Table 1 Summary of four patients with haemorrhagic vitreoretinal complications whilst taking combined antiplatelet agents

Case	Age (years)	Sex	Antiplatelet drugs	Presenting complaint	Uncontrolled intraoperative bleeding	Outcome/acuity
1	83	M	Clopidogrel, diclofenac*	BE vitreous haemorrhage	Extraocular + intraocular (first eye)	RE further surgery. HM LE 6/12
2	72	M	Clopidogrel, aspirin	Recurrent RD under oil	Intraocular only	Redetached—further surgery, HM 6/6
3	86	M	Dipyridamole mr, aspirin, piroxicam*	LE vitreous haemorrhage	Extraocular only	
4	76	M	Clopidogrel, aspirin	LE vitreous haemorrhage	—	6/12

*Non-steroidal anti-inflammatory agent potentiating antiplatelet effect; mr, modified release; BE, both eyes; LE, left eye; HM, hand movement; RD, retinal detachment.

Comment

The drugs described have varying degrees of antiplatelet activity. NSAIDs competitively and reversibly bind cyclo-oxygenase (COX) and are ineffective as antiplatelet drugs alone, but may potentiate other agents. Aspirin irreversibly inhibits COX and, as monotherapy is not associated with increased risk of haemorrhage in vitreoretinal surgery.⁴

Clopidogrel inhibits adenosine diphosphate activity, impairing platelet aggregation. Dipyridamole also inhibits platelet aggregation, but through a number of different mechanisms. The action of these two agents can be deliberately potentiated by combining them with aspirin.

Clopidogrel and aspirin in combination are widely used following unstable angina¹ and coronary artery stenting,² while dipyridamole with aspirin is used for secondary prevention of stroke.³

Prescribing information for clopidogrel advises discontinuation 7 days before surgery if antiplatelet activity is not required.⁵ Intraocular haemorrhage has recently been reported following peripheral iridectomy in a patient taking both clopidogrel and aspirin,⁶ but vitreoretinal complications have not previously been described.

Our four cases do not prove causation, but in the absence of alternative aetiology it seems likely that impaired platelet function lead to the presenting complaint in cases 1, 3, and 4 and the intraoperative complications in cases 1 and 2. The antiplatelet combinations encountered may be intentional (cases 2 and 4); inadvertent (case 1), or both (case 3).

Cessation of medication in selected cases may be justified, but must balance the risks of doing so (particularly with drug eluting coronary stents) versus the risks of surgery with impaired clotting. Combination treatment is usually indicated for a defined period (up to 1 year for clopidogrel and aspirin) and it may be possible to defer surgery in elective cases. Aspirin monotherapy may be an acceptable alternative after discussion with the patient's physician.

With increased use of combination agents⁷ these problems may be encountered more frequently. Our cases suggest that caution should be exercised when operating on patients taking clopidogrel if there is concurrent use of aspirin or NSAIDs.

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Isolated foveolar detachment in Irvine-Gass syndrome

Irvine-Gass syndrome, which describes visual loss after cataract surgery as a result of cystoid macular oedema (CMO), typically produces petalloid leakage around the fovea on fluorescein angiography (FA) often associated with late hyperfluorescence of the optic disc.¹ Recently, optical coherence tomography (OCT) has confirmed the presence of perifoveal cystic spaces in vivo, consistent with histological specimens with CMO. OCT is increasingly used as a first test, before FA, for diagnosing unexplained visual loss after cataract surgery. Here we report a case of Irvine-Gass syndrome that demonstrated perifoveal leakage and late optic disc staining on FA, whose predominant OCT finding was a localised foveolar detachment. Isolated foveal detachment has been described in diabetic macular oedema² but we are unaware of any reports of isolated foveolar detachment in Irvine-Gass syndrome.

Case report

A 65 year old woman complained of decreased visual acuity in the right eye

5 months after uneventful cataract extraction. Her medical history was unremarkable. Visual acuity was 20/80 right eye and 20/25 left eye. Dilated fundus examination revealed a loss of the foveal depression in the right eye and normal foveal reflex in the left eye. FA revealed perifoveal leakage right eye and late optic disc hyperfluorescence (fig 1). OCT revealed a foveal neurosensory detachment without cystic spaces around the fovea (fig 2). She was treated with Lotemax and Voltaren in the right eye four times daily and 5 weeks later the acuity improved to 20/25 right eye with resolution of the CMO clinically.

