher than the 0.6% annual rate seen in normal subjects.7 Topical CAIs can cause corneal oedema in compromised corneas—for example, those
with cornea guttata, but the oedema is reversible if identified early. Because initiating brinzolamide 1%, an ECC may be needed for high risk patients (that is, monocular, previous ocular surgery, corneal disease, etc.). Because of the potentially irreversible nature of the corneal decompensation, topical CAIs may be relatively contraindicated in patients with significant corneal disease.

Both dorzolamide and brinzolamide are both topical CAIs, their chemical formulas are different, and a side effect that is associated with dorzolamide may not necessarily be assumed to be associated with brinzolamide. It is important to be aware that brinzolamide can also potentially cause corneal oedema.

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An unusual cause of acquired horizontal diplopia in a young adult
Cysticercosis is caused by infection from the larval form of Taenia solium, is endemic to regions with poor sanitation. Human cysticercosis more commonly affects the central nervous system, with less common involvement of ocular tissues. Myocysticercosis is a subset of orbital cysticercosis and is considered a rare entity.

Case report
A 19 year old Nepalese housewife presented with left sided headache that had been present for 1 year. She had been treated for migraine headache in another hospital. Visual loss revealed orthoptia in primary gaze with Snellen acuity of 6/6 bilaterally. Both the Humphrey visual fields and the colour vision testing were normal in both eyes. There was no relative afferent pupillary defects detected. Ocular motility testing revealed left abduction deficit with the resulting horizontal diplopia. Her fundi examination were both normal and there were no optic disc swelling.

Magnetic resonance image (MRI) re-scan revealed no prop- tosis. Her fundi examination were both normal and there were no optic disc swelling.

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Use of scanning laser ophthalmoscopy in visual conversion reaction

Visual conversion reaction is a psychosomatic anomaly that manifests as reduced visual acuity (VA) and visual field defects. Scanning laser ophthalmoscopy (SLO) can detect a scotoma and VA under direct fundus observation. However, there have been no reports of patients diagnosed with visual conversion reaction using SLO. We report a patient with visual conversion reaction using SLO.

Case report

A 20 year old woman presented with a sudden bilateral loss of vision. She reported being under severe stress at work. The best corrected visual acuity (BCVA) was counting fingers in both eyes. The external eye examination and pupillary responses were normal in both eyes. Conventional ophthalmoscopy, funduscopy, and fluorescein angiography were unremarkable. The visual fields were constricted to within 5° of fixation using Goldmann perimetry (fig 1A). The results of magnetic resonance imaging, computer tomography of the brain and orbits, visual evoked potentials, and electroretinography were unremarkable. A general medical examination showed no abnormalities. There were no scotomas (based on Goldmann size III stimulus on the retina), and the stability of fixation was central and stable in both eyes using SLO microperimetry (fig 1B). The VA using SLO was 20/20 in both eyes. We diagnosed visual conversion reaction in this case.

Comment

This is the first report of a patient with visual conversion reaction using SLO. In this case, the BCVA was counting fingers in both eyes at the first visit. However, the VA using SLO was 20/200 both eyes and better than the conventional examination. The visual fields were constricted in both eyes to within 5° of fixation using Goldmann perimetry. However, there were no scotomas in either eye using SLO microperimetry. The distinction between the VA and visual fields between the conventional and SLO examinations was demonstrated over the 10 month follow up period. Ten months after the initial examination, the VA and visual fields were normal in both eyes by both conventional and SLO examinations. There was no distinction between them during the recovery period. Van de Velde reported that SLO results were comparable with those obtained during a conventional examination in normal subjects. The distinction between the VA and visual field between the conventional and SLO examinations may help in the diagnosis of patients with visual conversion reaction. Future clinical studies of several cases of visual conversion reaction using SLO are needed.

References


Figure 1 (A) The visual fields at the first visit using Goldmann perimetry (right eye, left; left eye, right). (B) The visual field at the first visit using SLO microperimetry (right eye, left; left eye, right).

Figure 2 (A) The visual fields using Goldmann perimetry 10 months after the first visit (right eye, left; left eye, right). (B) The visual fields using SLO microperimetry 10 months after the first visit (right eye, left; left eye, right).

Ten months later, the BCVA was 20/20 both eyes. The visual fields in both eyes using Goldmann perimetry were normal (fig 2A). There were no scotomas, and the stability of fixation was central and stable in both eyes using SLO microperimetry (fig 2B). The VA using SLO was 20/20 in both eyes. We diagnosed visual conversion reaction in this case.

