Case Record 3

Artery Occlusive Disease.

3a Central Retinal Artery Occlusion
3b Multiple fibrin-platelet arteriole emboli

June 2012

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aaron optometrists

The benefits of instigating outcome audits as part of clinical governance procedures for community optometrists.

Community optometrists work largely in isolation and in many cases deal primarily with healthy individuals. The strength for primary care clinicians is the ability to follow all interesting presentations, building patient trust as well as maximising every learning opportunity.

Kanski and Bowling (2011) suggest it is not uncommon to detect asymptomatic isolated vascular emboli in an aging demographic. While not routine this is certainly a regular finding in this practice, as well as assessment of amaurosis fugax for Anterior Arteritic Optic Neuropathy.

Presented however, are two cases of arterial occlusion; a Central Retinal Artery Occlusion (CRVO) and multiple, fibrin-platelet emboli. Both presentations are rare to see in community practice. Both were followed closely.

Case 3a CRAO
May 2012.

Salient information taken from electronic records

DATE: 2/5/12

Mrs Age : 78

Presenting Symptoms

04/12 – LE vision loss – GP sent to HES. Told she had stroke – not hospitalised. Feeling generally well, but already on multiple meds. (No motor defects, no sensory defects apart from vision, no aphasia).
Not bumping into things, navigational vision seems good but pouring fluids is difficult - lost vision in LE only, Annotate 3.1
HES did investigate – discharged but told to go onto Aspirin.

POH

Varifocals. Aware of cataracts. No previous ocular surgery or treatments.

FOH : Nil.

General Health and Medications
Stroke (?) 04/12 – Advised Aspirin. Already on Warfarin
Heart - New Valves - Warfarin, Digoxin, Isosorbide, Bisoprolol, Furosemide
Optical Migraine
Cramps - Quinine
Carpel Tunnel Syndrome - ops
Losec
Allergy - Flu Vaccine, Elastoplast
No hayfever, Non-Smoker

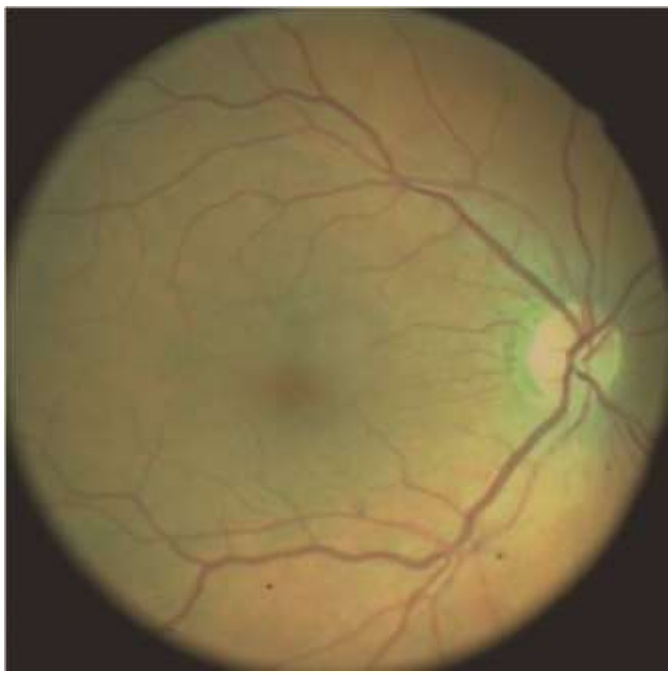
Refraction

R +4.25/-1.00x90 (6/7.6)	Add +2.50 N5
L +4.25/-1.25x90 (<6/120)	Add +2.50

Tensions (GAT) (11.12am)	R 20 L 19
Confrontation Fields	R Full to confrontation L Visual discrimination nil
Pupils	L RAPD

