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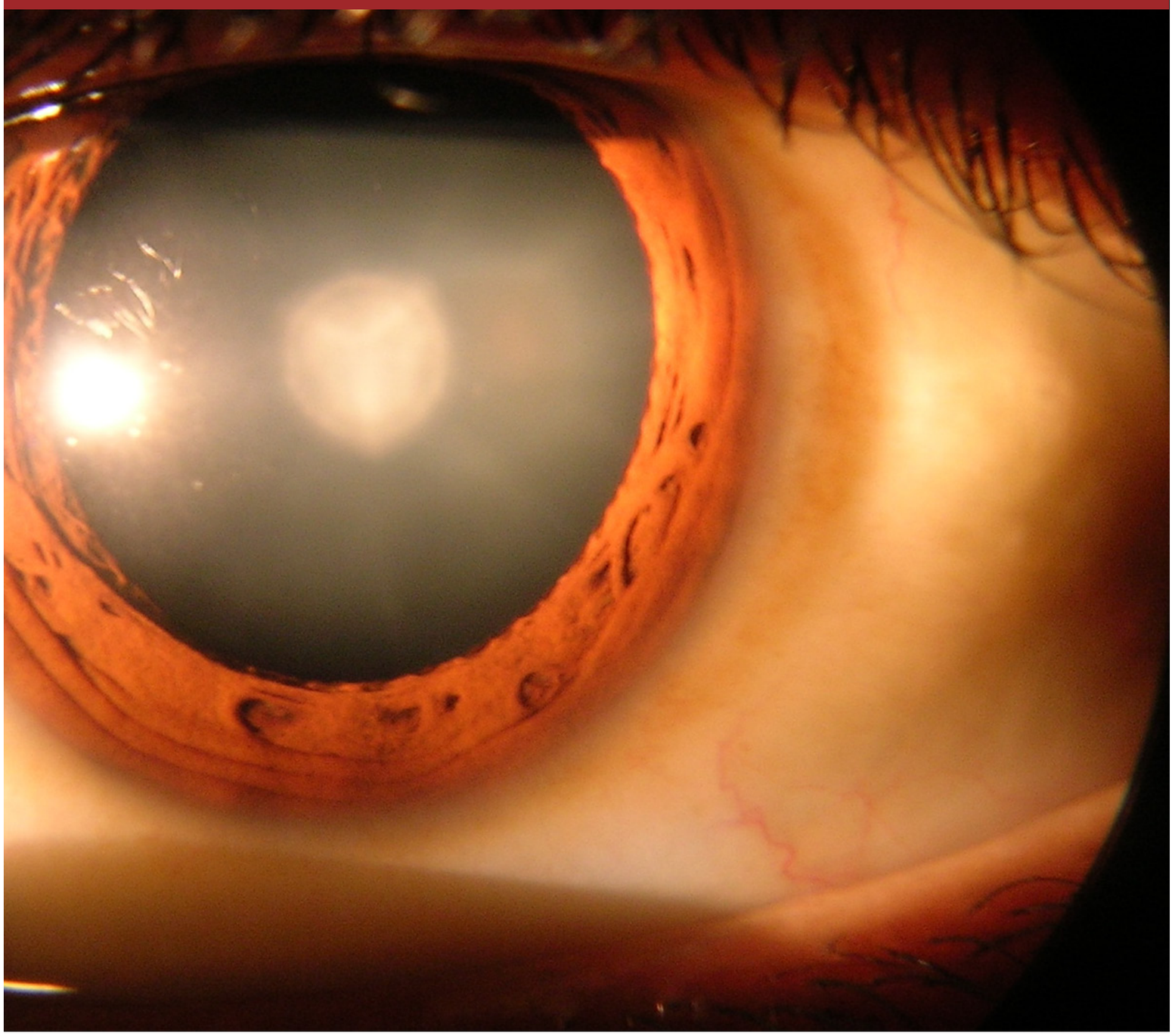
Volume 6, Issue 10: **Ophthalmology**



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Volume 6, Issue 10

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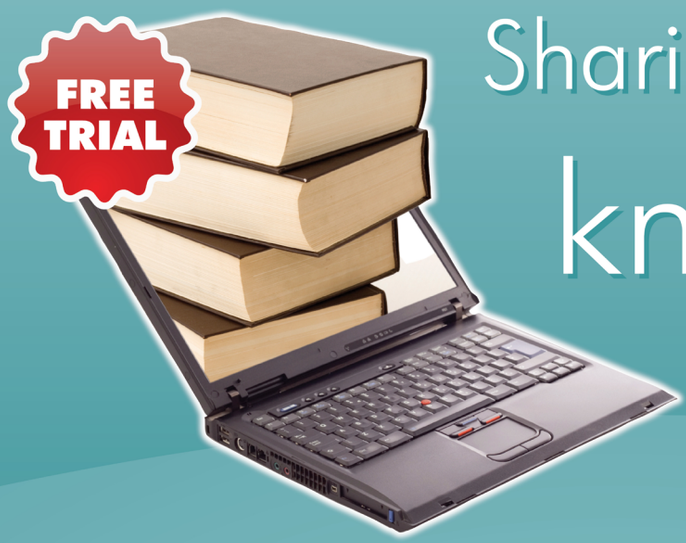

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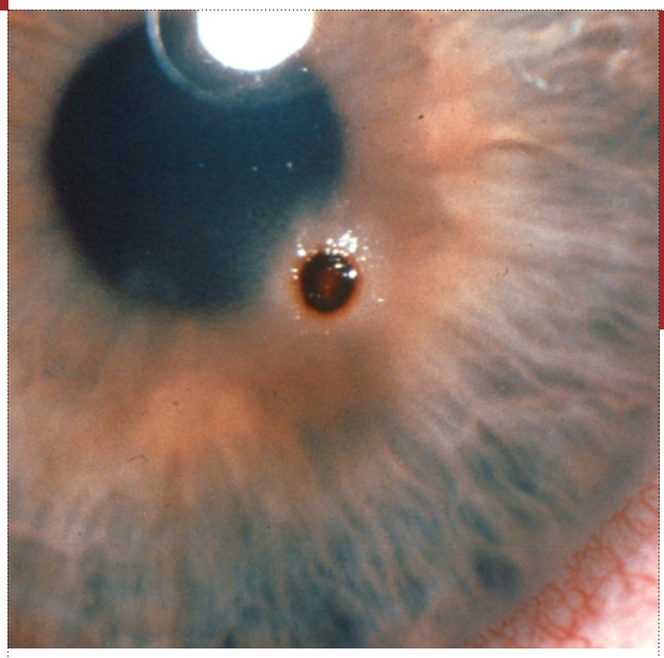
AN EMERGENCY PRESENTATION OF ACUTE ANGLE CLOSURE (AAC)

R Dickson-Lowe, J Keenan



Abstract

This article gives a brief history of a case of acute angle closure (AAC) presenting to the Medical Assessment Unit (MAU). This is followed by a discussion about terminology, demographics and predisposing factors, history taking (clinical signs and symptoms), eye examination, differential diagnoses and management strategy. In particular, important findings in a patient presenting with AAC that should not be missed and will aid with the decision process of 'admit or discharge' will be discussed.

Case History

A 64-year-old man presented to the Medical Assessment Unit, with a 12 hour history of sudden onset left-sided headache, centred around the left eye. On approach he had his left eye tight shut, his head bent to the left side and his hand over the affected eye. He described the eye as feeling gritty and sore and that keeping the eyelid closed lessened the intensity. The intensity was about 8-9/10 and was constant and not made better with analgesia (co-codamol 30/500) prescribed following his visit to the Emergency Department (ED) 8 hours previously.

Nothing made the pain better, but opening the eye made it somewhat worse. There were associated symptoms of vomiting (once) and constant nausea. There was no definite photophobia despite the pain being worse with the eye open because it was the same increased pain in the darkness.

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Patient Management

He had never had this experience before. The patient's concerns were that he had a tumour in his brain or that he had a bleed into his brain. As mentioned, he had been seen in the ED ten hours earlier with similar symptoms, but was sent home with analgesia, as this had some effect at the time, and the diagnosis of tension headache.

His observations showed that he was apyrexial, with a blood pressure of 142/85mmHg, a regular pulse of 100bpm and oxygen saturations of 99% on room air. His neurological examination showed normal appearance, tone, power, sensation, reflexes and coordination in the upper and lower limbs. The cranial nerve examination revealed a normal pupil on the right side which reacted to light and accommodation but a dilated and fixed left pupil with a consensual pupillary reflex present in the right eye when a light was shone into the left eye.

There was pain in the left eye in all ranges of movement when testing CN III, IV and VI. The visual acuity was 6/24 (the patient did not wear glasses) in the left eye, compared with 6/6 in the right eye. There was not however any photophobia or diplopia. The left eye looked red, with injected conjunctiva and sclera, a hazy cornea, and profuse tearing. Fundoscopy was difficult in the left eye as the cornea was hazy due to corneal oedema, and difficult to keep open. Fundoscopy on the right side was normal. GCS was 15/15 at all times. To complete the full medical examination, his heart sounds were normal, with no signs of heart failure, the chest was clear and the abdomen soft and non-tender with normal bowel sounds.

Initial investigations were similar from both the initial ED presentation and the following MAU assessment, and included a normal ECG (done as heart rate was around 100bpm) and no abnormality on Full Blood Count, Urine and Electrolytes, Liver Function Tests, Glucose, Clotting and C-Reactive Protein. No scans of the brain were undertaken after assessment.

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Discussion

What are your initial thoughts about this presentation to MAU and ED?

It appears that in this busy ED, important differentials may have been dismissed for the more straightforward diagnosis, despite the answer being present. The causes of sudden onset headache of this nature and acute red eye are important.

What differential diagnoses can you think of for sudden onset headache in this case?

