The ocular surface toxicity of paraquat

We describe the clinical appearance and progress of bilateral ocular chemical injury caused by paraquat, a herbicide. Paraquat is used more commonly in developing countries and it has been associated with severe and prolonged ocular surface abnormalities due to the nature of the chemical. The current concepts in managing such an injury are reviewed.

Case report

A 69 year old fruit farmer splashed a 20% solution of paraquat into both his eyes. The exposure of the right eye to the solution was more marked. The eyes were irrigated for less than 5 minutes at the time of the injury, and a formal irrigation, using Ringer’s solution for each eye, was not performed until presentation 5 hours later.

General medical history revealed that apart from controlled hypertension he was otherwise well and he had no previous history of ocular problems.

On arrival uncorrected visual acuity was 6/9 on the right and 6/12 on the left. The intraocular pressure was 14 mm Hg on the right and 19 mm Hg on the left. There was a circular 90% epithelial defect centred over the right cornea. The remaining corneal epithelium was opaque, leathery and oedematous (Fig 1). The left cornea was epithelialised although the surface was irregular and hazy in appearance (Fig 2). The conjunctiva was hyperaemic and chemotic, particularly on the right. The limbal vasculature initially appeared congested, but there was no evidence of episcleral ischaemia. The corneal stroma was clear. Examination did not reveal any abnormality of the anterior chamber, lens, or posterior pole in either eye.

The patient was commenced on our chemical burns protocol. This comprises topical citrate 10% drops every 2 hours, aspirate 10% drops every 2 hours, chloramphenicol ointment four times daily, atropine 1% drops three times a day, fluorometholone acetate 1% drops every 2 hours, oral aspirate 500 mg four times daily, and 4 g of a urinary alkaliniser and the steroid was changed to non-preserved dexamethasone 1% hourly by day.

At 6 days post-injury there was evidence of epithelial healing from the margin of the epithelial defect in the inferior fornix of the right eye. Topical citrate and ascorbate were stopped and treatment was altered to non-preserved topical medication only: topical dexamethasone 1% every 2 hours, chloramphenicol four times daily, and hyromellose every 2 hours. Autologous serum tears (diluted to 20% in sterile saline solution) were also commenced 2 hours.

Two weeks after the injury there were persistent epithelial defects in both eyes but the healing edge of conjunctival epithelium had reached the nasal limbus in the right eye. Best corrected visual acuity was 6/24 right and 6/6 left. At this stage there was no pseudomembrane present although the conjunctiva remained inflamed.

By 4 weeks after injury the ocular surface in both eyes had re-epithelialised. The cornea of the right eye at this stage was clear with no vascularisation. Visual acuity remained 6/24 on the right.

Impression cytology of both central corneas was undertaken at this point. The right cornea showed conjunctival-type epithelium with scattered goblet cells, polymorphonuclear leucocytes, and numerous apoptotic bodies. The left cornea demonstrated a corneal phenotype epithelium.

The last review was at 6 months post-injury. Uncorrected visual acuity was 6/9 in both eyes. The conjunctiva of both eyes remained minimally inflamed with mild erythema, chemosis, and subepithelial fibrosis now evident. These changes were more marked in the right eye (Fig 3). There was superficial pannus encroaching onto the right cornea predominantly superonasally. The affected area in the left eye shows subconjunctival fibrosis with vascular disorganisation and localised sceral translucency (Fig 4). Current treatment was dexamethasone 1% twice daily in both eyes.

Comment

Paraquat is a dipyridylidium quaternary ammonium salt that acts as an indiscriminate killer of all plant life. Preparations made commercially commonly contain the related compound diquat as well as surfactants to increase distribution over the leaf and aid penetration. Toxicity in humans is thought to relate to paraquat recycling in the redox reaction (Fig 5). This depletes NADPH and interrupts cell metabolism. The reduced
Paraquat ingestion can result in early fatal-ity due to multorgan failure. In less severe cases acute renal and hepatic toxicity is com-mon. Pulmonary fibrosis may result in death. Pulsed methylprednisolone and cyclophosph-amine during the inflammatory stages may have a profound effect on reducing the mortality. It is unclear whether a similar regimen would circumvent the long term sequelae of ocular paraquat injury.

The patient in this case had significant exposure to a commercial preparation with minimal first aid measures being implement-ed at the time of injury. He developed a severe surface injury over the first 48 hours with marked epithelial loss over one eye. This implied there were few, if any, viable limbal stem cells remaining. A severe pseudomembranous conjunctivitis then developed.

Previous reports of this extensive type of surface injury show that eventually conjunctival-isation of the cornea with vascular pannus is to be expected. Severe injury may result in a disordered ocular surface with dryness, sym-blepharon, ankylöblepharon, fornical short-ening, entropion, and trichiasis.

Patients with total limbal stem cell loss invariably develop superficial pannus and conjunctivalisation of the cornea. Evidence that previously conjunctival phenotypic epi-thelium can transdifferentiate into corneal type epithelium is derived from animal experiments with a debrided cornea and lim-bus where corneal epithelium healed the resultant defect. Viable limbal stem cells may thus have remained in the perilimbal crypts of Vogt. The right eye of this patient healed from conjunctival epithelium, confirmed by im-pression cytology, although most of the cornea remains clear without pannus. It has been reported that transdifferentiation is inhibited once neovascularisation occurs.1 In the left eye, which only had a small zone of limbal stem cell loss if any, the epithelium over the cornea was confirmed as corneal phenotype on impression cytology and there was no vascularisation.

A suitable microenvironment for healing should be encouraged through the use of medical therapy. Topical corticosteroids in the first week after injury promote successful healing of epithelial defects by controlling inflammation. Adequate lubrication is also important. Autologous serum tears have been reported to relieve the symptoms of dry eye1 and improve the ocular surface disease more successfully than conventional tear substiti-utes in dry eye states. These drops have also been instrumental in the healing of persistent epithelial defects.2 Essential components of the tear film present in serum tears include epidermal growth factor, vitamin A, and transforming growth factor β, which are important for the proliferation, differentia-tion, and maturation of the surface epithelium.3 We present a case of severe paraquat chemical injury to both eyes that had a good outcome from treatment. This case demonstr-ates a very good result from a usually dev-asating injury.