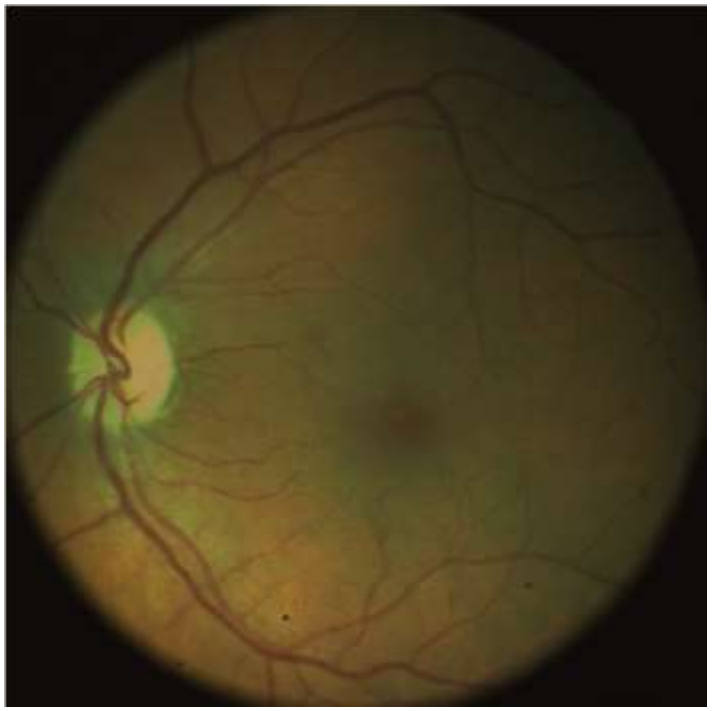
Dilated Fundsocopy (1.0% Tropicamide)
RIGHT CD 0.1 rims good, no bayoneting, no baring. Neural rims healthy. Vascular nipping and calibre changes – stable, of long standing.

RE 2008 RE 2/5/12



LEFT CD 0.1 Disc collaterals.
Attenuation and obliteration of retinal arterioles. Ischaemic zone with cherry red spot at macula.

LE 2008 LE 2/5/12



Advice and CMP

The patient was already fully aware of the poor prognosis for visual recovery in the affected eye. Advice on stereopsis and the use of other clues to help detailed daily tasks was given. Reassurance that, while the vision will not improve, adaption to the problem should. The mobility implications of a monocular, pre-chiasmal lesion, as opposed to a chiasmal or post-chiasmal lesion, were emphasised.

Unfortunately no reassurance that additional systemic management could guarantee protection of the fellow eye from a similar occurrence could be given.

Discussion

Case histories should guide the clinician toward tailored examination techniques.
Gross examination of the patient as she moved into the examination room, a potentially overlooked process, did not 'red flag' anything of note.
A report of stroke immediately suggested the possibility of a homonomous field defect; the primary expectation being one of homonomous hemianopia. (Budenz 1997, Lee et al 2010). The presenting symptoms did not correspond. The patient did not report, or demonstrate, gross signs of motor or sensory defects or aphasia. The patient, when questioned specifically, did not report mobility problems; found clinically to be a virtually universal complaint with homonomous hemianopic loss. Rather more specific frustrations associated with detailed tasks such as pouring drinks was reported. Confrontation fields demonstrated a full field for the right eye but no discernable vision for the left; this corresponded to a significant relative afferent pupil defect.

It was post refraction that dilated fundoscopic examination revealed the underlying visual problem.
The term 'stroke' is a poor one. Cerebrovascular accident (CVA) is the loss of brain function due to compromised blood supply. Broadly this compromise will be due to either ischemia or bleeding, but a plethora of primary aetiologies are possible (WHO 1978).

Rudkin et al (2010) describe CRAO as a 'stroke' of the eye, caused by occlusion of the central retinal artery, usually by embolus or thrombus. However, other causes of CRAO specifically can include a fall in perfusion pressure in the central retinal artery, induced by nocturnal hypotension or shock (Hayreh 2005). Kanski and Bowling (2011) and Jain (2012) also mention inflammatory aetiologies such as endarteritis and vasospastic phenomenon including retinal migraine. Jain (2012) suggests the mechanism of obstruction may be obvious from comorbid systemic disease or fundoscopic findings but this did not seem to be the case for this patient.