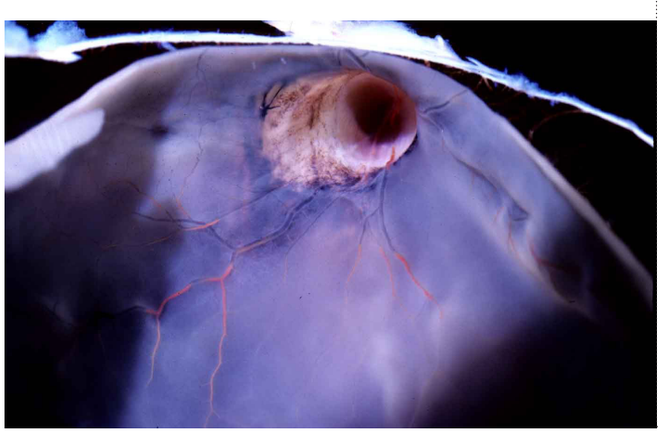
Sub-arachnoid haemorrhage
Migraine
Tension-type headache
Cluster headache
Giant Cell Arteritis (GCA) / Temporal Arteritis
Trigeminal neuralgia
Carbon monoxide poisoning

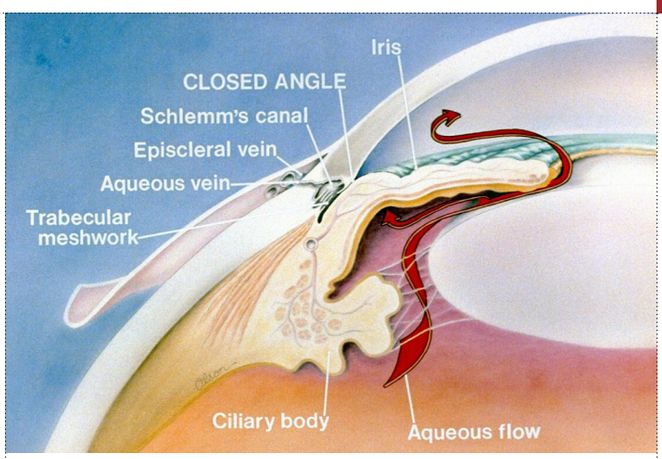
What differential diagnoses for the acute red eye can you think of and what might you find on examination?

Cause	Pain		Vision	Pupil	Conjunctival injection	Secretions
	Severity	Uni/Bi				
Conjunctivitis	-/+	Uni/Bi	Normal	Normal	Diffuse	Pus
Anterior Uveitis	+/++	Uni	↑	Constricted Irregular	Circumcorneal	Serous
Acute angle closure	+++	Uni	↓	Dilated	Diffuse	Serous

Table 1: The Acute Red Eye (1)

Other common causes of red eye are dry eyes and blepharitis but these are often bilateral. Initially it was the headache that was the main presenting symptom (not interpreted as 'eye pain') but with a more detailed history and examination of the eye on re-presentation to MAU it became clear that this was eye pain and the 'red eye' presentation.





How can we use the correct terminology?

Primary angle closure (PAC), primary angle closure glaucoma (PACG), acute angle closure (AAC) and acute angle closure glaucoma (AACG) are terms that are often used interchangeably for the situation of raised intraocular pressure (IOP). In fact the angle may be closed acutely (as in AAC and AACG) or insidiously (as in PAC and PACG).

Glaucoma is a disease in which the optic nerve is damaged and can result in irreversible loss of vision usually associated with raised intraocular pressure. (2) AAC is an ophthalmic emergency requiring urgent treatment otherwise permanent visual loss may ensue due to damage of the optic nerve and glaucoma.

Some people's angles are naturally very narrow which predisposes the angle to blocking off. Severe hypermetropes fall into this category. In this case it is known as primary AAC. Other susceptible patients include those with a thin iris, a naturally thick lens and a shorter axial length of the eyeball (front to back). Secondary AAC occurs as a result of forces exerted on the iris either anteriorly (e.g. secondary to peripheral anterior synechiae pulling the iris up) or posteriorly (e.g. the lens bulging forward as a result of swelling).

This can be contrasted with secondary open angle glaucoma where the iridocorneal angle remains open but is blocked off by blood (e.g. hyphaema), blood vessels (e.g. advanced diabetic eye disease) or proteins (e.g. hypertensive uveitis). This is managed within the context of ophthalmology clinics and is not discussed in this article.

AN EMERGENCY PRESENTATION OF ACUTE ANGLE CLOSURE (AAC)

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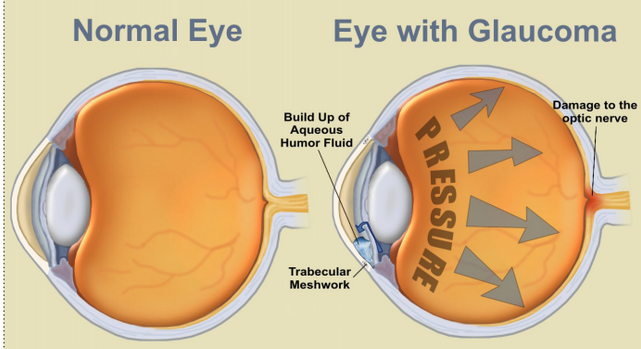

The Clinical Presentation

It causes a painful, red eye with blurring of vision, haloes around bright lights, headache, nausea and vomiting, all of acute onset and often occurring at night (3). In some cases there are no eye symptoms. (4)

Signs of AAC

Semi-dilated and fixed pupil which is unresponsive to light, a hazy cornea, reduced visual acuity, tense eye which is painful and red. (2,3)

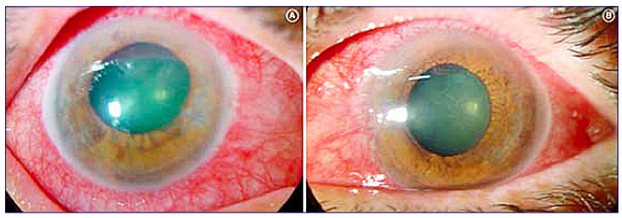

Figure 2. Conjunctival hyperaemia, corneal oedema, iris atrophy (it is a late sign), and fixed, moderately dilated pupils are seen in the left (A) and right (B) eyes. (5)

On re-examination of the History how could this have been recognised more easily?

We know that the patient presented with a 'headache' to ED 8 hours previously, which was on the left side and focused around the eye with intensity about 8-9/10. This was the worst pain in his head that the patient had ever had. There was one episode of vomiting and nausea. There was no history of trauma, recent sexual activity (relevant in coitus cephalgia) and no significant previous medical history (relevant for sarcoidosis or ankylosing spondylitis which would suggest an inflammatory eye condition such as uveitis).

However family history had not been explored and a full CN and eye examination had not been done with no attempt recorded to look at the retinae. This patient had a significant family history of glaucoma (most likely primary open angle glaucoma and not acute angle closure glaucoma) in the females in his family.

What predisposing factors for AAC are there?

Anatomical factors have been mentioned earlier with terminology for AAC. Epidemiological factors include age, sex and race. Consistent with the case presentation described, females are affected more than men at 3-4:1, which may be related to their gonoscopically narrower angles compared with men. (6) Increasing age is also a major risk factor. (7)

It is present in about 0.1% of the general population over 40 years old, but up to 1.5% of the Chinese population over 50. Singapore has the highest incidence of primary AACG at 12.2 per 100 000 per year in those over 30. The prevalence of primary acute angle closure glaucoma is highly race-dependent, with lowest rates in European whites and highest in Inuits, with East Asians in between.(7) This patient was of white British nationality.

What would be the important points to consider in suspicion of AAC?

1. Is acuity affected? – YES, in this case there was blurring of vision.

2. Is the globe itself painful? – YES, with and without palpation.

3. Does the pupil react to light? – NO, and the pupil was significantly dilated.

4. Is the cornea intact? – YES, but there was corneal oedema (haziness) and thus deeper structures were not easily visible.

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Should this man have been sent home from ED?

The pain went down to 4-5/10 with co-codamol in ED. The answer can be argued either way – a severe headache that he has never had before with no trigger for onset and causing an ED visit by a man who never goes to the doctor. A watch and wait management plan does seem acceptable and was approved ultimately by the senior ED staff member, as recorded in the notes. On the other hand you could argue that the relevant questions were not asked, or not recorded, and the relevant examination wasn't done, or not recorded, and thus they may have recognised AAC earlier and not sent him home. The message here is that any presentation of acute headache or pain focused around an eye should warrant full cranial nerve, neurological and eye examinations, and recording in the notes!

Eye Examination

A foundation doctor should be undertaking and recording at the very minimum the findings from the eye examination outlined below:

1. External examination – eye lid, conjunctiva, cornea, sclera, iris, pupil

2. Visual acuity – Snellen chart (CN II)

3. Colour vision – Ishihara charts (CN II). Often Ishihara plates are not available in non-ophthalmic departments, instead red desaturation test is much easier to perform with any available red target.

4. Visual fields (CNII)

5. Pupils – reaction to light and accommodation (CN II and Autonomic system)

6. Eye movements (CN III, IV, VI)

7. Ophthalmoscopy/Fundoscopy

With all this information from the examination in a patient who presented with sudden onset painful, red eye with reduced visual acuity, a hazy cornea, and a fixed, dilated pupil, making a diagnosis of AAC appears more straightforward.

How does AAC happen?

The only treatable risk factor for glaucoma is raised intraocular pressure, which is a function of production of liquid aqueous humor by the ciliary processes of the eye and its drainage through the trabecular meshwork. (8) Aqueous humour flows from the ciliary processes into the posterior chamber, bounded posteriorly by the lens and the zonules of Zinn and anteriorly by the iris. (9) It then flows through the pupil of the iris into the anterior chamber, bounded posteriorly by the iris and anteriorly by the cornea. (2,8)

From here the trabecular meshwork drains aqueous humor via Schlemm's canal into scleral plexuses and general blood circulation. (9) In primary acute open angle there is reduced flow through the trabecular meshwork; in primary acute angle closure, the iris is pushed forward against the trabecular meshwork, blocking drainage (2,8,9) as described earlier.

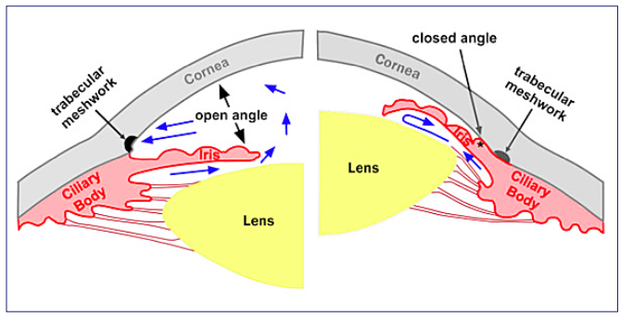
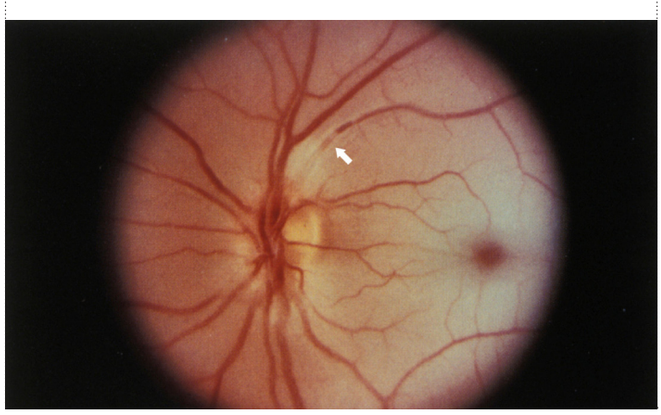


Figure 1. A schematic outlining the mechanisms behind open angle (left side of the diagram) compared with closed angle (right side of the diagram) (10)

What should be done in a case where there is high suspicion of AAC?