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The severe acute respiratory syndrome coronavirus in tears

We welcome the article by Loon et al.1 Earlier, we published our finding of the SARS coronavirus in specimens collected by the novel technique of conjunctiva upper respiratory tract irrigation (CURTI), but not in paired nose and throat swabs, very early in the course of the disease.2 In designing CURTI, we considered safety to medical personnel to be the first priority, and also, finding a method that samples all three portals of entry for upper respiratory viral pathogens—the eyes, nose, and mouth. Loon et al’s findings complement our study by showing that SARS coronavirus can indeed be found in tears.

While we agree with their conclusion that the ability to isolate the virus early in the course is important, we do not think that the eyes are important organs that propagate the virus, other than to ophthalmologists and to unwary close contacts. For instance, the eyes cannot generate infectious aerosol. Rather, we feel that the eyes are important portals of entry and have not been given sufficient attention—witness medical personnel in full personal protection gear and N95 masks but without watertight goggles, and sometimes without splashguards.

We also think that employing the services of ophthalmologists for the purpose of collecting tear specimens for the diagnosis of SARS would fail to achieve in most medical environments. On the other hand, our method of CURTI is entirely self-help, deployable in quarantine locations, and avoids unnecessary contact between an infectious source and susceptible individuals.

The finding of SARS CoV in tears raises several additional questions:

1. How does the virus end up in the tear? Was it the result of direct inoculation at the time of infection into permissive conjunctival epithelial cells, either by hand or aerosol, or was it the result of secretion from a lacrimal gland infected haematogenously? The lacrimal glands are not very different anatomically from the salivary glands. Yet saliva has been shown to be a poor specimen for the laboratory diagnosis of SARS.3

2. Was there any evidence of conjunctivitis, lacrimitis, or evidence of infection of the globe or nasolacrimal sac?

3. Is there any means or advantage in sampling the nasolacrimal sac, to which the tear drains, and could the nasolacrimal duct system be itself a hiding place for the SARS coronavirus during the incubation period?

References


Comments on using fibrin glue in pterygium surgery

I read with great interest the article by Koranyi and coworkers, who evaluated a new technique for pterygium surgery using a fibrin tissue adhesive (Tissel Duo Quick).2 In their randomised trial the authors concluded that using the glue instead of sutures caused less postoperative pain and shortened the surgical time. Nevertheless, the timing of the randomisation is not clearly stated in their report.

Whether or not the surgeon knew the patient’s group (sutures or fibrin glue) at the time of pterygium removal and conjunctival graft harvesting may have influenced the extent of the removal and the size of the graft. Therefore, the differences in postoperative pain and/or recurrence could be related to that initial factor. However, the authors refer to the final step, as the authors suggest. Ideally, the surgeon should be informed if the conjunctival graft should be sutured or glued after harvesting it.

Additionally, in their discussion the authors did not mention the risk of infection when using fibrin glue. Some viruses, such as parvovirus B19 (HPV B19) are particularly difficult to remove or inactivate, and human infection has been reported after the use of fibrin glue.3 In thoracic surgery, epidemiological evidence suggests that more than 20% of uninfected people were subsequently infected with HPV B19 by use of fibrin during the procedure.2 Prions are also of concern. The direct application of any of the apparently effective methods of prion decontamination to plasma products is inappropriate because they are harsh and denaturing.4 Although the risks for both diseases are minimal, the authors should have addressed this issue in their discussion.

Authors’ response

We thank Dr Alvarenga for his comment on our article about using fibrin glue for pterygium surgery.

We write in the “Material and methods” section that only the thickened and keratized portion of the conjunctiva was excised and the graft was prepared to have the same size as the nasal conjunctival defect, regardless of which group the patient was randomised to. Thus, the knowledge of the surgery method did not influence the results or conclusions of the study. It is also impossible to randomise the patients after surgery to the surgeon’s knowledge, because the grafts were handled somewhat differently in the different methods described.

The fibrin adhesives in the references mentioned by Alvarenga are clearly not Tissel Duo Quick (Baxter). In the article by Hino et al., the specific trade name of the fibrin sealant is not given, but the text states that the sealant in question is treated by dry heat to inactivate viruses—Tissel is vapour heated, so it cannot be Tissel. In the article by Kawamura et al. the product is Beriplast (Aventis Behring). Nevertheless, we will pay attention to this matter in the future. We have not been able to find any report of HPV B19 infection when fibrin glue was used in minor surgery. To date we have used the fibrin glue in more than 700 eye procedures since 1999. Any clinical infection with HPV B19 has not yet been detected in our patients.