Preventing conjunctivalisation of the cor-neal surface after total loss of the limbal stem cell population remains a challenge. Thera-peutic intervention may help restore a more functional surface visually. The use of inten-sive early antioxidant therapy followed by autologous serum tears and non-preserved ocular lubricants may have improved the out-come.

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References

Henchon-Schönlein purpura with bilateral central retinal artery occlusion

Henoch-Schönlein (H–S) purpura is an acute leucocytoclastic vasculitis that primarily affects children and mainly involves skin, joints, gastrointestinal tract, and kidney.1 Reported ophthalmic manifestations of Henoch-Schönlein purpura include episcleritis, scleritis, keratitis, anterior uveitis, and central reti-nal vein occlusion.2 3 However, central retinal artery occlusion, to the best of our knowledge, have not been reported. We report on a girl with H–S purpura complicated with bilateral central retinal artery occlusion.

Case report
A 6 year old girl visited our paediatic depart-ment with the chief complaint of multiple erythematous rashes over the lower extremiti-ess and buttock for 2 weeks. Under a presumptive diagnosis of H–S purpura, oral prednisolone was prescribed. Nevertheless, arthralgia, haematuria, and moderate hyper-tension developed 3 weeks later. The histopathological findings of renal biopsy were compatible with H–S purpura nephritis. Un-fortunately, acute renal failure occurred de-spite aggressive systemic treatment and haemodialysis was started.

Two days before haemodialysis, the patient noticed sudden visual loss. Visual acuity was hand movement in both eyes. Anterior seg-ment and intraocular pressure were normal. Fundus examination revealed a cherry red spot with severe retinal oedema at the macular and peripapillary area in both eyes. Disc oedema and venous engorgement were also noted in both eyes (Figs 1 and 2). The retinal manifestations were compatible with bilateral central retinal artery occlusion. Fundus fluo-rescein angiography was not performed be-cause of her poor general condition.

Three days after haemodialysis, her sys-temic condition deteriorated with drowsiness that was proved to be cerebral vasculitis by brain computed tomography.

Figure 1 Fundus photograph of the right eye demonstrating cherry red spot with severe retinal oedema at macular and peripapillary area, disc oedema, and venous engorgement.

Figure 2 Fundus photograph of the left eye demonstrating cherry red spot with severe retinal oedema at macular and peripapillary area, disc oedema, and venous engorgement.
One month later, her visual acuity was counting finger in both eyes. Fundus examination revealed a pale disc, arterial sheathing, and drusen-like RPE change at foveal area in both eyes. Six months later, her best corrected visual acuity was 6/30 in the right eye, and 6/60 in the left.

**Comment**

The predominant clinical manifestations of H-S purpura are cutaneous purpura (100%), abdominal pain (63%), gastrointestinal bleeding (33%), and nephritis (40%)." In general, H-S purpura is an acute, self-limiting illness though one third of patients will have one or more recurrences of symptoms. H-S purpura was the cause of renal failure in 2% and 5% of groups of children undergoing haemodialysis in California and France, respectively. Although the aetiology of H-S purpura remains unknown, it is clear that IgA has a critical role in the immunopathogenesis. The clinical features of H-S purpura are a consequence of widespread leukocytoclastic vasculitis due to IgA deposition in vessel walls. Treatment is limited to symptomatic and supportive care. Corticosteroids are often used depending on the severity of the disease.

According to the previous reports, the ocular manifestations of H-S purpura are rare, including episcleritis, scleritis, keratitis, anterior uveitis, and central retinal vein occlusion. In this case, H-S purpura vasculitis may have an important role in the pathogenesis of bilateral central retinal artery occlusion. To our knowledge this might be the first case of H-S purpura complicated with bilateral central retinal artery occlusion in the literature.

**Case 1**

A 15 year old boy presented with occasional watering in the left eye for 4 years. There was no history of any visual disturbance.

On examination his left eye was apparently normal and Bertel exophthalmometry readings were 19 mm in the left eye and 13 mm in the right eye) with bar reading of 105. He had a ptosis of 2 mm (Fig 1, top right) with good levator function. The appearance of the left upper lid was suggestive of plexiform neurofibromatosis and there were no systemic features of diagnostic significance.

On fundus examination there was an oblique insertion of the disc, with inferonasal crescent and an oval cup involving 0.8:1 part of the disc (Fig 1, bottom right). A B-scan ultrasound of orbit confirmed that the apparent ptosis of the left eye was due to a longer axial length (24.36 mm compared with 22.60 mm in the right eye). There were two cafe au lait spots on the trunk but there was no other systemic abnormality. None of the family members had similar ocular findings or any evidence of neurofibromatosis.

The IOP responded poorly to topical medication and a trabeculectomy was performed which controlled the IOP.

**Case 2**

A 5½ year old girl presented with gradual enlargement of the right eye since the age of 6 months. There was a history of mild photophobia but none of lacrimation or deterioration of vision.

On examination the patient's Snellen acuity was 20/240 in the right eye (−1.50 sph, −4.00 ast, ×105°).

The left eye had extensive hyperplasia of the iris pigment epithelium which overlay the stroma of the iris in an irregular manner but did not reach the angle (Fig 1, top left). The normal iris architecture was lost, but no nodules were seen. There was pigment on the anterior surface of the lens but the iris did not transilluminate. There was anisocoria of 1.5 mm, the left pupil being larger but round, regular and reacting to light. Indirect gonioscopy (Fig 1, bottom left) demonstrated an open angle with iridotrabeculodysgenesis in the form of an anterior insertion of the iris into the trabecular meshwork with excessive pigment deposition and a prominent Schwalbe's line, this being very marked in some areas. The right eye had a fully open angle with all structures clearly seen. Intraocular pressure (IOP) on the first examination was 52 mm Hg in the left eye and 12 mm Hg in the right. On fundus examination there was an oblique insertion of the disc, with inferonasal crescent and an oval cup involving 0.8:1 part of the disc (Fig 1, bottom right).

In the right eye there was no evidence of neurofibromatosis and reviews the available literature on this rare abnormality. In one case the presentation of glaucoma was in adolescence and in infancy in the other. Associated ocular features are described. In these two patients there were no systemic features of diagnostic significance.

In this case, H-S purpura vasculitis was due to a longer axial length (24.36 mm compared with 22.60 mm in the right eye), there were two cafe au lait spots on the trunk but there was no other systemic abnormality. None of the family members had similar ocular findings or any evidence of neurofibromatosis.