The first thing that was done here was to inform my seniors immediately with a view to arranging an immediate and urgent referral to ophthalmology. Acute angle closure is sight-threatening and 12 hours after onset, this was an emergency that could not be put off. The medical registrar on-call quickly reviewed the patient after my assessment and made a call to the eye clinic within the hospital and the patient was wheeled down to the Eye Unit immediately.

There was a significiantly raised intraocular pressure in the left eye but also in the right eye and the diagnosis of acute angle closure was made. The patient received pilocarpine 4% drops which were taken hourly, acetazolamide 500mg IV and morphine for the pain (which had already been given in MAU) and metoclopromide for the nausea, as immediate treatment. Following this the patient was booked in for a peripheral iridotomy using laser for the following day, which entails removal of a part of the iris to allow free circulation of aqueous humour.



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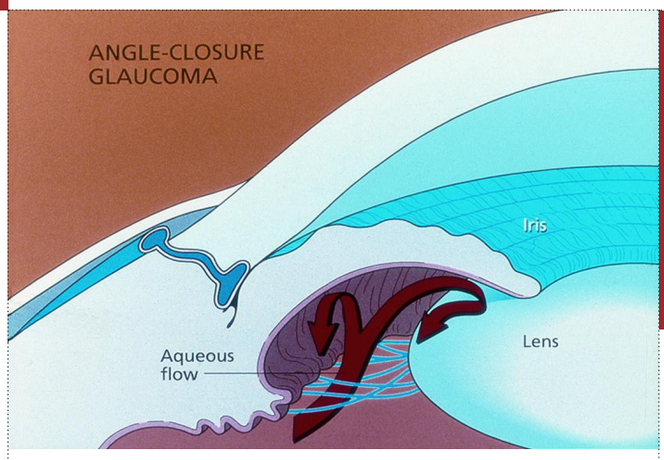

More specific investigations (carried out by the ophthalmologist in the Eye Clinic) (8,9)

1. Slit lamp examination – eyelid, cornea, conjunctiva, sclera, iris, lens, pupil

2. Indirect ophthalmoscopy – retina, retinal vessels, optic nerve

3. Tonometry – measure of intraocular pressures

4. Gonioscopy – looks at the iridocorneal junction (useful in glaucoma)

Management of acute angle closure

1. EMERGENCY – contact ophthalmology specialist
2. Analgesia e.g. morphine as pain is severe, given IV as patient often vomiting
3. Antiemetics e.g. metoclopromide, given IV as patient often vomiting
4. Medications given by ophthalmologist

a. Pilocarpine eye drops (miotic/parasympathomimetic) – constricts the pupil by contracting the iris sphincter muscle. (11,12) Also causes the cililary muscle to contract and therefore opens the trabecular meshwork through increased tension of the scleral spur.

b. Acetazolamide (carbonic anhydrase inhibitor) – reduces IOP by decreasing aqueous secretion from the ciliary body. (11,12)

c. Timolol - reduces IOP by decreasing secretion from the ciliary body.(8,9) N.B. beta blockers should be avoided in asthmatics.

d. Latanoprost (prostaglandin analogue) – increases uveoscleral outflow of aqueous humour thus decreasing IOP (11,12)

e. Topical betamethasone/hydrocortisone (steroid) – to reduce inflammation

5. Laser/surgery – this is often carried out soon after the initial presentation to prevent recurrence and sometimes the lens/cataract is removed to create more space for the iris to fall back. This involves a peripheral laser iridotomy to improve drainage by reducing the angle between the iris and the trabecular meshwork. (11,12) The unaffected eye is always prophylactically lasered.

6. Follow up with a fully qualified ophthalmologist (13) if the AAC is not successfully treated.

An emergency presentation of Acute Angle Closure (AAC)
Patient Management

Key Learning Points

1. Acute angle closure is a medical emergency and needs urgent referral.

2. Never dismiss acute angle closure in presentations to ED involving the eye.

3. Take a thorough history, especially previous medical history, family history and drug history.

4. Ensure that pain relief is given early on to allow for a more fruitful history and allow for a more thorough examination.

5. Follow up should be regular at the initial stages with a fully qualified ophthalmologist to prevent visual loss. Once the pupil block is broken and the IOP controlled the patient will eventually be discharged – often after 12 months follow up.

Questions

1. With regards initial presentation of acute angle closure, the answer most likely to be correct is:

a. Pain is often manageable at home
b. The pupil is dilated but still reacts to light and accommodation
c. Flashing lights are often experienced
d. The fundus is always normal
e. Vomiting is common

2. With regards the action of a foundation doctor diagnosing acute angle closure, all can be undertaken except:

a. An ophthalmologist should be contacted as soon as possible
b. Morphine and Metoclopromide should be given IV to manage symptoms
c. A CT scan should be arranged immmediately to rule out intracranial pathology
d. The drugs that will be most effective in treatment are pilocarpine and acetazolamide.
e. Patient's drugs history should be noted including allergies and any drugs causing the presentation stopped.

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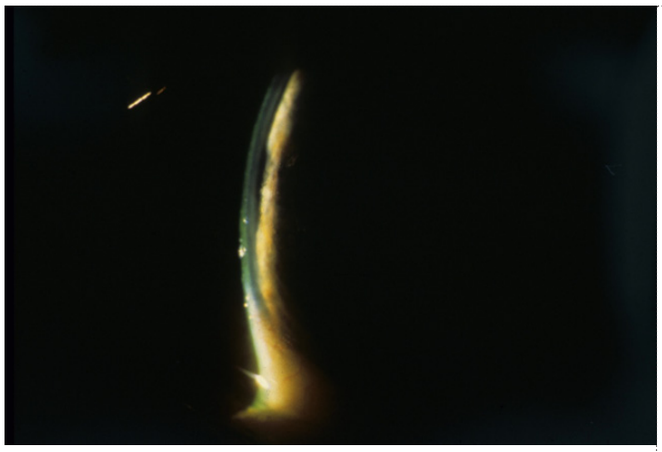

Answers

1. Answer: e

Is the most likely answer to this question. Pain is often very severe and the cause for presentation to ED. As in the case above, the patient who rarely went to the doctor presented to ED twice due to the pain of the headache and in the eye. The pupil is dilated but reacts poorly, if at all to light and accommodation. Haloes may be present around lights, most noticeable at night (a common time for presentation) but not always. Flashing lights are often experienced with retinal detachment. The fundus can be normal, but not always, as 'cupping' and pallor of the optic disc can be seen due to loss of neural tissue.

2. Answer: c

Is most likely to be the right answer. In a case where acute glaucoma is highly suspicious the need for a CT scan is outweighed by the need for the patient to be seen by an ophthalmologist and acute management started. Acute angle closure is a medical emergency as it is a cause for loss of sight.

It is very painful on presentation and often accompanied by nausea and vomiting and so morphine and metoclopromide would be effective. Given IV is sensible if the patient is vomiting. Pilocarpine (a miotic) 4% drops every hour will constrict the pupil and acetazolamide (a carbonic anhydrase inhibitor) will decrease secretion of aqueous from the ciliary body, and both are important in management of acute glaucoma.

Drug history is important because acute glaucoma can be caused by some medications (e.g. adrenergic agents (e.g. local such as phenylephrine drops, nasal ephedrine, nebulised salbutamol and systemic such as epinephrine), anticholinergics (e.g. tropicamide and atropine drops), tri- and tetracyclic antidepressants and cholinergic agents (e.g. pilocarpine), and allergies and contraindications (e.g. beta blockers in asthmatics and acetazolamide in sickle cell anaemia) are important in any case as management may be affected.

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Conflicts of interest: None declared.

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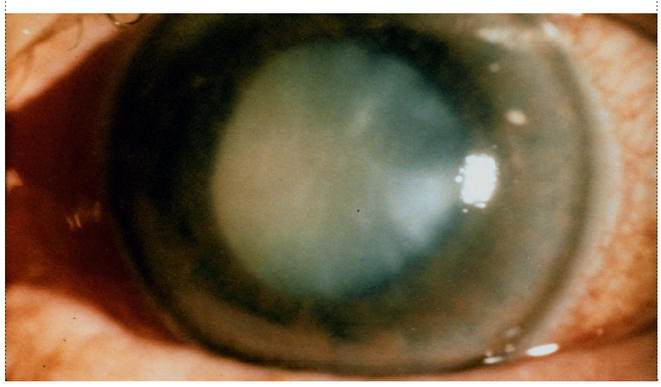
Conflicts of interest: None declared.



AGE RELATED MACULAR DEGENERATION - FOCUS ON WET MACULAR DEGENERATION

S Paneerselvam, N Narendran, M Tahir, Y Yang

Abstract

Age related macular degeneration (ARMD) is a very common cause of visual impairment in the elderly. This case report describes a very typical scenario and highlights important aspects in diagnosis and management of macular disease which junior doctors in training should find useful.

Case History

A 69 year old caucasian female presented to the eye casualty with a one week history of blurred vision in her left eye. She had not noticed any visual symptoms in her right eye. When questioned in more detail, she described the blurring in her left eye as a central patch where objects appeared to be distorted. She was otherwise fit and well but had controlled hypertension and had an older sister with macular degeneration. She was a non- smoker and lived on her own and was coping with daily living activities independently.

On examination her visual acuity was 20/40 in her right eye and 20/80 in her left eye. Amsler Chart testing illustrated a small area of distortion or metamorphosia in the central field of the left eye. Anterior segment examination appeared normal in both the eyes. Posterior segment examination of the right eye revealed yellowish spots in the central macular area, typical of macular drusen, indicating a diagnosis of dry age related macular degeneration. In the left eye there was a haemorrhagic central macular lesion, indicating a diagnosis of wet macular degeneration. (fig1) which was confirmed with Optical coherence tomography (fig2) and fluorescein angiography (fig 3). Treatment with intra-vitreal ranibizumab was urgently arranged for her left eye. She was given a fairly good prognosis due the presentation at an early stage of the disease and relatively good visual acuity at the time of initiating therapy.

