Figure 2 Colour fundus photo of the right eye at presentation (A) and after 4 weeks of isoniazid treatment (B). The black arrow indicates an arteriovenous crossing and likely site of occlusion. The two white arrows mark the nasal border of the serous retinal detachment.

tap for analysis of tuberculosis, and a presumptive diagnosis of bartonella retinitis was made. As several cases of bartonella infections have been reported to resolve without treatment, it was decided to observe the patient while also ruling out a hypercoagulable aetiology.² An extensive hypercoagulability examination was non-contributory.

Over 8 weeks of observation, the subretinal fluid and vein occlusion worsened clinically, and visual acuity fluctuated between 20/70 and 20/400 OD. Repeated *Bartonella* titres 6 weeks after the original titres remained at 1:128 IgG, IgM negative. At this point, the patient again deferred anterior chamber paracentesis. Given the failure of the fundus findings to resolve, or *Bartonella* titres to increase, empirical isoniazid treatment was started. A medicine consultation deferred four-drug treatment on the basis of a positive PPD being the only direct evidence of tuberculosis. At 4 weeks after beginning isoniazid, visual acuity improved to 20/30, with marked resolution of the serous detachment. A medicine consultation was again sought but four-drug treatment was deferred on similar grounds. After 10 weeks on isoniazid treatment, visual acuity was 20/25, with no subretinal fluid on optical coherence tomography. Repeat fluorescein angiogram (fig 1) was negative for any vasculitic process. Colour fundus photos (fig 2) showed subtle lines at the level of the retinal pigment epithelium outside the superior arcades, indicating the furthest extent of the subretinal fluid.

Comment

Ocular tuberculosis is regarded as a rare manifestation of tuberculosis infections, historically occurring in only 1.46% of patients with systemic disease.³ Vein occlusions have been described previously with ocular tuberculosis; however, in the absence of any evidence of vasculitis, vitritis or uveitis, this is a rare presentation.⁴ Mechanistically, the vein occlusion could have been the result of compression from a retinal or choroidal tubercle. The presence of retinal pigment epithelial changes argues for a deep retinal or choroidal process. Although a focal area of vasculitis obscured by retinal exudation could also be responsible, the lack of any vitreous cell argues against this.

In the absence of an anterior chamber or vitreous sample for culture or polymerase chain reaction analysis, only a presumptive diagnosis can be made. However, an extensive investigation ruled out any other infectious, systemic (including diabetes and hypertension), or

hypercoagulable aetiology. Furthermore, marked clinical improvement was achieved rapidly after the onset of anti-tuberculosis treatment. The failure of the patient's IgG *Bartonella* titres to increase, or IgM positivity to develop, despite two separate tests separated by 6 weeks, greatly lessens the likelihood of this aetiology.¹ In summary, we report an unusual presentation of presumed ocular tuberculosis in the setting of a branch retinal vein occlusion without associated vasculitis or evidence of intraocular inflammation. This case highlights the need for the pursuit of a systemic aetiology of vein occlusions in otherwise healthy, young individuals.

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Use of anterior segment optical coherence tomography in a penetrating eye injury

The Anterior Segment Optical Coherence Tomography (ASOCT) is a new imaging modality that allows cross-section imaging of the anterior segment at high resolution. This can be performed even in presence of corneal opacification.¹ We report a case of penetrating corneal injury in a professional falconer. Following primary repair, the anterior segment was assessed post-operatively using ASOCT, which revealed the extent of anterior segment injury despite the presence of corneal oedema.

Case report

A 23-year-old falconer presented with a penetrating injury to his left eye sustained while flying a red kite (*Milvus milvus*). The bird's "talon" had caught the eye on landing. Examination showed hand-movements vision, a 3.5 mm nasal corneo-limbal perforation with iris prolapse, hyphaema and clear lens. The intraocular pressure (IOP) was 12 mm Hg.

Surgery was performed immediately. In addition to the pre-operative findings, vitreous prolapse was noted. Surgery involved abscission of iris, anterior vitrectomy and corneal suturing with 10-0 nylon; intravitreal vancomycin, amikacin and amphotericin were injected. Post-operatively he received topical ofloxacin 0.3% and dexamethasone 0.1% for a month. At 3 weeks follow-up, left eye examination showed vision of 6/9 uncorrected, intact corneal wound with surrounding corneal oedema, quiet looking anterior chamber, distorted pupil and IOP of 25 mm Hg (fig 1A,B). Gonioscopic view was obscured due to corneal oedema. Fundal examination was normal. Anterior segment Optical Coherence Tomography (Visante™ OCT, Carl Zeiss, Dublin, California, USA) was performed, which demonstrated posterior corneal wound malposition, partial loss of iris tissue and

Figure 1 (A) Slit-lamp photograph of left eye showing repaired corneal perforation and nasal iris defect. Assessment of anterior chamber angle and zonular damage at wound site is difficult due to corneal oedema. White dashed line shows the plane of OCT section. (B) Slit-lamp retroillumination photograph of left eye showing difficult anterior segment assessment.