The IOP responded poorly to topical medication and a trabeculectomy was performed which controlled the IOP.
In the normal iris the pigment epithelium forms a double layer that includes itself into the pigment granules as the pigmentation. A congenital exaggeration of this process occurs where the pigmented layer advances over the anterior surface of the iris to a varying degree, so that the pupillary circumference is involved in PIPH is variable, but characteristically ends short of the angle. The pupil may be normal in size in some cases and larger than its counterpart in others. It may be round and reactive to light but may be oval and less reactive to light. The iris stroma is generally hypoplastic to a variable degree, with a smooth anterior surface with absence of circular concentric folds and radial folds. The condition is usually unilateral but a bilateral case has been reported.

Both the cases in this report had extensive PIPH, loss of normal iris architecture, and pupillary abnormalities. The aetiology of the condition remains unknown but is probably due to imbalance of growth between the ectoderm and the mesoderm, usually a hyperplasia of the latter. It may represent a clinical manifestation of a pathological process related to abnormal neural crest cell migration.

Histological studies have shown that the ectodermal layer is the result of hyperplasia over the pigment epithelial layer. There is no evidence that the condition is hereditary in uncomplexed cases, however, those which are associated with conditions such as Rieger's dysgenesis is not uncommon or occurred in the same way as the major deformity. A variety of systemic disorders have been reported in association with PIPH. Neurofibromatosis is the disorder most commonly associated with PIPH. In one report three of out eight were associated with neurofibromatosis. In addition, Ritch et al reported neurofibromatosis, a case of associated diabetes mellitus and left facial hemiatrophy, and one each of Prader-Willi syndrome and right facial hemiatrophy. Hetzberg reported a case with associated asthma and a dental anomaly. Not all cases have systemic associations. None of the nine patients reported by Dowling et al had any systemic associations. None of the two cases reported in that report had any identifiable systemic association.

Besides the iris and pupillary abnormalities discussed above, PIPH is frequently associated with significant ocular abnormalities. Spinal- ceral mild ptosis with good levator function was presented in six of nine cases reported by Dowling et al and in one each by Ritch et al and Herzberg. The eye may be slightly proptotic in some cases. The appearance of the upper lid may suggest plexiform neuroma even though no mass may be palpable. All these features were present in the first case in this report. Angle anomalies appear to be universal in most of the cases demonstrating anterior insertion of the iris, a prominent Schwalbe's line, prominent iris processes and/or increased pigment insertion of the iris, are usually present in these cases. In this report, cases with late onset also has been reported. The intraocular pressure shows an initial decrease when the pressure is instituted but rises again quickly and filtering surgery was required in most cases. Surgical intervention was necessary in both the cases in this report. The features of PIPH detailed in this report are in concordance with those previously published. There may be other associated ocular abnormalities, ptosis being the commonest. Neurofibromatosis is the most frequently associated systemic disorder. Glaucoma may develop at any stage, therefore these cases should be recognised early by healthcare professionals and followed for development of glaucoma.

References
2 Dieflin TS, Jacob PC, Kneigelstein GK. Primary congenital ectropion uvæ associated with vitreoretinal degeneration. Ophthalmologica 1998;212 63–5.

Unilateral vitreous haemorrhage secondary to caudal epidural injection: a variant of Terson's syndrome

Terson’s syndrome is characterised by vitreous haemorrhage accompanying subarachnoid or any other form of intracranial haemorrhage. Although Litten reported vitreous haemorrhage in association with intracranial haemorrhage in 1881, it was Terson who described the syndrome in 1900. Haemorrhage in Terson’s syndrome may be contained between the internal limiting membrane and retina or extend into the vitreous cavity. Other findings include multiple periretinal, intraretinal, subretinal haemorrhages, as well as macular rings and epiretinal membranes.

Case report
A 47 year old man with chronic back pain due to epidural adhesions (confirmed in magnetic resonance imaging (MRI)) was referred to the anaesthetic department for caudal epidural injection of steroids and saline to break up the epidural adhesions for pain relief. He received the injection through the sacral hiatus between the spinal dura and spinal canal in the caudal epidural space under general anaesthesia. Following recovery from general anaesthesia, he stated that he had lost the sight in his left eye. Ophthalmic examination at that time confirmed vision of 6/60 left eye with no afferent pupillary defect, normal anterior segments, and the presence of a dense left vitreous haemorrhage with a normal right eye. There were no other predisposing factors for vitreous haemorrhage. Post-epidural MRI scan of brain and spinal cord was performed. There was no intracranial haemorrhage but the epidural adhesions were still noted to be present. The patient’s vision gradually improved to 6/12 over a 3 year period but because of residual severe symptomatic floats he underwent left pars plana vitrectomy, which allowed his vision to improve to 6/6 unaided.

Comment
Terson proposed that intraocular haemorrhage occurred secondary to intracranial hypertension, which resulted in elevated peri- papillary capillary pressure. The most commonly cited mechanism is that elevated intracranial pressure results in subarachnoid haemorrhage transmitted within the optic nerve sheath and obstructs intracranial venous drainage causing distension and rupture of peripapillary and retinal capillaries resulting in significant haemorrhage in the subhyaloid space or vitreous cavity.
In our patient during the caudal epidural injection the fluid wave of injection did not spread uniformly in the epidural space and break the adhesions but, instead, got trapped in tiny compartments formed by the adhesions and caused lateral pressure on the spinal cord, thus increasing subarachnoid space pressure, resulting in vitreous haemorrhage in the left eye (Fig 1).

Therefore, we feel the cases with pathogenesis of intraocular haemorrhage, secondary to a sudden rise of intracranial pressure, should be considered as the variant of Terson’s syndrome.

The clinical course of Terson’s syndrome is typically one of gradual resolution of the vitreous haemorrhage and return of normal vision. Haemorrhages that do not resolve may require vitrectomy, but several studies have proposed that early vitrectomy also may improve visual outcome.

Indications for early pars plana vitrectomy include vitreous haemorrhage in infants (prevent amblyopia) and in adults with bilateral vitreous haemorrhage. Indications for conservative management include rapidly clearing vitreous haemorrhage, mild haemorrhage, unilateral cases with normal vision in the fellow eye, and pre-existing ocular damage precluding a favourable visual outcome.