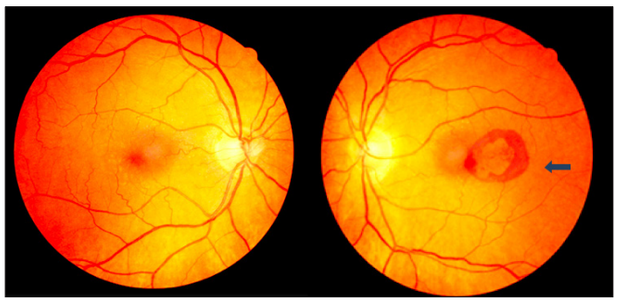


Figure 1: Macula of the left eye showing haemorrhage.

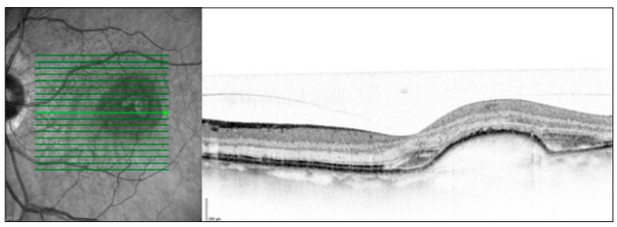


Figure 2: Optical Coherence Tomography of the left eye showing pigment epithelial detachment with subretinal fluid.

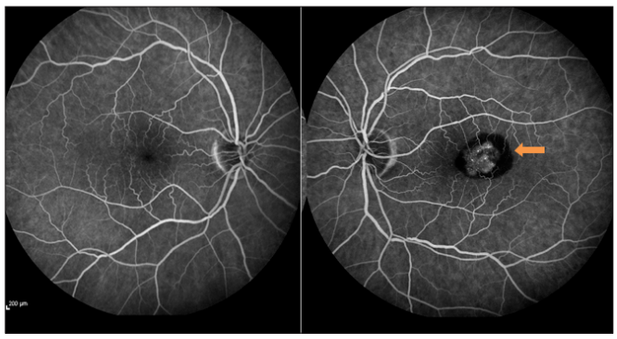


Figure 3: Fluorescein angiogram of left eye showing occult type of CNV.

AGE RELATED MACULAR DEGENERATION - FOCUS ON WET MACULAR DEGENERATION

S Paneerselvam, N Narendran, M Tahir, Y Yang

Discussion

This case report, of the initial management of a patient with wet ARMD, highlights several noteworthy issues for junior doctors working in ophthalmology, the primary care sector or other fields of hospital medicine. Firstly, awareness of the epidemiology and pathophysiology is important as age related macular degeneration is very common in the elderly population. The incidence of sight-threatening wet ARMD occurs in about 5-10 per 1000 individuals over 55 years every year (1). This means that in the course of our daily practice, encountering hundreds of elderly patients, there is a fair likelihood of coming across a patient who is just beginning to develop the early stages of wet ARMD.

Understanding the pathophysiology of a condition helps us to explain the nature of their symptoms to patients and also to understand the mechanisms of the disease process and objectives of various pathways that are targeted therapeutically. For the purposes of a junior doctor encountering their first patient with suspected wet ARMD and having to explain to the patient why their symptoms are making him suspect this diagnosis, a three step mental image of the pathophysiology is very helpful. Firstly, in health the retina is a sensory organ consisting of distal photoreceptors, which are connected to other nerve cells in the retina and the optic nerve which carries fibres to the visual cortex. Just external to the retina is the retina pigment epithelium (RPE), which is a single layer of highly specialized pigmented cells and external to the RPE are the choroidal vessels (fig 4a).

Secondly, in aging, there is slow degeneration of the RPE, which is called dry macular degeneration, diagnosed by the presence of yellow spots in the macular area called drusen or areas of depigmentation due to atrophy of the RPE cells. (fig 4b). This usually causes slow and moderate visual loss only. Thirdly, abnormal angiogenesis or neovascular proliferation can occur from the choroidal vessels causing the vascular tissue to invade inwards towards the RPE and the retina layer (2). This is called choroidal neovascularisation and if not treated, usually grows uncontrollably resulting in oedema, haemorrhage fibrosis and rapid cell death of the retinal photoreceptors and the other retinal cells leading to severe loss of central vision (fig4 c and d).

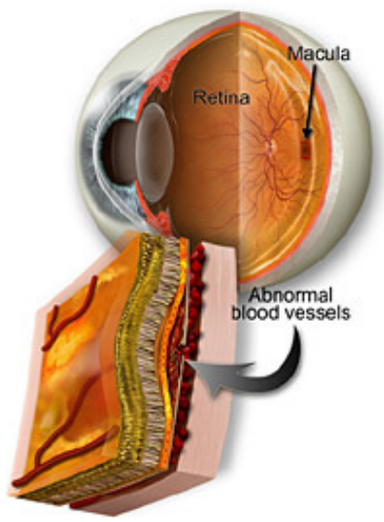


Figure 4: showing (a) normal retina with microscopic anatomy (b) early AMD (c) Choroidal neovascularisation (d) End stage wet ARMD with the formation of disciform scar. (courtesy of Chameleon Medical communications, UK).

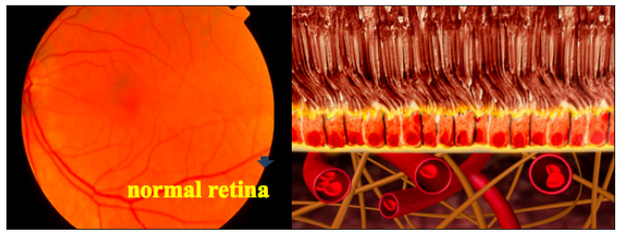


Figure 4 (a)

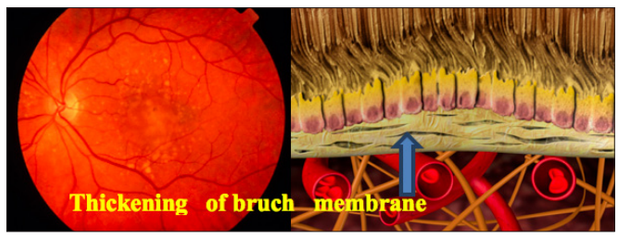


Figure 4 (b)

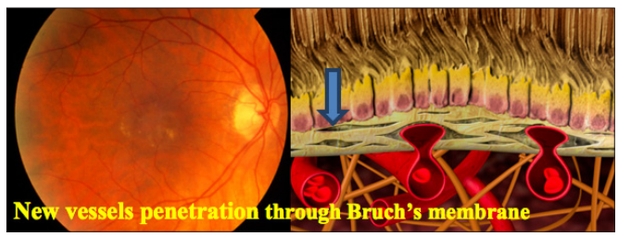


Figure 4 (c)

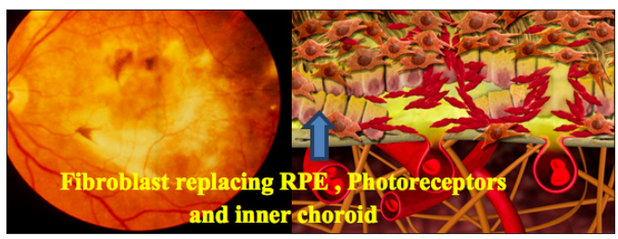


Figure 4 (d)

AGE RELATED MACULAR DEGENERATION - FOCUS ON WET MACULAR DEGENERATION

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Choroidal neovascularisation can occur secondary to a variety of underlying pathological conditions such as ocular trauma or high myopia but by far the most common cause is wet ARMD. Armed with these three mental images of wet AMD and some basic knowledge of epidemiology, our junior doctor when faced with a patient giving the classical history of recent central visual disturbance (scotoma), with distortion of vision (metamorphoposia) (fig 5), can display clinical confidence in explaining to the patient why she is experiencing these symptoms so suddenly and that it is probably at a very early stage.

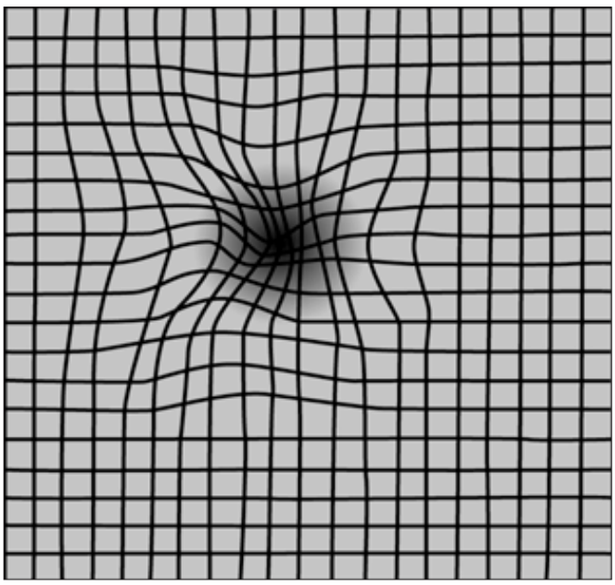


Figure 5: Amslers grid chart showing distortion.

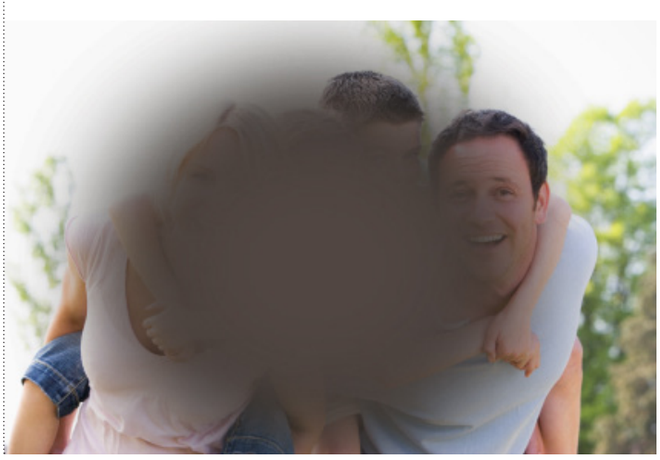
The next noteworthy point was the inclusion in the history of an enquiry about potential risk factors. Although age, family history, Caucasian race, female gender are known risk factors there are modifiable risk factors worth remembering for ARMD which include systemic hypertension, cardiovascular disease, hypercholesterolaemia and smoking status.(3) This patient therefore had several risk factors.