Figure 2 (A) Anterior segment OCT image (at low resolution; coronal section across the dashed line in Figure 1) of left eye showing posterior corneal wound (CW), nasal iris tissue defect with peripheral anterior synechiae (PS) and abnormal angle (A). (B) Anterior segment OCT image at high resolution showing posterior corneal wound edge (CW), abnormal angle (A), zonular dehiscence (ZD), lens subluxation (LS) and vitreous in pupillary area (V).

peripheral anterior synechiae in the adjacent angle (fig 2A). In addition, ASOCT showed zonular dehiscence with vitreous prolapse and mild lens subluxation localised to the injury site (fig 2B). At 7-weeks follow-up (dexamethasone stopped 3 weeks earlier), his uncorrected vision improved to 6/6 with an IOP of 40 mmHg. Oral acetazolamide and topical antiglaucoma medications were prescribed to lower IOP. Residual vitreous in anterior chamber was now visible clinically using conventional methods.

Comment

ASOCT is a high-resolution, non-contact method of anterior segment imaging that uses 1310-nm light.¹ It shows the anatomical relationships of cornea, iridocorneal angle and other anterior segment structures in real time.² It enables measurement of anterior segment structures such as corneal thickness, anterior chamber depth and anterior chamber angle. In addition to preoperative and post-operative assessment, ASOCT can be used intraoperatively which can help in the intraoperative diagnosis and subsequent surgical decision making.³

In this case, ASOCT showed angle damage, zonular dehiscence and residual vitreous prolapse, and suggested residual vitreous in anterior chamber as the cause of raised IOP. Due to the presence of corneal oedema, these findings could not be determined during the early postoperative period using conventional examination methods such as slit-lamp examination and gonioscopy. Although high-frequency ultrasound biomicroscopy could have revealed such clinical information, it would be unsuitable in a case with perforating eye injury, due to the immersion method required.

Anterior segment OCT is a novel imaging modality, which is valuable in the evaluation of eye injuries and especially useful in the management of these cases where a non-invasive method of anterior segment assessment is required.

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Figure 1 Fundus photograph of the right eye showing exudative retinal detachment involving the macula.

eye revealed severe submacular lipid exudation with an inferior exudative retinal detachment involving the macula. The left eye had a waxy disc, some midperipheral subretinal flecks, a characteristic macular sheen and no visible subretinal exudation. Fundus fluorescein angiogram showed massive capillary dropout and mid-peripheral neovascularisation in the right eye. The left eye only showed hyperfluorescence from subretinal flecks. There was no angiographic evidence of retinal ischemia in this eye. Based on this, a clinical diagnosis of exudative retinopathy in the right eye was made. Management was difficult in view of the poor visual acuity in the right eye with such a dramatic exudative retinal detachment. After discussing treatment options with the parents, they opted for retinal cryotherapy to prevent the development of a painful, rubeotic eye.

The patient was the second child of first cousin parents. She had a cousin and two siblings similarly affected. They had a similar phenotype with cardiac failure in infancy, early deafness, retinal dystrophy and diabetes mellitus. Molecular analysis confirmed linkage to the Alström locus on chromosome 2p13. Mutation analysis identified a 1444 nucleotide deletion (IVS8 + 895 del 1444) encompassing the whole of exon 9 of the *ALMS1* gene. This is a novel mutation for Alström syndrome, identified in this single family and has recently been reported by our institution.⁵

Comment

Alström syndrome is a rare autosomal recessive disorder caused by mutations in the novel gene *ALMS1*² which has been shown to be widely expressed and localised to centrosomes and the base of cilia.^{3,4}

A review of ocular features in Alström Syndrome reported poor visual acuity in the order of 20/200 or worse by 10 years.⁶ Unlike other retinal dystrophies, this condition can result in vision as poor as perception of light by the end of the second decade. However exudative retinopathy has not been documented in any of the above 22 cases in this series.

Nonetheless, exudative retinopathy has been demonstrated uncommonly in longstanding retinitis pigmentosa. Mutations in the crumbs homologue 1 (*CRB1*) gene are strongly associated with the development of Coats-like exudative vasculopathy in these patients.⁷ This entity differs from classic Coats disease in terms of relatively higher incidence of bilateral involvement in exudative retinopathy, equal gender incidence, mode of inheritance and location of retinal pathology.¹ We are reporting a unique mutation associated with

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Exudative retinopathy in a girl with alström syndrome due to a novel mutation

Coats disease is an uncommon telangiectatic retinal exudation that is most often seen in males. Similar changes are an uncommon but well known complication of retinitis pigmentosa, occurring in up to 3.6% of the affected individuals.¹ This association has not been reported with Alström syndrome. Alström syndrome is a rare autosomal recessive disorder caused by mutations in a novel gene of unknown function, *ALMS1*,² widely expressed in centrosomes and the base of cilia.^{3,4} We report an Asian girl with bilateral congenital cone-rod dystrophy due to Alström Syndrome who developed subretinal exudation resembling Coats disease.

Case report

A twelve year old girl of ethnic Pakistani origin presented with marked deterioration of vision in her right eye over the previous year. She had attended our institution with cone-rod retinal dystrophy (ERG demonstrated absent photopic response & markedly reduced rod response), infantile-onset obesity, cardiomyopathy, sensorineural deafness, diabetes mellitus with hyperinsulinaemia, hepatosplenomegaly and recurrent urinary tract infections for several years. There was no history of polydactyly. On this basis, a clinical diagnosis of Alström syndrome was made.

On examination, there was no demonstrable vision in the right eye and only bare perception of light in the left. Slit-lamp examination revealed quiet anterior chambers without evidence of rubeosis in either eye. A right exotropia on penlight examination with manifest horizontal pendular nystagmus was noted. Fundal examination (fig 1) of the right