**References**


**Severe macular pucker after retinal detachment surgery in an infant**

The appearance and clinical course of rhegmatogenous retinal detachments in children are more complicated than those of adults, because the retinal detachments are generally associated with trauma or congenital anomalies. In an infant, especially, an unexpected course may develop; however, the incidence of these detachments is so low that treatment and complications have scarcely been described in the literature. We report an infant who developed severe macular pucker after retinal detachment surgery.

**Case report**

A 2-week-old male infant was referred with a diagnosis of bilateral congenital cataracts. The pregnancy and delivery had been uncompli- cated. His mother also had had congenital cataracts. Both eyes of the infant had dense zonular cataracts. The patient had no associ- ated ocular problems on slit lamp biomicroscopy or gonioscopy and no abnormalities on physical examination. He underwent bilateral lensectomy and anterior vitrectomy by limbal approach at 3 weeks of age; both fundi were unremarkable. Although his right eye under- went sufficient anterior vitrectomy, retropillary membrane developed on the residual vitreous surface and the pupil closed 3 months postoperatively. During the second surgery, pupilloplasty was performed and we found a tear in the ciliary epithelium of the pars plana resulting from traction of the retropillary membrane tissue. We also discovered a localised detachment of the retina and ciliary epithelium (Fig 1A). We performed cryopexy with encircling scleral buckle, vitrecomy, and fluid-air exchange, and the retina was reattached. One month later, macular pucker rapidly developed and enfolded the entire posterior retina (Fig 1B). The original tear was completely sealed, and signs of the recurrent rhegmatogenous retinal detachment with proliferative vitreoretinopathy, including vitreous haze and wrinkling of retina in the other location, were not identified. After additional vitrectomy to remove residual cortex and to perform membrane peeling, the retina was finally reattached with a residue of degeneration near the fovea (Fig 1C).

Hematoxylin and eosin and Masson trichrome stains and transmission electron microscopy of the surgically removed membrane showed pig- mented cells, fibroblast-like cells, vessels, and collagen fibres (Fig 2A). The membrane was immunostained with antibodies for cytokera- tin and α-smooth muscle actin (Fig 2B, C), while there was slight immunoreactivity for von Willebrand factor (Fig 2D).

**Comment**

Macular pucker in this infant is significantly different from that in adults. The severe posterior retinal folds may be formed by strong contraction of the epiretinal membrane, a firm vitreoretinal juncture, and an extensible retina. A previous histopathological report on recurrent proliferation after vitrectomy in two children with tractional retinal detachments showed that retinal glial cells were the main reactive cells. However, in our case, the major component of the pucker was retinal pigment epithelium (RPE) cells, the same as in adults with rhegmatogenous retinal detachment. Cytoplasmic actin within the membrane, rather residual vitreous cortex on the retinal surface, may be involved in forming strong contractile elements, which also may underlie idiopathic cases in children. Vessels in the pucker may have originated from the adherent retina, because no residual hyaloid vessels were identified during surgery. Because excessive cryopexy tends to disperse and activate RPE cells, minimal cryopexy is recommended; laser may be preferable.
to treat rhegmatogenous retinal detachment in infants. Enzyme assisted vitrectomy to separate the firm vitreoretinal juncture may prevent the development of subsequent macular puckering. Frequent examinations and careful management are required even after successful reattachment surgery.

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References

Microbial keratitis associated with extended wear of silicone hydrogel contact lenses

Traditional hydrogel soft contact lenses (SCL) have limited oxygen permeability. Recently introduced silicone hydrogel SCL have much higher oxygen transmissibility (Dk/t O₂), allowing near normal oxygen supply to the cornea during extended lid closure, and are hoped by some to address most of the problems related to corneal hypoxia encountered with previous extended wear soft contact lenses. They have therefore been approved for up to 30 days of continuous wear in both Europe and Australia.

Four cases of microbial keratitis in patients who were using silicone hydrogel SCL (either CibaVision Focus Night and Day lenses (Lotrafollicon A, fluorosiloxane hydrogel) or Bausch & Lomb PureVision lenses (Balaficlon A, silicone hydrogel)) on an extended wear basis are presented. The minimum amount of continuous wear was 24 hours. All cases were treated either in private or at the corneal clinic of the Royal Victorian Eye and Ear Hospital from December 2000 to February 2001. All the patients underwent a complete ophthalmic examination by a corneal specialist. Microbiological specimens were taken from all patients via cornea scrapings and were submitted for Gram and Blankophor staining, and bacterial and fungal cultures via direct inoculation onto sheep blood agar. Bacterial sensitivities of cultured organisms were also obtained. Where possible, the contact lenses themselves were also sent for microbial cultures.

Each case is described in brief, and a summary presented in Table 1.

Case 1

This 22 year old man presented with a 2 day history of left ocular injection, pain, photophobia, and blurred vision. He was wearing CibaVision Focus Night and Day SCL continuously for 10 days at a time, discarding the lenses after a month of use. He had swum in a river in these lenses 1 week earlier, after which he removed the lenses and disinfected them with “Renu” multipurpose solution (boric acid, edetate disodium, poloxamine, sodium borate, sodium chloride, and polyaminopropyl biguanide, manufactured and distributed by Bausch & Lomb, Greenvile, SC, USA). Continuous wear was recommenced within a few hours.

Examination revealed an uncorrected visual acuity of 3/60 in both eyes, improving to 6/12 in both eyes with pinhole. A paracentral 3 mm epithelial defect with underlying dense infiltrate was noted in the left eye with anterior chamber inflammation of 1+ cells and multiple scattered KP. 1+.

Corneal scrapings revealed fungal elements on Gram and Blankophor staining. Cultures grew Acinetobacter species in the enrichment broth, sensitive to ciprofloxacin, chloramphenicol, and tobramycin. Penicillium was later grown on the Sabouraud agar slope. A yeast (not Candida albicans) was grown on Sabouraud agar from the left contact lens, with Penicillium species grown from the right.

Topical ciprofloxacin 0.3% was commenced hourly, after which his symptoms and signs markedly improved. The ciprofloxacin was tapered and changed to topical chloramphenicol 0.5% (Chlorisig, Sigma) 8 days after presentation.

Two weeks later, the epithelial defect had resolved but significant subepithelial scarring remained. His best corrected visual acuity was 6/6 right eye and 6/7.5 left eye.

Case 3

A 21 year old man was referred to MSL with a 2 day history of right eye injection, pain, photophobia, and decreased vision. He was wearing PureVision lenses on a daily wear basis, but changed to continuous wear 24 hours before the onset of his symptoms.