When told about their diagnosis and the visual prognosis, most patients with newly diagnosed wet ARMD ask the question 'Why me?' The most appropriate answer is probably that they have a genetic predisposition to the extreme effects of aging and wear and tear on the retinal tissues, which can be exacerbated by additional factors such as blood pressure and smoking.

Another aspect in the case report was that the diagnosis of wet ARMD was made clinically and then confirmed by retinal imaging. This is often the case in many ophthalmic conditions where diagnosis is based on clinical findings alone. Ophthalmic trainees spend many years examining thousands of patients and become very good at taking and interpreting findings from history and examination and develop an astute clinical acumen in clinical diagnosis. The macular lesion in the patients left eye would have been visualised stereoscopically (3-D) on a slit lamp with a hand held 60-90 dioptre convex biomicroscopy lens by the examining ophthalmologist who would instantly recognise the elevated contour of the lesion, the subretinal location of the blood and the bulge in the subretinal, RPE layer caused by the neovascularisation.

Clear sub retinal fluid would also have been visible clinically. In the right eye, the examiner would have noted the drusen beneath the retina without any elevation, blood, fluid or bulging. These signs would have indicated a clinical diagnosis of wet ARMD in the left eye and dry ARMD in the right. Retinal imaging, using optical coherence tomography, demonstrate the cross section view of the retinal layers, and fluorescein angiography is used to demonstrate the extent of choroidal neovascularisation and its exudative nature as the fluorescent dye escapes thorough the vessel walls into the extravascular space and accumulates as a hyperfluorescent pool of dye on the angiogram.

Lastly, this case report mentions that treatment is available and that preservation of visual function is more likely if treatment is commenced at an early stage before irreversible photoreceptor cell death occurred. In the ophthalmic literature and texts, many clinical trials have been published on treatment of wet ARMD using various agents such as laser, photocoagulation, photodynamic therapy and recently, monoclonal antibodies targeting vascular endothelial growth factors or Anti-VEGF agents. (2,3,4)



AGE RELATED MACULAR DEGENERATION - FOCUS ON WET MACULAR DEGENERATION

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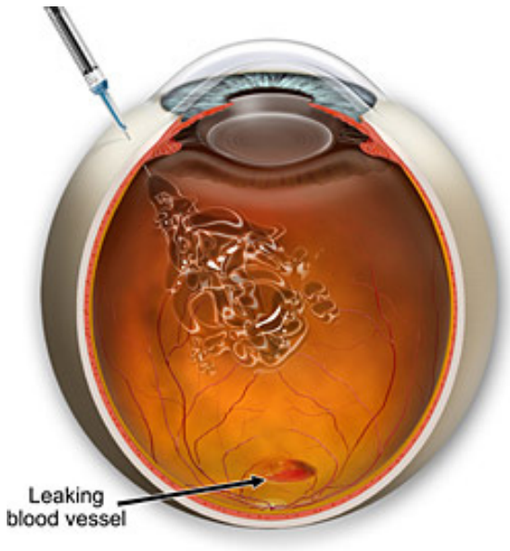
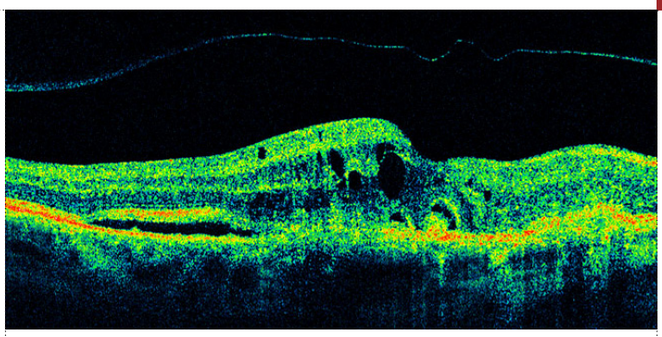
This therapeutic area is as good as example as any in the field of medicine of how systematic basic research and well designed clinical trials have lead to successful implementation of effective therapy. Early trials using thermal laser showed some, albeit small, benefit. In 1997, photodynamic therapy was found to prevent visual loss more effectively than laser or sham therapy. (3,4) The big leap forwards occurred in the past 10 years by shifting from a vaso-occlusive approach using laser and photodynamic therapy to an anti-angiogenesis approach targeting the main drivers of angiogenesis using anti-VEGF agents.(5,6,7) Nowadays, prompt commencement of intravitreal anti-VEGF therapy with ranibizumab or bevacizumab gives at least a 90% chance of visual stability. (8)

In the interest of brevity, our case report ended at a point when the patient was diagnosed and reassured that she would have a reasonably good outcome with therapy as she had presented at an early stage. This would have been a great relief to her as many patients fear the threat of blindness more than other morbidities. Our patient lived on her own at home and good visual function would be important to her for maintaining her own independence at home with daily living tasks such as cooking and reading. (9, 10) This aspect was also importantly noted by the junior doctor taking the social history at the beginning of the clinical evaluation demonstrating a good level of thoroughness.

In summary, this is a case report of an elderly patient with AMD. It is a clinical scenario that every junior doctor should come across directly or indirectly as it is so common in elderly patients. The case report should hopefully provide the opportunity for junior doctors in training to gain knowledge about wet ARMD from a slightly different approach to that provided by standard texts and published journal papers on clinical trial data. Case reporting of a common and typical case can help to illustrate that evidence base practice is not necessarily confined to therapy but to also understanding the wider evidence and using the knowledge base of the pathophysiological, diagnostic and social aspects to evaluate and management patients as fully as possible for their comprehensive needs.





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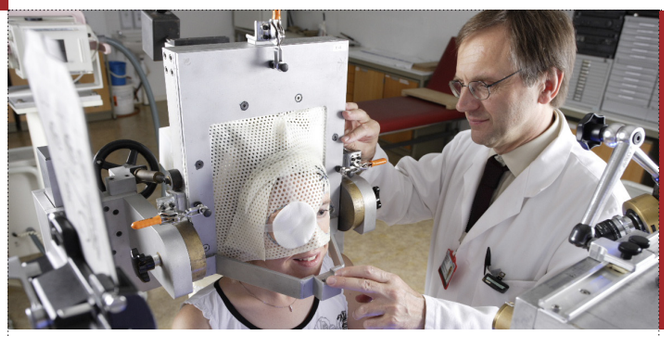
DIFFUSE CHOROIDAL HAEMANGIOMA ASSOCIATED WITH STURGE WEBER SYNDROME TREATED WITH PROTON BEAM THERAPY

G Mc Gowan, D Mc Gowan, E Kemp, P Cauchi, W Kincaid, A Kacperek, D Errington



Diffuse Choroidal Haemangioma associated with Sturge Weber Syndrome treated with Proton Beam Therapy
Patient Management

Abstract

We report a rare case of diffuse choroidal haemangioma associated with Sturge Weber Syndrome in a young girl. The clinical course and treatment of this condition is discussed and diagnostic clues are highlighted.

An eleven year old girl was referred to the Scottish National Ocular Oncology Clinic in Glasgow after a macular disturbance was noted in her right fundus. Her visual acuity in her right eye had reduced from 6/9 to 6/36 over a three month period. She had previously been started on topical treatment for early right open angle glaucoma to reduce her intraocular pressure, which was under control. The vision in the left eye was normal.

Clinical Findings

Examination of her pupils using the the swinging light test revealed a right relative afferent pupillary defect. Slit lamp anterior segment exam showed dilatation of the episcleral vessels. Fundal examination with the use of both direct ophthalmoscope and more detailed slit lamp bioomicroscopy using a superfield lens showed elevation of the retina and pigmentation centred around the macula and optic nerve with obvious inferior lying subretinal fluid.

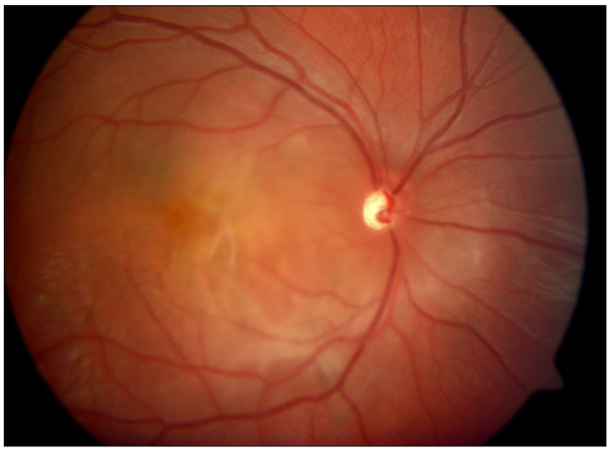


Figure 1a: Fundus image shows subretinal fluid and a classical diffuse orange/red thickening of the choroid- the so called "tomato ketchup fundus".

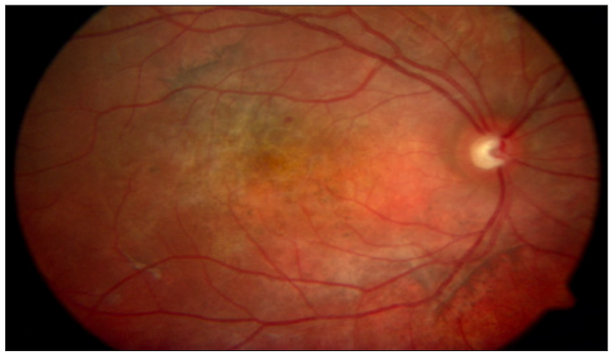


Figure 1b: shows resolution of the subretinal fluid.

This patient underwent further non-invasive investigation to assess the size, extent and density of the fundal lesion including Optical Coherence Tomography and B-Scan Ultrasound, which are both used regularly in Ophthalmic practice alongside detailed clinical examination to assess any fundal abnormality.

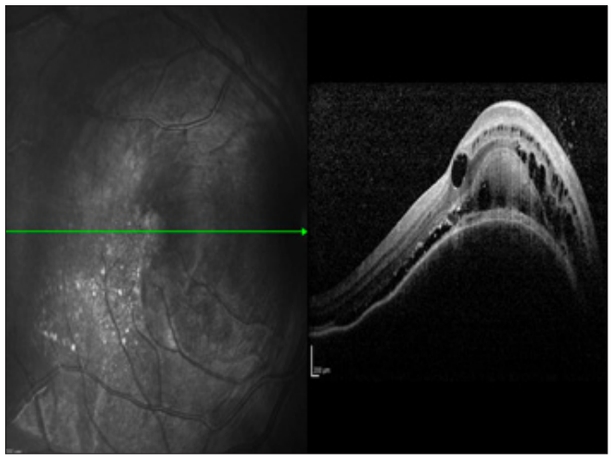


Figure 2a: OCT - Optical Coherence Tomography showed subretinal fluid + intraretinal cysts + impression of choroidal thickening.