Emboli are visible in only 20% of CRVOs (Graham 2012), although frank detection does not eliminate the possibility; amaurosis fugax, regardless of the absence fundoscopic signs is considered a symptom of transient retinal artery embolism (Cugati et al 2006). Hayreh and Zimmerman (2005) consider embolism the primary cause of CRVO,

although this is in conflict with the general view that atherosclerosis is the primary aetiology (Graham 2012, Kanski and Bowling 2011). Graham (2012) stresses the need to assess for this malady with appropriate blood screens as well as for other, less common, aetiologies including anaemia and platelet disorders, inflammatory disorders especially arteritis, and clotting disorders.

Acute treatment of CRVO is time critical; Hayreh and Zimerman (2005) suggest that unless caught within 240 minutes, irreversible retinal damage will occur. Kanski and Bowling (2011) and Jain (2012) report more generous windows of opportunity but add there is little evidence of benefit; Rudkin et al (2010) certainly indicate that there is little quality evidence of efficacy. By the time this patient was seen within ophthalmology there was no question of attempting the plethora of poorly evaluated techniques summarised by Hayreh and Zimmerman (2005), Kanski and Bowling (2011) and Rudkin et al (2010).

Prophylactic treatment modalities should reflect the aetiology. Kanski and Bowling (2011) as a general text mention lifestyle modification, antiplatelet therapy, anticoagulants and carotid endarterectomy surgery as possible managements.

This patient was already maximally treated. The patient has prosthetic heart valves which necessitates Warfarin. She is under very regular cardiology review and complies with regular mandatory blood screens. As well as being licensed for prophylaxis of systemic embolism after insertion of prosthetic heart valves, Warfarin is also used for general systemic and pulmonary emboli, venous thrombosis and transient cerebral ischaemia (eMC 2010). The Summary of Product Characteristics (eMC 2010) report exaggerated anticoagulation as a primary risk and concomitant therapy with any drug that increases the risk of bleeding is contra-indicated.

Aspirin was recommended. Unless the aetiology was specifically ascertained this recommendation could be unnecessary but more importantly concomitant use with warfarin could trigger a haemorrhagic cerebrovascular accident with potentially devastating consequences.

Case 3b
Multiple fibrin-platelet arteriole emboli
December 2009

Salient information taken from electronic records

DATE: 1/12/09

Mr Age : 57.
Address

Presenting Symptoms
Current specs seem poor Near and Dist – wants better spectacles. Duration of symptoms – unsure seems gradually getting worse. Last exam 6/12 ago elsewhere. No Headaches. No diplopia. No Flashes/floaters.

POH
Near spectacles only. No previous ocular surgery or treatments.

FOH :
Nil.

General Health and Medications
BP – Lisinopril, Atenolol
Cholesterol - Atorvastatin
Chronic Pain- Amitriptyline, Codydromol
No allergies, No hayfever
Smoker.

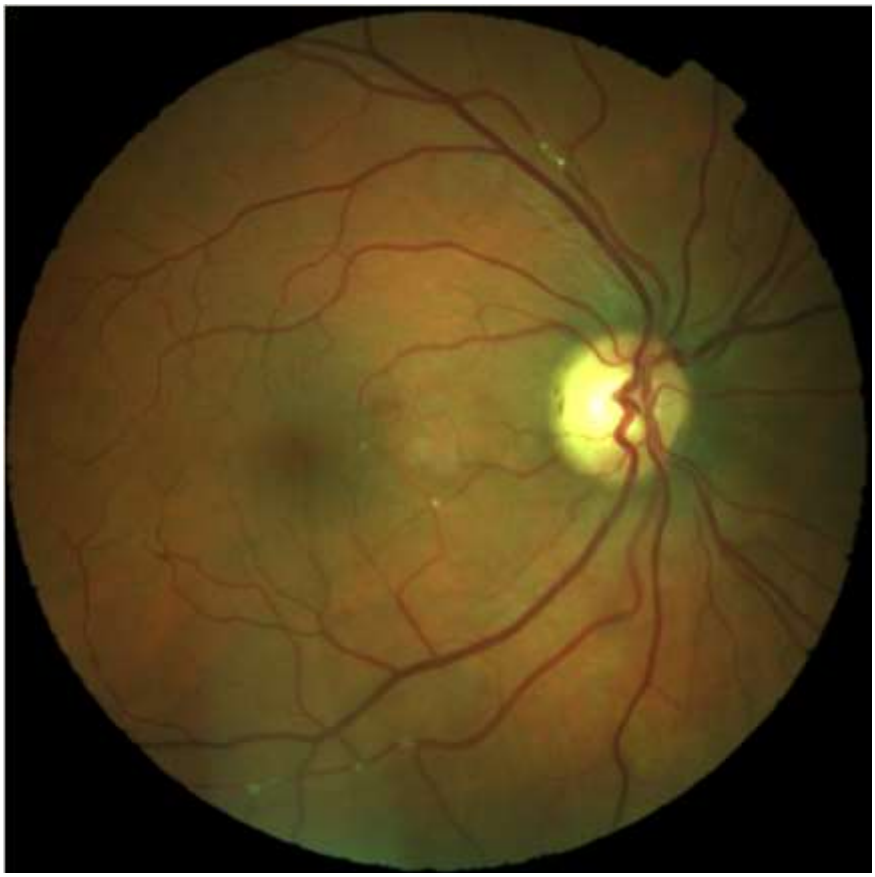
Refraction
R +1.00DS (6/6) (but searching) Add +2.50 N5
L +1.25DS (6/6) Add +2.50 N5