Four cases of microbial keratitis in patients who were using silicone hydrogel SCL (either CibaVision Focus Night and Day lenses (Lotrafollicon A, fluorosiloxane hydrogel) or Bausch & Lomb PureVision lenses (Balaficlon A, silicone hydrogel)) on an extended wear basis are presented. The minimum amount of continuous wear was 24 hours. All cases were treated either in private or at the corneal clinic of the Royal Victorian Eye and Ear Hospital from December 2000 to February 2001. All the patients underwent a complete ophthalmic examination by a corneal specialist. Microbiological specimens were taken from all patients via cornea scrapings and were submitted for Gram and Blankophor staining, and bacterial and fungal cultures via direct inoculation onto sheep blood agar. Bacterial sensitivities of cultured organisms were also obtained. Where possible, the contact lenses themselves were also sent for microbial cultures.

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Examination revealed a visual acuity of 6/6 right eye and 6/5 left eye with his spectacle correction. Marked right eye ciliary injection and anterior chamber activity were noted with cells, flare, multiple scattered KPs, and a small paracentral epithelial defect with underlying infiltrate.

Corneal scrapings revealed polymorphs on Gram stain (no organisms seen), and a heavy growth of *Corynebacterium* species on the sheep blood agar plate, sensitive to penicillin, ciprofloxacin, and chloramphenicol. Culture of the contact lenses was impossible as they had been discarded.

Treatment consisted of hourly topical ciprofloxacin 0.3%, topical fluoromethalone acetate 0.1% (Flarex, Alcon, Fort Worth, TX, USA) was added four times daily after clinical improvement. No organisms on Gram stain, but grew *α* haemolytic streptococcus from the enrichment broth. Conjunctival injection was noted in the left eye, with a 3 × 4 mm paracentral area of stromal haze and an associated area of subepithelial infiltrate. The overlying epithelium was intact.

Corneal scrapings revealed no polymorphs or organisms on Gram stain, but grew an *Acinetobacter* (enrichment broth) from the corneal scrape and Blankophor stain from the corneal scrape and *Penicillium* (sheep blood agar plate) not available. Treatment was with hourly topical ciprofloxacin 0.3%, with tapering after 48 hours. Review 1 week later revealed a persisting subepithelial scar and a best corrected spectacle acuity of 6/7.5.

**Comment**

Extended wear of soft contact lenses for up to 6 days has been advocated in various forms since the 1980s with traditional hydrogel lenses. However, owing to the relatively high rates of microbial keratitis, extended wear of soft contact lenses has not had widespread use.

The advent of high oxygen permeability silicone hydrogel soft contact lenses has again made extended wear a viable option, as the increased oxygen permeability is thought to reduce the risk of development of a hypoxic epithelial defect, which can serve as a portal of entry. Pre-release extended wear studies did not reveal any cases of microbial keratitis but these studies were relatively small. Lenses with a Dk/t O₂ greater than 50 × 10⁻⁶ have also been shown to have a lesser affinity for *P. aeruginosa* binding during extended wear, further decreasing the risk of microbial keratitis.

Our experience suggests that extended wear with even these newer SCL is still a risk factor in the development of microbial keratitis. All four patients had central or paracentral infiltrates, with three patients presenting with an associated epithelial defect. All four patients also had a positive culture or Gram/Blankophor stain from the corneal scrape and had residual scarring after resolution of the acute episode. Although *Corynebacterium* species are considered by some to be a non-pathogenic organism, it has been described as the causative organism in several cases of microbial keratitis. "We therefore feel that it is very unlikely that any of these cases represent a more benign non-infectious contact lens complication such as CLPU (contact lens induced peripheral ulcer), CLARE (contact lens induced acute red eye), or 1K (infiltrative keratitis), which are all described as being conditions that resolve after cessation of contact lens wear alone, without the development of residual corneal scarring."

Previous studies have shown that the most important risk factor for the development of microbial keratitis in soft contact lens wearers is the duration of contact lens wear, where overnight wear in particular aggravates the relatively hypoxic of the cornea. However, there are other risk factors such as hypercapnia, trauma, biofilm alterations/contamination, altered corneal sensation, altered tear volume, and composition. Only hypoxia and hypercapnia should be improved by increased contact lens gas permeability.

Three of the four patients described had swum in their lenses within weeks of their presentation. This might be an important risk factor in the development of their microbial keratitis in association with their silicone hydrogel SCL (as it is with other SCL), although the organisms involved were not those typically associated with microbial keratitis from contaminated water exposure. All four of the patients were also males between the ages of 16 and 22 years. These two demographic factors have been linked to an increased risk of microbial keratitis in contact lens wearers.

Recent studies have shown that bacterial populations grown from silicone hydrogel SCL in asymptomatic wear were not statistically different in comparison with those grown from standard HEMA based SCL. This
suggests that a silicone hydrogel SCL can still be a means of contamination in the pathogenesis of SCL microbial keratitis. Certainly, some of the lenses in this small series did grow the same organisms as the corneal lesions themselves.

Our experience supports a multifactorial causality for the development of microbial keratitis in extended SCL wearers, rather than just corneal epithelial hypoxia, particularly in high risk groups such as the four patients described where high risk behaviour is also undertaken. Further investigation needs to be done on the effects these lenses have in extended wear with regard to the development of microbial keratitis before their long term safety can be assured.

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References
2 Fatt I. Comparative study of some physiologically important properties of six brands of disposable hydrogel contact lenses. CLAO J 1997;23:49–54.

Serious corneoscleral complications after pterygium excision with mitomycin C

The use of topical mitomycin C (MMC) to prevent recurrence after pterygium surgery is increasing since its introduction by Kunimoto and Mori in Japan, and its subsequent popularisation in the United States by Singh and associates.1 Low dose MMC (0.02%) twice daily for 5 days after the operation has been prospectively studied with long term follow up, and few serious side effects have been noted.2,3 Intraoperative MMC appeared to be an effective and safe adjunctive treatment of primary pterygium excision.4

Case reports

We retrospectively analysed three patients who presented at the Haemek Medical Central, Afula, Israel with scleral melting which developed after pterygium excision between October and November 2000 with intraoperative application of MMC (0.02% for 3 minutes). The MMC was washed out immediately with an abundant amount of balanced salt solution for at least 3 minutes. During the past 10 years we performed over 300 pterygium excisions with intraoperative use of MMC in Haemek Medical Center with three cases having serious complications.