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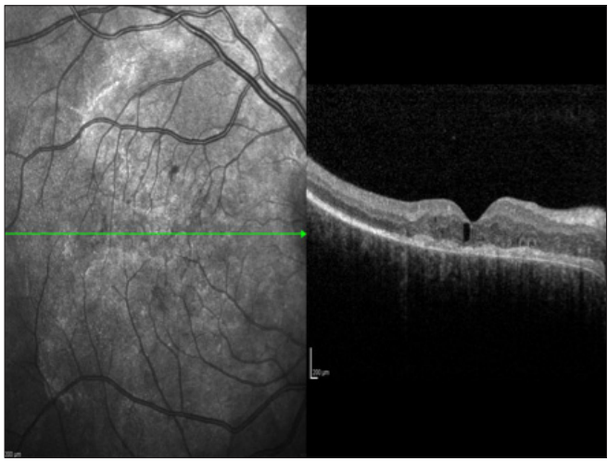


Figure 2b: complete resolution of subretinal fluid with one intraretinal cyst remaining following treatment.

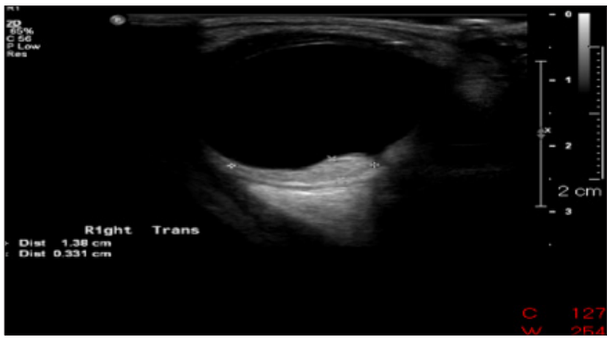


Figure 3a: Ultrasound B-scan showed a hyperfluorescent area of choroidal thickening measuring 1.38mm x 0.33mm.

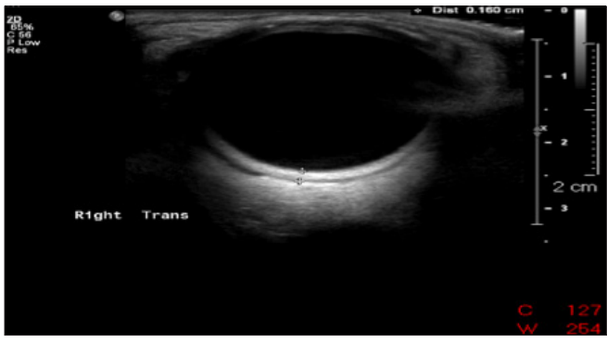


Figure 3b: shows complete resolution following treatment.

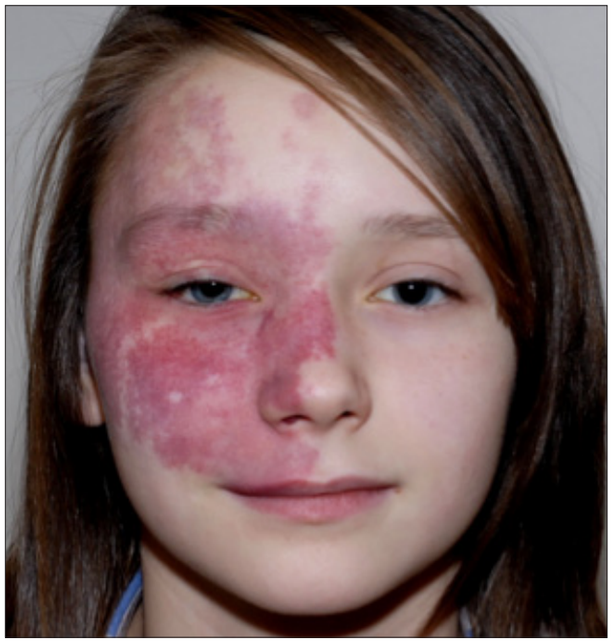
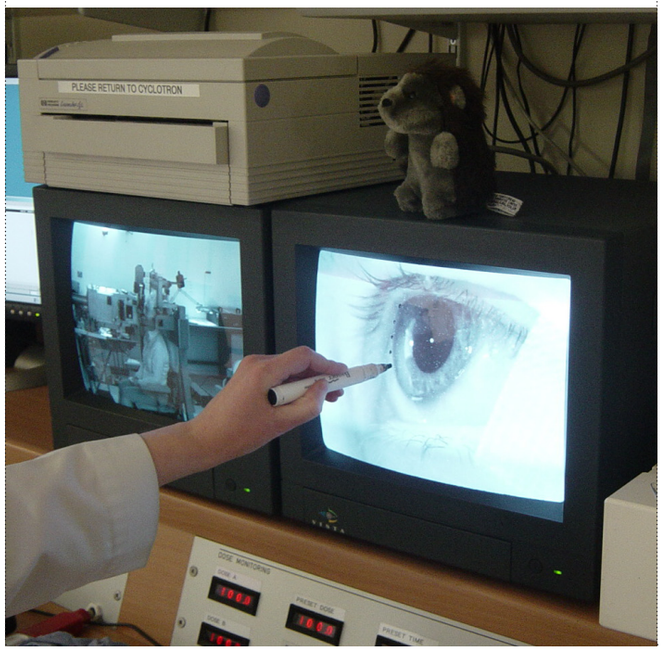


Figure 4: Full facial picture - shows port wine stain (nevus flaemmus).

Diagnosis

She was diagnosed with diffuse choroidal haemangioma secondary to Sturge Weber syndrome. This is a benign vascular tumour. Unlike the circumscribed form, diffuse choroidal haemangioma has poorly defined borders and extends over a broad area of posterior choroid. It is generally located ipsilateral to a facial haemangioma and can be associated with both Sturge Weber and Klippel-Trenaunay-Weber syndrome.



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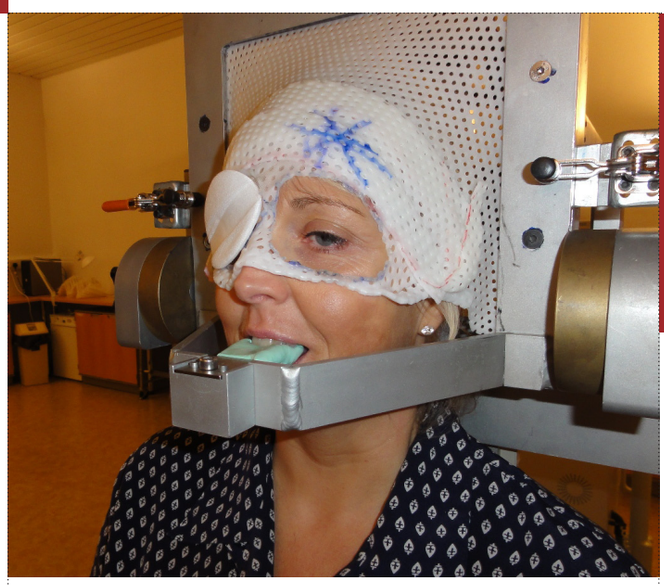

In further contrast to circumscribed variety of choroidal haemangioma, the diffuse type is characteristically associated with cutaneous, ocular or CNS findings. The median age of diagnosis is only eight years old and pathogenesis is poorly understood. Visual prognosis is variable and is dependent on the location and rate of growth of tumour.

Treatment

The main aims of treatment are the preservation of vision and prevention of neovascular glaucoma which can lead to painful blindness.

Treatment options included:

- Observation
- Proton Beam Therapy (1,2,3,4)
- Photo Dynamic Therapy
- External Beam Radiation
- Sclerotomy+cautery+ subretinal fluid drainage+ injection of BSS in to Anterior / posterior chamber+ argon laser photocoagulation
- Pars Plana Vitrectomy +Gas
- Enucleation (4)

Diffuse Choroidal Haemangioma associated with Sturge Weber Syndrome treated with Proton Beam Therapy

Patient Management

We opted to treat this patient with Proton Beam Therapy. She underwent 4 treatments over 4 days. The possible advantages of PBT include reduced radiation dose and targeted treatment to localised area. Proton therapy is a type of external beam radiotherapy which uses ionizing radiation.

A particle accelerator is used to target the tumour with a beam of protons. These charged particles damage the DNA of cells, ultimately causing their death or interfering with their ability to proliferate. Due to their relatively large mass, protons have little lateral side scatter in the tissue; the beam does not broaden much, stays focused on the tumour shape and delivers only low-dose side-effects to surrounding tissue. All protons of a given energy have a certain range of which few penetrate beyond. (4)

Post treatment there was a reduction in tumour size, absorption of subretinal fluid and stabilzation of visual acuity at 2 years. See post-op figures.

Chan RV et al- looked retrospectively at 19 patients with diffuse and circumscribed haemangiomas treated with Proton Beam Therapy x 4 fractions. Minimum follow up of 6 months. They found visual acuity improved or remained stable in 78% of patients.

Subretinal fluid was initially present in 16 of 19 eyes (84%), and completely resolved in all 16 eyes. Tumor height, as measured by B-scan ultrasonography, decreased in 18 of 19 eyes and remained stable in 1 of 19, as of the last examination. Complications of radiation developed in 9 of 19 eyes including retinopathy, eyelid irritation, ocular surface disease.