Tensions (GAT) (3.45pm) R 10 L 10
AMSLER Fields R Isolated Scotomas
Pupils E&A D,C& N

Slit Lamp
VH 4 Angles open, Iris configuration Flat. AC Clear – no pigment. Corneas clear.

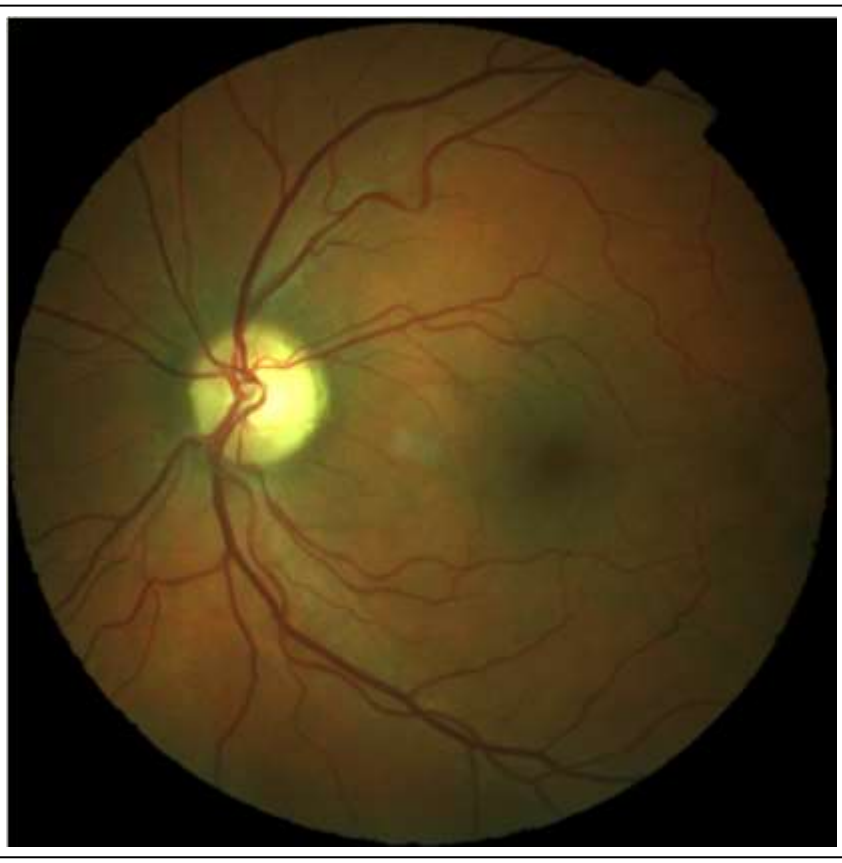
Dilated Fundsocopy (1.0% Tropicamide)

RIGHT CD 0.4
Rims good, no bayoneting, no baring.
AV 2/3, no nipping, no calibre changes.
BUT
Numerous emboli throughout superior and inferior vascular tree- appearance fibrin-platelet.
Possible intra-retinal haemorrhages peri-macular.