Case 1 (Fig 1)

A previously healthy 50 year old man underwent recurrent pterygium excision of the right eye using a bare sclera technique with intraoperative application of MMC 0.02% for 3 minutes, having undergone pterygium excision with MMC 10 months earlier in his right eye. Visual acuity was 6/9+ SC. Preoperative opthalmological examination revealed a nasal flashy wide lesion 4.5 mm over the limbus and inferior symblepharon, with the remainder of the examination being normal. He was released on the same day, following surgery at which time there were no complications. On the first (postoperative day 1) and second (postoperative day 7) follow ups no complaints or complications were noted. However, on postoperative day 30, corneal limbal perforation and iris incarceration in the wound was noted. Immediately, he underwent right eye corneal tectonic graft surgery. At his last follow up (12 weeks after the pterygium excision with MMC), the best corrected visual acuity was 6/24 CPH, the graft has good adaptation, no gap and no rejection signs.

Case 2 (Fig 2)

A previously healthy 37 year old man underwent pterygium excision of the left eye using a bare sclera technique with intraoperative application of MMC 0.02% for 3 minutes. His presenting symptoms were cosmetic only. Visual acuity was 6/6 partial SC. Preoperative opthalmological examination revealed nasal flashy wide lesion 2.8 mm over the limbus with the remainder of the examination being normal. Two years previously he had undergone pterygium excision of the right eye using a bare sclera technique with intraoperative application of MMC 0.02% for 3 minutes. The right eye procedure had been successful and was performed in the same institute. Three weeks after the surgery, on routine follow up scleral melting and necrosis were noted. The scleral defect was unresponsive to ocular lubricants, topical antibiotics, topical steroids, and patching. Ten weeks after the left eye pterygium excision and MMC application, he underwent autologous conjunctival graft surgery to repair the defect. Follow up 2 weeks later showed that the graft has good adaptation and re-epithelialisation was noted.

Case 3

A previously healthy 70 year old man underwent pterygium excision of the right eye using a bare sclera technique with intraoperative application of MMC 0.02% for 3 minutes. On preoperative examination visual acuity was 6/24 partial SC in the right eye and 6/60 SC in the left. Anterior segment was quiet except for a nasal temporal pterygium 7 mm over the limbus partially covering the pupil axis in the right eye. The procedure of the right eye pterygium excision with MMC was successful and performed in the same institute. No complaints or complications were noted at the first two follow ups; however, 3 weeks postoperatively right eye temporal scleral melting and ectasia was seen (3 mm x 3 mm in size). Conservative topical treatment with antibiotics, topical anti-inflammatory agents, and steroids failed to resolve the situation. Consequently, 8 weeks after the initial right eye surgery he underwent right eye conjunctival flap grafting over the area of scleral melting. At his last examination best corrected visual acuity was 6/36 partial, blood vessel growth was noted towards the bare sclera and re-epithelialisation with minimal staining. He is still being treated with topical antibiotics with some improvement.

Figure 1

Figure 2
Comment
Mitomycin C is an antineoplastic antibiotic agent isolated from the fermentation filtrate of Streptomyces caespitosus. Its action is similar to alkylating agents; it alkylates and crosslinks DNA and, in addition, may generate crosslinks to alkylating agents; it alkylates and crosslinks DNA and, in addition, may generate crosslinks to alkylating agents. Mitomycin C is an antineoplastic antibiotic agent isolated from the fermentation filtrate of Streptomyces caespitosus. Its action is similar to alkylating agents; it alkylates and crosslinks DNA and, in addition, may generate.

In this series, all patients underwent pterygium excision in the bare sclera technique with the low concentration of 0.02% of MMC and a short application of 3 minutes. Among our patients one had corneal perforation that was treated by tectonic keratoplasty in the macular region with associated subretinal fluid. The lesion was initially treated with argon laser photocoagulation, but within a month the decision was made to re-treat the lesion with TTT. Over the next 7 months, visual acuity deteriorated to 20/200. The lesion exhibited persistent elevation and subretinal fluid. By ultrasound, a change in the retro scleral echogenicity was observed, precipitating referral to an ophthalmic oncologist whereupon a diagnosis of choroidal melanoma with extrascleral extension was made. The patient was then referred to UCSF for consideration of proton beam therapy.

On examination, all abnormal findings were confined to the right eye. The patient’s visual acuity was counting fingers at 2 feet. Funduscopy revealed a raised pigmented tumour centred on the fovea, measuring 7 mm vertically by 10 mm horizontally, extending to within 2.3 mm of the disc. Subretinal fluid was present and extended over the nasal aspect of the tumour (Fig 1A). A flat lesion with associated subretinal fluid.

Table 1

<table>
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<tr>
<th>No</th>
<th>Age/sex/race</th>
<th>Presenting symptom</th>
<th>Clinical finding</th>
<th>Treatment</th>
<th>Preop VA v postop</th>
<th>Outcome</th>
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<tbody>
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<td>1</td>
<td>50/M/white</td>
<td>Blurred vision, dust sensation</td>
<td>Limbal perforation with iris incarceration in the wound</td>
<td>Tectonic corneal graft</td>
<td>6/9+ SC 6/24 CPH</td>
<td>Good graft adaption. No recurrence of pterygium after 12 weeks</td>
</tr>
<tr>
<td>2</td>
<td>37/M/white</td>
<td>Cosmetic disturbance</td>
<td>Scleral nasal melting (3 mm x 2 mm)</td>
<td>Trial at conservative topical treatment failed. Autologous conjunctival graft</td>
<td>6/9+ SC 6/12+ SC</td>
<td>Good graft adaption at last follow up</td>
</tr>
<tr>
<td>3</td>
<td>70/M/white</td>
<td>Pterygium covering the pupil visual axis</td>
<td>Scleral temporal melting (3 mm x 3 mm)</td>
<td>Trial at conservative topical treatment failed. Conjunctival flap</td>
<td>6/24 pt CC 6/36 pt CC</td>
<td>Blood vessels growth towards the bare sclera with re-epithelialisation. No recurrence of pterygium after 12 weeks</td>
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Extrasceral extension of a choroidal melanoma after argon photocoagulation and transpupillary thermotherapy

The optimal management of small posterior choroidal melanomas remains controversial, especially for tumours located near the optic disc and fovea. Although with increasing rarity, argon laser photocoagulation continues to be used in the primary treatment of small tumours, despite data suggesting that other therapeutic methods may be more successful. More recently, transpupillary thermotherapy (TTT) has emerged as a therapeutic option for the primary treatment of small choroidal melanomas. Initial results are promising, but like any new treatments, more widespread use and longer follow up are needed for a thorough assessment of its efficacy. As a cautionary reminder that additional study is required to define the potential complications of these treatments, we present a case of choroidal melanoma in which treatment with primary argon photocoagulation followed by TTT was associated with extrasceral extension of the tumour.