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Diagnostic clues

What to look for in choroidal haemangioma:

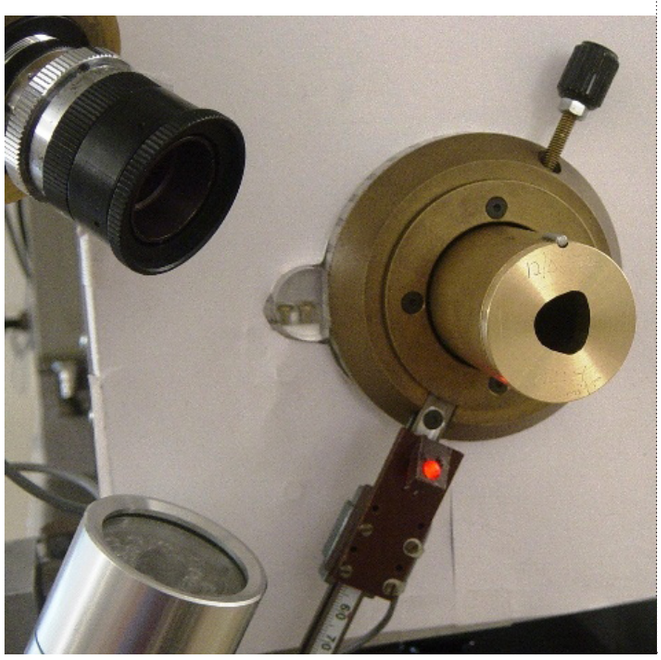
- Ipsilateral visual loss
- Hyperopic amblyopia
- Tomato ketchup fundus (brilliant red reflex)
- Dilated tortuous vessels
- Tumour may be thickest in macula area
- Cystoid degeneration of overlying retina + RPE disruption
- Severe cases- total retinal detachment, secondary cataract, leukocoria (white pupillary reflex)
- A-Scan: high internal reflectivity
- B-Scan: markedly thickened choroid-acoustic solidity

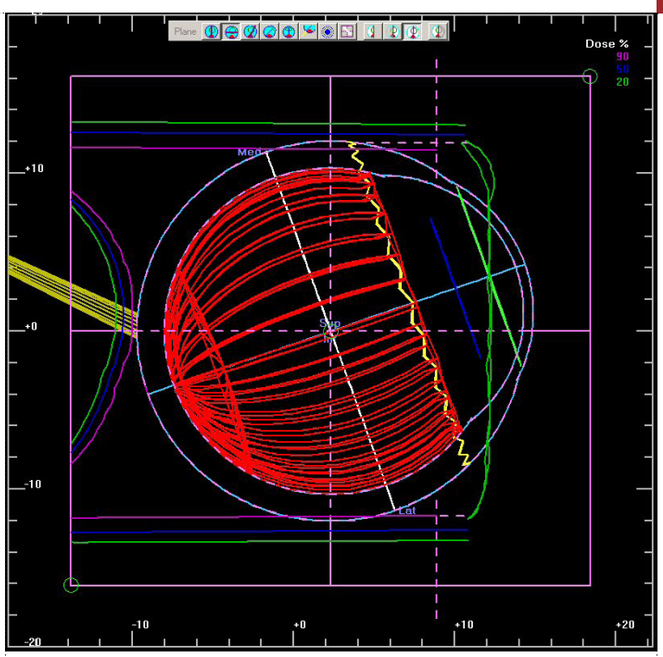
Sturge-Weber syndrome - Encephalofacial haemangiomatosis - what to look for:

- Consists of facial haemangioma, buphthalmos, seizures and radiographic evidence of intracranial calcification (haemangiotosis)
- No hereditary pattern/predisposition for race/sex
- Cutaneous distribution of 5th cranial nerve
- Ocular manifestations

Conclusion

This is a very rare condition with minimal data in the literature on diagnosis and treatment. Referral to Clatterbridge, Liverpool for Proton Beam Therapy is the treatment of choice in the National Ocular Oncology Centre in Glasgow, Scotland, as PBT has the ability to precisely localise radiation, and potentially limit collateral damage.





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FITNESS TO DRIVE

A Silvester, M Batterbury

Abstract

The Driving Vehicle Licensing Agency (DVLA) is an Agency of the Department for Transport and has a responsibility to ensure that all license holders are fit to drive. The DVLA set minimum standards for vision, a driver must have good central vision and adequate peripheral vision, if not met the driver must not drive and the license must be refused or revoked. This article summarizes the DVLA vision requirements, provides practical advice and discusses ethical considerations.

Case history

Mrs A is a 53 year old lady with primary open angle glaucoma. A visual field test showed a significant defect sufficient to disqualify her from driving. Working in a car garage required her to drive on a daily basis and when in attendance at clinic she stated that she would continue to drive.

Legal requirements

The Driving Vehicle Licensing Agency (DVLA) is an Agency of the Department for Transport and has a responsibility to ensure that all licence holders are fit to drive. Section 92 of the Road Traffic Act 1988 defines a relevant disability as any medical condition that is likely to render the person a source of danger while driving.

Thus, a driver must have good central vision and adequate peripheral vision. The DVLA set minimum standards for vision (see table below). If not met the driver must not drive and the license must be refused or revoked.

Fitness to drive
Good Clinical Care

Practical points

	Group 1 Motor cars, motor cycles	Group 2 Large lorries and buses
Visual acuity	Read a registration plate in good light (with glasses or contact lenses if required) at 20 metres.	Barred in law from holding licence if vision in better eye worse than 6/9 or 6/12 in the other eye.
Visual field	A field of at least 120 degrees on the horizontal, no significant defect in binocular field which encroaches 20 degrees of fixation above or below the horizontal meridian.	Normal binocular field required.
Monocular	Must notify DVLA. Can drive when adapted to disability. Remaining eye must satisfy visual acuity above and have normal visual field.	Barred in law from holding licence if loss of vision in one eye.
Diplopia	Cannot drive until diplopia controlled by glasses or a patch. Stable uncorrected diplopia > 6 months may be able to drive if satisfactory functional adaptation (supported by consultant).	Permanent refusal or revocation if insuperable diplopia. Patching is not acceptable.

Adapted from At a glance Guide to the current Medical Standards of Fitness to Drive, DVLA, December 2011.

It is the duty of the driver to inform the DVLA of their medical condition. Once informed, the DVLA will write to the ophthalmologist for a medical report and may arrange for a visual field test to be performed. Visual fields are performed on an Esterman perimeter with both eyes open and with glasses or contact lenses (if required). The result is reviewed by the DVLA medical advisor and a decision made. The driver has a statutory right to appeal to a magistrates court, but should be advised to seek medical and legal advice before doing so since such an appeal can be costly. Driving without a valid licence invalidates car insurance and is a serious offence.

FITNESS TO DRIVE

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Ethical considerations

Patient confidentiality is overridden by the duty to protect individuals or society from risks of serious harm.

The General Medical Council has issued detailed advice on reporting patients to the DVLA.

1) Seek the advice of an experienced colleague or the DVLA medical adviser if you are unsure about a patient's fitness to drive.

2) Explain to the patient that the condition may affect their ability to drive and that they have a legal duty to inform the DVLA

3) If a patient refuses to accept the diagnosis or effect of the condition on their ability to drive, suggest they seek a second opinion and advise them not to drive in the meantime.

4) If a patient continues to drive, make every reasonable effort to persuade them to stop

5) If you do not manage to persuade the patient to stop driving, or discover they are continuing to drive against your advice, you should contact the DVLA immediately and disclose any relevant medical information to the medical advisor.

6) Before contacting the DVLA, try to inform the patient of your decision to disclose personal information and inform them in writing once you have done so.

Multiple choice questions.

1) Which of the following may still be fit to drive?

a) a bus driver with monocular vision
b) a lorry driver with a small visual field defect
c) a motorcyclist with monocular vision
d) a motor car driver with new onset diplopia

2) What would be the most appropriate action to take if you became aware that a patient was continuing to drive against your medical advice?

a) make a citizen's arrest
b) disclose medical information to the DVLA
c) do not get involved
d) phone the police

Answers

1) C

A motorcyclist with monocular vision may still be able to drive if the other eye satisfies visual acuity requirements, has no field defect and they have adapted to the disability. Class 2 licence holders e.g. lorry and bus drivers cannot drive with monocular vision, any field defect or insuperable diplopia (patching is not acceptable). Motor car drivers with new onset diplopia must refrain from driving until corrected with glasses or a patch. Those with a longstanding diplopia (>6 months) who show functional adaptation may still be able to drive a car.

2) B

The most appropriate action would be to disclose medical information to the DVLA. It is good medical practice to try to inform the patient that you intend to contact the DVLA and inform the patient in writing when you have done so.

Further information

1. GMC guidance on DVLA. http://www.gmc-uk.org/Confidentiality_reporting_concerns_DVLA_2009.pdf_27494214.pdf
2. DVLA current medical standards of fitness to drive. http://www.dft.gov.uk/dvla/medical/ataglance.aspx

Authors

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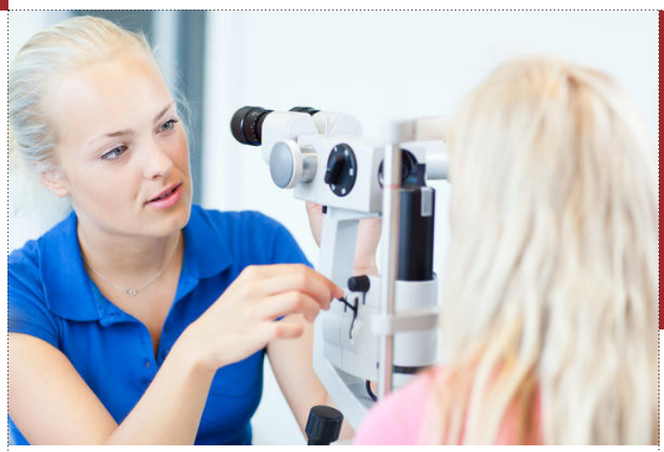
AN ETHICAL DILEMMA IN OPHTHALMOLOGY UNDERSTANDING THE PATIENT'S AGENDA

GS Williams, M Radwan, R Ram



Abstract

We present the case of a young woman presenting with retrobulbar neuritis in which the disease course becomes increasingly bizarre until it is realised that the correct diagnosis is that of malingering.

We discuss the pitfalls in diagnosing and treating retrobulbar neuritis generally, as well as the medico-legal minefield of non-organic visual loss. Some tests for diagnosing non-organic visual loss are discussed as well as the importance of remaining professionally detached in these and other similar cases in which the patient's actions can have profound effects on our emotions.

Keywords: Medical, Ethics, Malingering, Simulated visual loss

Case History

A 22 year old single mother of two young children presented to our eye casualty department with a three day history of progressive vision loss in her right eye, accompanied by pain on eye movement and a right sided headache.

At the time of this initial visit her visual acuity was measured at counting fingers in her affected eye, being an excellent 6/4 in the healthy eye. She had no past medical history of any note other than what was described as 'mild depression' for which she was taking citalopram. She was taking no other medications, had no known allergies and no family history of any note.

The examination of both right and left anterior and posterior segments of the eyes were unremarkable, with normal eye movements and, crucially, normal looking optic discs and no obvious relative afferent pupillary defect (RAPD). Ishihara testing resulted in a score of 17/17 on the left side with no plates being seen at all on the abnormal right side.