LEFT CD 0.4
Rims good, no bayoneting, no baring.
Vascular tree Normal. AV 2/3, no nipping, no calibre changes.

No emboli



Differential diagnosis of Emboli – 1) Fibrin-Platelet 2) Cholesterol

Advice and CMP

Advised that spectacles not the problem. Urgent referral for cardiac and vascular assessment required. Letter delivered to GP by hand.

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INDEPENDENT Prescribing
OPTOMETRISTS
Bellway House, Woodhorn Road, Ashington,
Northumberland NE63 0AE, Tel:01670 813185

Dr
Lintonville

Re

1/12/09

Dear Dr

Mr presented reporting blurriness, which he felt was the spectacles. Subjective refraction gave :

R +1.00 DS (6/6)	Add +2.00 (N5)
L +1.25 DS (6/6)	Add +2.00 (N5)

Dilated fundsocopy showed extensive emboli throughout the vascular tree in the right eye only. The left eye is clear.
I am very concerned that these emboli could be fibrin-platelet possibly suggesting severe stenosis of the right carotid artery. Mr requires urgent blood screen and possible referral to ophthalmology.

Yours faithfully

Peter Frampton

Company Registration No 07022426
VAT Registration 979 4947 34

Winner Outstanding Optometrist
Northumberland NHS Care Trust Winner Innovation in Practice Awards
Winner Technology Practice of the Year

Outcome Audit – February 2010.

Carotid Endarterectomy – Freeman Hospital

HES - Mr Ophthalmology – January 2010 - Discharged VA 6/5, 6/4

Medications:
BP – Lisinopril, Atenolol
Aspirin
Cholesterol - Atorvastatin
Chronic Pain- Amitriptolone, Codydromol
No allergies, No hayfever
On cessation of smoking programme.

Discussion

Sharma et al (1998) and Law (2010) indicate the difficulty in differentiating emboli type based on fundoscopic examination alone; systemic management should not rely on qualitative fundoscopic appearance. Howard and Russell (1987) report that fibrin-platelet emboli are rarely observed, inducing symptoms of amaurosis fugax but passing through the retinal vasculature before being observed. Indeed, these authors only identified one patient with fibrin-platelet emboli in their study.
Regardless of type, emboli represent a significant indicator of life threatening pathology. Howard and Russell (1987), Klein et al (1999) and Jain (2012) all report the increased risk of stroke in patients with visualised retinal emboli and amaurosis fugax (Poole and Russell 1985). Surprisingly however, Sharma et al (1998) indicate that observation of retinal emboli did not significantly alter the probability of anti-coagulant treatment or cardiac surgery. This may reflect the fact that these patients are already being treated for systemic disease but also, since emboli are rare (Klein et al 1999), studies do not necessarily differentiate emboli type or number. Klein et al (1999), for instance, simply differentiated dull and bright emboli. Howard and Russell (1987) did differentiate emboli type but the authors only detected one patient with fibrin-platelet emboli making realistic prognostic estimates impossible; this patient did not suffer visual loss, did undergo carotid endarterectomy but subsequently still suffered a myocardial infarction.

These reports do not alter the urgency with which patients need to be assessed but rather reflect the poor sensitivity of identifying underlying

causes based on emboli visualization. Howard and Russell (1987) make the sobering observation that, considering the volume of cerebral and retinal circulation, for every embolus observed in the retina a much larger number must have been carried to the brain.

This patient underwent emergency Carotid Endarterectomy. Klein et al (1999) indicate that the risks with this procedure, outlined by Biller et al (1998) make the decision to operate depend on severity of carotid stenosis, age, severity of co-morbidities and indeed the skill of the surgeon. Biller et al (1998), also stress the need to manage concurrent risk factors, particularly hypertension, smoking, blood lipids and alcohol consumption.

Final Statement

Notwithstanding the earlier discussion of diagnostic sensitivity of emboli identification within the primary setting, the number and symptomatic nature of the emboli strongly suggested serious underlying health issues. The surgical outcome was no surprise.

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