Case report
A 38 year old woman presented with decreased visual acuity in her right eye. An ophthalmologist noted a pigmented choroidal lesion with associated subretinal fluid. The lesion was initially treated with argon laser photocoagulation, but within a month the decision was made to re-treat the lesion with TTT. Over the next 7 months, visual acuity deteriorated to 20/200. The lesion exhibited persistent elevation and subretinal fluid. By ultrasound, a change in the retro scleral echogenicity was observed, precipitating referral to an ophthalmic oncologist whereupon a diagnosis of choroidal melanoma with extrascleral extension was made. The patient was then referred to UCSF for consideration of proton beam therapy.

On examination, all abnormal findings were confined to the right eye. The patient’s visual acuity was counting fingers at 2 feet. Funduscopy revealed a raised pigmented tumour centred on the fovea, measuring 7 mm vertically by 10 mm horizontally, extending to within 2.3 mm of the disc. Subretinal fluid was present and extended over the nasal aspect of the tumour (Fig 1A). A flat lesion with associated subretinal fluid.
photoacoagulation is typically used as an adjunct to other treatments, but in select cases has been used as primary therapy for choroidal melanoma. Typically, photoacoagulation is reserved for small tumours (less than 3-4 mm in thickness and less than 10 mm in diameter) that are close to the fovea and/or the optic disc in eyes with good vision. Because the level of tumour necrosis with laser photoacoagulation is shallow (0.2-0.8 mm), multiple treatments are often necessary. Therefore, the greatest challenge to successful photoacoagulative therapy in choroidal melanomas is determining when the tumour has been fully ablated. TTT shares the advantage that photoacoagulation has over radiotherapy, including the more rapid visible reduction of tumour size, the relative sparing of adjacent normal tissue, and the convenience and economy of an outpatient procedure. In contrast with the shallow penetration of the argon laser, however, TTT employs near infrared light to produce up to a 3.9 mm depth of tumour necrosis. The therapeutic dose of irradiation is used as its exclusive primary treatment, with TTT therapy being used as a local control for small posterior choroidal melanomas with worrying early results. It has been shown that recurrences occur following apparently successful photoacoagulation or TTT because invisible nests of malignant cells can infiltrate the sclera, a histologically documented phenomenon. The presented case reinforces this as a reminder that this possibility is not trivial. Exterals extension, presumably from tumour out of reach of initial argon phototherapy and subsequent TTT, resulted in a requirement for aggressive local surgical therapy in addition to TTT and adjuvant systemic therapy to reduce the risk of metastatic disease. The unusual degree of extrascleral extension for a small melanoma also raises the possibility that either photocoagulation or, more likely, TTT produced some reduction in scleral integrity allowing focal egress of tumour cells. Reports of complications following argon laser therapy are likely to become more frequent as advances in modern radiotherapy have made primary photoacoagulation an uncommon treatment. The use and investigation of TTT, however, continue to increase and the risks for extrascleral extension remain undefined.

**Comment**

Options for the management of choroidal melanoma include observation, laser photoacoagulation, transpupillary thermotherapy, charged particle radiotherapy, brachytherapy, local resection, and enucleation. Argon laser photoacoagulation is typically used as an adjunct to other treatments, but in select cases has been used as primary therapy for choroidal melanoma. Typically, photoacoagulation is reserved for small tumours (less than 3-4 mm in thickness and less than 10 mm in diameter) that are close to the fovea and/or the optic disc in eyes with good vision. Because the level of tumour necrosis with laser photoacoagulation is shallow (0.2-0.8 mm), multiple treatments are often necessary. Therefore, the greatest challenge to successful photoacoagulative therapy in choroidal melanomas is determining when the tumour has been fully ablated. TTT shares the advantage that photoacoagulation has over radiotherapy, including the more rapid visible reduction of tumour size, the relative sparing of adjacent normal tissue, and the convenience and economy of an outpatient procedure. In contrast with the shallow penetration of the argon laser, however, TTT employs near infrared light to produce up to a 3.9 mm depth of tumour necrosis. The therapeutic dose of irradiation is used as its exclusive primary treatment, with TTT therapy being used as a local control for small posterior choroidal melanomas with worrying early results. It has been shown that recurrences occur following apparently successful photoacoagulation or TTT because invisible nests of malignant cells can infiltrate the sclera, a histologically documented phenomenon. The presented case reinforces this as a reminder that this possibility is not trivial. Exterals extension, presumably from tumour out of reach of initial argon phototherapy and subsequent TTT, resulted in a requirement for aggressive local surgical therapy in addition to TTT and adjuvant systemic therapy to reduce the risk of metastatic disease. The unusual degree of extrascleral extension for a small melanoma also raises the possibility that either photocoagulation or, more likely, TTT produced some reduction in scleral integrity allowing focal egress of tumour cells. Reports of complications following argon laser therapy are likely to become more frequent as advances in modern radiotherapy have made primary photoacoagulation an uncommon treatment. The use and investigation of TTT, however, continue to increase and the risks for extrascleral extension remain undefined.

**Case report**

A 69 year old white woman presented 1 day after uneventful right phacoemulsification and intraocular lens implantation with a vision of counting fingers (CF), a relative afferent pupil defect, hypopyon with anterior chamber fibrin, and normal intraocular pressure. We were unable to visualise the fundus although a red reflex was noted. Ultrasound examination showed patchy increased vitreous reflectivity with a flat retina and choroid. Pars plana vitreous tap of 0.2 ml was performed followed by injection of 1 mg/0.1 ml of vancomycin and 0.4 mg/0.1 ml of amikacin. Sterile dilution was conducted with intravitreal aminoglycosides.