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Korean kindergarten vision screen programme

Lim et al report a large scale effort to mix home plus health centre acuity screening in preschool children.1 We are very encouraged by the work of Lim et al, particularly concerning the frequency of ocular symptoms in the Korean preschooler, the number of children who were not dismissed from specified follow up (presumed amblyopia risk), and the inclusion of a simple, home administered test which over 97% of children were able to pass. It is of high merit that parents were carefully instructed to place tissue and tape over the non-tested eye.
though this does not preclude peeking if the parent is not paying particular attention. Positive answers to the parental questionnaire were not very specific for eye disease and therefore could greatly increase societal cost if used as a screening method. We have a few points of clarification for these authors: How was the home acuity test initially validated? The number of children who passed their home exam have gold standard confirmatory exams from which false negative and true negative rates could be estimated? The positive predictive value estimates utilise gold standard exam criteria that need further definition and/or standardisation. (1) It is not clear whether amblyopia was diagnosed at multiple eye clinics and by general or paediatric ophthalmologists, it is not clear what criteria are used to define amblyopia, and the criteria to be included as a “significant” cycloplegic refractive error vastly overestimates risk factors compared to a recently published attempt to standardise reporting of vision screening research.

We would urge the authors to perform additional calculations on the breakdown of gold standard exam “significant” refractive errors and better define how amblyopia was diagnosed.

**References**


**IVF and retinoblastoma**

I read with great interest the letter published in the BJO by Lee et al.1 It reports on the first child born after in vitro fertilisation (IVF) and harbouring a unilateral retinoblastoma in the United States. However, it should be noted that this reported child is the eighth documented child (not the sixth as mentioned by the authors). The first child ever observed was reported by our group in 2001. He had a bilateral retinoblastoma.2 In 2002, a second child with bilateral disease was documented in the Netherlands.3 In 2003, five additional cases were reported from the Netherlands (two with bilateral disease and three with unilateral disease). In this paper, an estimated relative risk of 4.9 to 7.2 for an IVF born child

in the Netherlands to develop retinoblastoma was surmised.4 The issue of the possible association of assisted reproductive techniques (ART) with an increased risk of retinoblastoma has raised great concern worldwide. The interest of this association is highlighted by the fact that the expression of retinoblastoma in childhood is influenced by epigenetics—a regulatory mechanism not involving DNA sequence which could be affected by the various ART techniques.

In recent years, tens of thousands of children were born after ART. However, not one single case of retinoblastoma was observed until 2001. The possible reasons for this phenomenon were discussed.5 Awareness regarding the occurrence of retinoblastoma in ART born children sparked by our original observation of the first case in 2001 has probably been a trigger for the unveiling of additional cases. Therefore, more cases are to be expected in the near future.

Whether the increased number of observed cases indicates that ART born babies have a higher risk of developing retinoblastoma remains to be carefully investigated. None the less, a thorough prospective assessment of the possible association between ART and retinoblastoma is mandatory. Ongoing multicentre and multinational control studies will hopefully provide the needed answers to this “thorny” but most crucial aspect of ART. Till then, accurate accounting of previous observations is, of course, a key factor for a better insight into these issues.

**CORRECTIONS**

In the paper titled Prospective case control study on genetic association of apolipoprotein e2 with intraocular pressure (Br J Ophthalmol 2004;88:581-2), the authors have been listed incorrectly. The correct listing is A Juzenma, S Bleich, U Reulbach, K Henkel, N Wakili, G Beck, B Rautenstrauss, C Mardin, G Oh Naumann, A Reis, J Kornhuber. The journal apologises for this error.

In the letter titled Bilateral decompression retinopathy after orbital decompression surgery (Br J Ophthalmol 2004;88:1605-6), the authors have been listed incorrectly. The correct listing is G J Ben Simon, R A Goldberg, J D McCann. The journal apologises for this error.

**Notices**

**Worldwide clinical trials for new technique for early detection of eye disease**

A unique new non-invasive technique for high resolution optical imaging of the eye is receiving global acclaim. By combining two high-resolution imaging technologies, the new technique provides doctors with 3-D images of the retina, macula and the optic nerve.

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