Comment

Several facts support the diagnosis of Irvine-Gass syndrome in this case, including the poor initial visual acuity, the presence of perifoveal leakage and late disc hyperfluorescence on FA, the onset of symptoms 5 months after cataract extraction, and the improvement in vision on topical steroid and non-steroidal drops.³ In Irvine-Gass syndrome, leakage from the retinal vasculature typically leads to accumulation of intraretinal fluid with secondary visual loss. Accumulation of fluid beneath or within the retina requires disruption of cell-cell contacts that ordinarily maintain relative dehydration of potential extracellular spaces. Cell-cell apposition within the retina is mediated by cadherins on Müller cells and retinal neurons.⁴ Accumulation of intraretinal fluid in Irvine-Gass syndrome implies that leakage from the retinal vasculature exceeds the rate of fluid resorption, and that there is disruption of cadherin mediated cell-cell contact within the retina. Our observation of isolated subretinal accumulation of fluid indicates that in this case there was disruption of the normal apposition between the retinal pigment epithelium and photoreceptors, rather than disruption of cadherin mediated cell-cell contacts within the retina. Clinicians should be aware that the clinical and/or OCT finding of an isolated neurosensory detachment, which has previously been seen in diabetic macular oedema, can also occur in Irvine-Gass syndrome.

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Figure 1 Fluorescein angiogram at presentation. (A) Patient's right eye at 3:02 post-injection demonstrates perifoveal leakage in a petaloid pattern and hyperfluorescence of the optic disc, both strongly associated with the Irvine-Gass syndrome. (B) Patient's left eye at 3:09 post-injection, shown for comparison. Note the absence of any perifoveal staining and absence of disc hyperfluorescence.

Figure 2 Optical coherence tomography of the right eye at presentation. Note the serous neurosensory detachment and the loss of the foveolar depression. There is no visible intraretinal oedema or cystic change in the perifoveal area.

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Two cases of *Burkholderia cepacia* endophthalmitis

Although *Staphylococcus epidermidis* is responsible for the majority of cases after cataract surgery, any bacteria may cause postoperative endophthalmitis. *Burkholderia cepacia* (previously known as *Pseudomonas cepacia*) is a Gram negative, oxidase positive, non-fermentative bacilli. *Burkholderia cepacia* does cause significant infection not only in cystic fibrosis, chronic granulomatous disease, and immunocompromised patients, but also in healthy individuals.¹ It has been shown to be resistant to some antiseptics such as benzalkonium chloride, chlorhexidine at standard or high concentrations, and even be alive in those antiseptics.²

Case 1

A 63 year old diabetic patient was diagnosed with endophthalmitis after an uneventful phacoemulsification and foldable intraocular lens (IOL) implantation. A vitreous tap was performed. Intravitreal vancomycin (1 mg/0.1 ml) and ceftazidime (2.25 mg/0.1 ml) were administered. Intravenous vancomycin (500 mg twice daily) and ceftazidime (500 mg twice daily) in addition to hourly topical fortified tobramycin and hourly cefazolin were added to the therapy. The vitreous culture grew *Burkholderia cepacia* on the third day. The identification of the organism was performed by standard biochemical tests and also with Analytical Profile Index for Gram Negative Identification (API 32 GN, Bio Merieux, France). It was sensitive to ceftazidime, ciprofloxacin, ofloxacin, and was resistant to ceftriaxone, cefoxitin and tobramycin, amikacin, and gentamicin.

Best corrected visual acuity (BCVA) was hand movements (HM) on the fourth day of the therapy. Pars plana vitrectomy (PPV) and injection of ceftazidime (1.12 mg/0.1 ml) were performed. Repeated vitreous sampling grew the same organism and antimicrobial susceptibility test was also the same as the previous culture. The patient was discharged from the hospital with a quiet anterior chamber and clear vitreous cavity. Two years later, the BCVA remains at 20/63.

Case 2

A 72 year old man presented with decreased vision and pain in the left eye 15 days after an uneventful phacoemulsification with foldable IOL implantation. BCVA was HM. Slit lamp examination findings were the same with patient 1. Treatment consisted of tap and injection of same dose of intravitreal vancomycin and ceftazidime. Three days later, BCVA was still HM and the hypopyon had not resolved. PPV and intravitreal reinjection of vancomycin and ceftazidime was performed. Hourly topical fortified tobramycin and cefazolin were added to the therapy. Vitreous culture grew *Burkholderia cepacia* on the fourth day. Antimicrobial susceptibility test was the same as the other patient. During the following days the anterior chamber and vitreous cavity cleared. At the 6 month follow up, his BCVA was 20/50.

Comment

To our knowledge, there are only two previously documented postoperative endophthalmitis cases following cataract surgery that were caused by *Burkholderia cepacia* (Medline search).^{3,4} One of the cases was a recurrent endophthalmitis.⁴ The organism has an unusually large genetic make up that accounts for its microbiological versatility. It also produces lipopolysaccharide and β lactamase that renders some antibiotics ineffective against it.⁵ Resistance to aminoglycosides noticed in the previously reported cases^{3,4} was also noted in our both patients. Close follow up for possible early PPV might be considered as our cases were not cured after the first intravitreal injections even though appropriate antibiotics were used.

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MAILBOX

Dietary lutein and zeaxanthin: authors' response

We were interested to see the letter by Mitchell,¹ written in response to our earlier publication.²