References


Macular infarction after intravitreal amikacin: mounting evidence against amikacin

Retinal toxicity attributable to intravitreal use of aminoglycosides for endophthalmitis has been reported. Campochiaro and Conway reported 101 cases of retinal damage due to intravitreal aminoglycosides. Amikacin, an aminoglycoside, is in widespread use in the United Kingdom for the treatment of Gram negative organisms in endophthalmitis. We report a case of macular toxicity following the use of intravitreal amikacin for postoperative endophthalmitis, outlining mechanisms of retinal toxicity, and offer alternatives to amikacin. We believe that enough evidence now exists to support a change in the current Royal College of Ophthalmologists’ endophthalmitis treatment guidelines that are based on the Endophthalmitis Vitrectomy Study.

Figure 2

(A) B-scan ultrasonography demonstrates extrascleral extension of the tumour and exhibits the choroidal excavation, acoustic quiet zone, and orbital shadowing (Fig 2A). A-scan demonstrated spontaneous pulsation, low to medium internal reflectivity, and a sharp posterior spike (Fig 2B). The intraocular thickness was 3.0 mm with 7.7 mm of extrascleral extension. These findings are consistent with choroidal melanoma with posterior extraocular extension. Systematic evaluation revealed no signs of metastasis. The potential for orbital contamination by tumour made focal therapy by proton beam a less desirable alternative. Therefore, enucleation with en bloc resection of the extrascleral tumour was recommended and subsequently performed. Pathological examination confirmed the diagnosis of malignant choroidal melanoma, mixed cell type, with extensive extrascleral extension and focal vascular invasion. The patient elected to undergo adjunct post-surgical external beam irradiation to reduce the rate of orbital recurrence, but with the understanding that this treatment, while not definitively harmful, is of unproved benefit. She was also referred to the medical oncology service for systemic therapy and has begun an experimental treatment protocol using interferon alfa. Systemic chemotherapy is currently under consideration.

**Figure 2** (A) B-scan ultrasonography demonstrates extrascleral extension of the tumour and exhibits the choroidal excavation, acoustic quiet zone, and orbital shadowing characteristic of a uveal melanoma. Arrows surround areas of tumour extension. (B) A-scan ultrasonography shows spontaneous pulsation, low to medium internal reflectivity, and a sharp posterior spike. The measured intraocular tumour thickness was 3.0 mm with 7.7 mm of extrascleral extension.
we support a change in current UK treatment guidelines. Choices will remain controversial until the incidence of toxicity for both amikacin and ceftazidime is determined by a prospective randomised controlled study; however, on the evidence currently available we suggest that ceftazidime should replace amikacin as the first line agent of choice against Gram negative organisms in postoperative endophthalmitis.

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References

Spontaneous involution of retinal and intracranial arteriovenous malformation in Bonnet-Dechaume-Blanc syndrome

Intracranial arteriovenous malformations are capable of spontaneous regression.25 There are also numerous recorded events of vascular remodelling, thrombosis, and autoinvolution in retinal arteriovenous malformations.26 This report documents a self obliterated retinal arteriovenous malformation in a patient with Bonnet-Dechaume-Blanc syndrome who developed neurological symptoms due to spontaneous regression of the intracranial component of the angiomatic malformation.27

Case report
A 32 year old man from Guam was evaluated for a history of right parietal headaches for several months and acquired temporal hemianopia in the left eye. He had a history of blindness in the right eye from early childhood, and had recently become aware of a temporal hemianopia in the left eye. Visual acuity was no light perception in the right eye and 20/20 in the left eye. The right pupil was unresponsive to light. The left pupil was sluggishly reactive and there was a right afferent pupillary defect. Slit lamp examination showed conjunctival venous engorge ment in the right eye. Retinal examination disclosed white, sclerotic major retinal vessels, with no evidence of retinal vascular perfusion in the right eye (Fig 2). The major retinal vessels were surrounded by non-perfused clusters of white, racemose, telangiectatic, vessels (Fig 1). The left optic nerve showed band atrophy with corresponding nerve fibre layer dropout but no other retinal abnormality.

Magnetic resonance imaging showed numerous vascular channels permeating the right basal ganglia, anterior portion of the midbrain, prefrontal gyri, optic chiasm, and the orbit right. The deep hemispheric portion of the lesion showed surrounding oedema. CT scanning showed punctate and conglomerate calcifications in the malformation, as well as enlargement of the right optic canal. Cerebral angiography demonstrated an angiomatous vascular malformation that permeated the basal ganglia as well as the optic chiasm region and extended into the right orbit (Fig 2). There was a relative lack of deep venous drainage in the chiasmal region of the malformation, with diversion to the Sylvian vein system and over the convexities to the sagittal sinus. The lack of hypertrophy in these draining venous channels, together with the

Figure 1 Fundus photograph demonstrating self obliterated retinal arteriovenous malformation. Sclerotic major retinal vessels are surrounded by smaller racemose ghost vessels.

Figure 2 Internal carotid artery angiogram, arterial phase, demonstrating marked enlargement of the ophthalmic artery (bottom right, arrow). The right optic nerve is honeycombed by a vascular malformation (open arrow).

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regional oedema on magnetic resonance imaging, suggested a recent obstruction of vascular flow within the aniomatous malformation.

Comment

The syndrome of unilateral retinocephalic arteriovenous malformation was first described in 1937 by Bonnet et al. Six years later, Wyburn-Mason published his report in the English language. These congenital unilateral retinocephalic arteriovenous malformations may involve the visual pathways from the retina to the optic nerve to the ipsilateral occipital cortex, and may involve the chiasm, hypothalamus, basal ganglia, midbrain, and cerebellum. Since these arteriovenous malformations are high flow systems in which veins are exposed to arterial blood pressures, they are susceptible to turbulent blood flow and to vessel wall damage which can lead to thrombosis and occlusion. Over time, components of an angiomatous malformation can grow, haemorrhage, sclerose, thrombose, and involute.

Our patient had longstanding involution of his retinal arteriovenous malformation, with new neurological symptoms resulting from thrombosis of the intracranial component of the tumour. Spontaneous occlusion of the major venous drainage within the deep cerebral hemisphere and optic chiasm may have caused headaches by producing regional oedema or by diverting flow to other venous structures. Since the major venous drainage within the malformation was already occluded at the time of presentation, no treatment was advised. The complex evolution of clinical signs in our patient underscores the need to distinguish disease progression from spontaneous involution in symptomatic patients with Bonnet-Dechaume-Blanc syndrome.

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References