An Ethical Dilemma in Ophthalmology Understanding the Patient's Agenda
Patient Management

Based mainly on the negative physical findings a diagnosis of right sided retrobulbar neuritis was made and appropriate investigations initiated. These consisted of an array of blood tests; including ESR, CRP, FBC, thyroid function tests, anti-nuclear antibodies and ACE to rule out other less likely pathologies, and the ordering of an MRI scan to confirm a retrobulbar inflammatory element and assess the brain for other potential areas of demyelination that could point towards multiple sclerosis.

While the initial blood tests, including inflammatory markers, were normal, the patient re-presented to the eye casualty four days later with a one day history of exactly the same symptoms in her previously unaffected left eye. Examination was, as before, entirely normal, and a tentative diagnosis of bilateral retrobulbar neuritis was made.

The patient was admitted for pulsed intravenous steroid therapy with 500mg methylprednisolone and for urgent further investigations. These consisted of further blood tests and to expedite the MRI scan.

The following morning, having received only one of the planned three steroid infusions, the patient reported dramatically improved vision in her right eye. The visual acuity in this eye now measured a highly unexpected 6/5 while the left eye remained at the counting fingers level with the physical examination continuing to demonstrate complete normality in both eyes.

This sudden changing of the clinical picture aroused suspicion that some form of medically unexplained visual loss was responsible and after further testing a diagnosis of malingering was made. The patient had all further steroid injections suspended and was discharged the following day having completely recovered her vision in both eyes.

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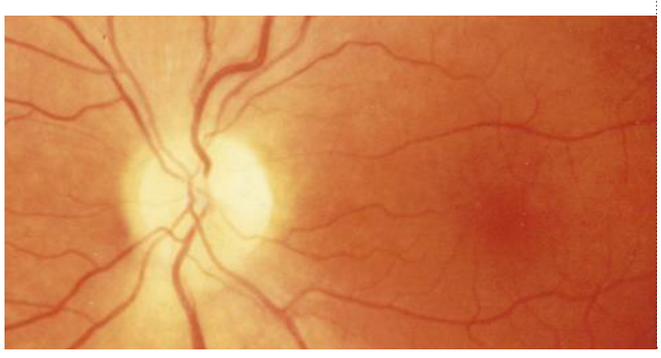
Discussion

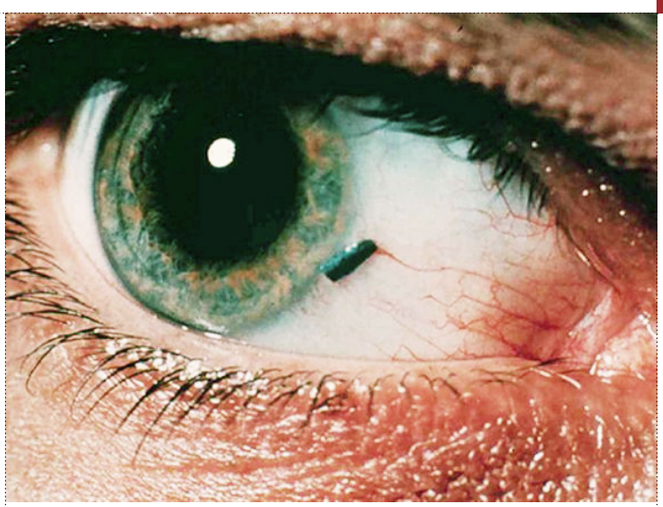
A certain amount of faith is always needed when making a diagnosis of retrobulbar neuritis for the simple reason that for it to be retrobulbar the optic disc by definition has to look normal. Often the only objective finding is an RAPD in the affected eye. When this patient initially presented to our department a diagnosis of retrobulbar neuritis was made despite the absence of an obvious RAPD as the clinical picture fitted so well with this condition and when she presented the second time with bilateral involvement the value of comparing the pupillary reaction of one eye against the other had been lost as both eyes were supposedly affected. An important take home message at this point is to make full use of all of your examination findings and not to ignore those that inconveniently contradict your working diagnosis. Perhaps it is not the finding that is wrong, it is the diagnosis.

Whereas bilateral retrobulbar neuritis is much rarer than unilateral retrobulbar neuritis it is still more likely than the main (organic) differential diagnoses. These consist mainly of Devic's disease, a form of acute disseminated encephalomyelitis; Leber's hereditary optic neuropathy, which would have been even more unlikely in a female patient by some degree of magnitude; an ischaemic optic neuropathy, syphilis, Lyme disease and thyroid eye disease. All the blood tests that had been processed prior to the eventual correct diagnosis being made had pointed away from these other differentials.

The treatment of optic neuritis in general is another important consideration. The Optic Neuritis Treatment Trial was a well performed randomised trial that showed no benefit in ultimate visual outcome following steroid treatment, although the speed of recovery was improved somewhat. For that reason the tendency is to monitor unilateral optic neuritis while bilateral disease is considered for steroid treatment, depending on the situation. Similarly, steroid treatment is also considered in patients presenting with unilateral disease if the unaffected eye has some other visual problem severely limiting its visual acuity.

Ultimately it is only when the sudden and unexpected improvement occurred in the patient's right eye were suspicions aroused that there might be a non-organic element. Medically unexplained visual loss can be divided into four main categories; undiagnosed organic disease, functional visual loss, malingering and Munchausen's syndrome. Functional visual loss describes a condition whereby the patient unconsciously loses the ability to see properly whereas what distinguishes malingering from Munchausen's syndrome can be distilled down to the notion of secondary gain.





A conversation with the patient's general practitioner undertaken shortly after the patient's disease was recognised to be non-organic revealed an increasing inability of this single mother to cope with two young children that had manifested itself in the recent past with other conditions, undisclosed by the patient, that had resulted in her parents having to look after her children for short periods of time.

The secondary gain in this instance therefore was postulated to be a rest period in the hospital away from the demands of everyday life. Indeed in retrospect the patient did seem to accept being admitted to hospital with very good grace considering her social situation and was also generally unperturbed when returning for the second time with bilateral severe loss of vision.

Diagnosing non-organic severe vision loss in patients is in fact surprisingly easy, though one needs to suspect the condition first. If purportedly affecting a single eye a selection of tests can be used to confirm the diagnosis. The optokinetic reflex (OKN) drum (Figure 1) can detect whether a supposedly non-seeing eye can see as the induced optokinetic nystagmus, produced by turning the drum at approximately 8-10 rpm at 24-30 inches away from the patient's eye, is completely immune to being voluntarily suppressed. In supposed single eye involvement the 'good' eye needs to be occluded before commencing the test.

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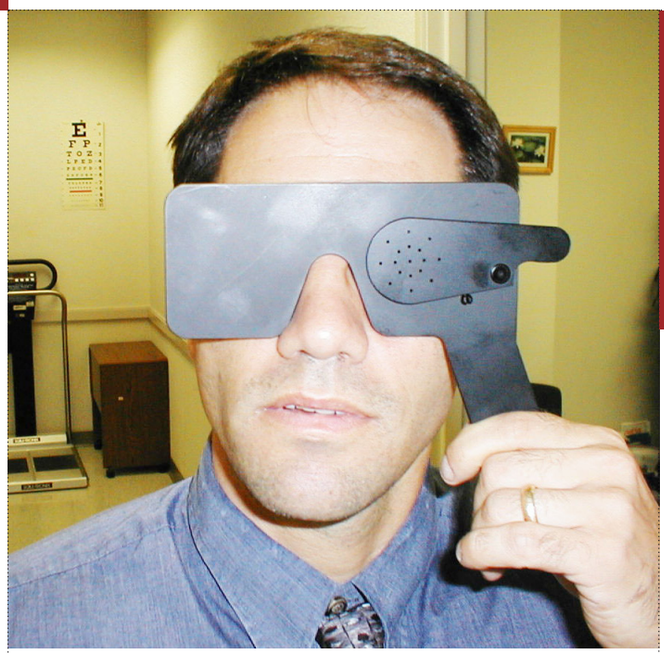

The same applies to the Schmidt-Rimpler test, where a patient is asked to look at their own hand held up before them, a task which should be easily accomplished by anybody with an intact sense of proprioception. Duane's method involves placing a vertical prism in front of one eye and asking the patient to read. In a patient able to see with both eyes reading will be extremely difficult due to the vertical diplopia induced by the prism.

This patient was caught out by the pinhole test. In this case a pinhole is placed in front of the 'good' eye and the patient then asked to read the Snellen chart; the pinhole is then either surreptitiously covered or significant blinding induced by placing a very strong lens in front of it ('you should see very well now with this nice strong lens') and the patient asked to read again unaware that they are using the supposedly diseased eye alone. There are a myriad of other tests that it is also possible to perform, but by far the most useful thing of all is simply observing a patient moving around a room and judging whether their speed and navigational sense is in keeping with their supposed visual acuity.

An Ethical Dilemma in Ophthalmology
Understanding the Patient's Agenda
Patient Management

Figure 1: OKN Drum.

The famous American sociologist Talcott Parsons first described what came to be called the 'sick role' in 1951, describing the rights and obligations of a patient in both the doctor-patient relationship and in society in general. Doctors do not expect their patients to purposefully mislead them and thus malingering is probably a massively underdiagnosed condition seen in clinics and hospitals across the land.

Doctors are also human and although an end diagnosis of malingering can arouse feelings of anger against the patient it is of the utmost importance to remain professional at all times and not to allow this transference to affect the doctor patient relationship. Indeed malingering is by itself a medico-legal minefield and one would do well to avoid becoming embroiled in a worsening situation due to unprofessional behaviour.

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It is recommended not to directly confront the patient about the diagnosis once it is made with certainty but to allow the patient to retreat with dignity. This patient was told the examination findings were all normal and the next day the vision would be back to normal again. When this was indeed the case it was emphasised that is was an extremely unusual clinical picture and not in keeping with any known disease, in order not to facilitate repeat offending, and the patient was discharged without incident. Three months have since elapsed and the patient remains well at home, a testament that contrary to the increasing importance of evidence based medicine the work of a doctor is still as much an art as it is a science.

MCQ's

1) For the best ultimate visual outcome optic neuritis is best treated with:

a) Oral steroids

b) Intravenous steroids

c) Both oral and intravenous steroids

d) No medication

e) Methotrexate

2) Which one of the following is not an appropriate test for assessing non-organic visual loss if proprioceptive dysfunction is also present:

a) Schmidt-Rimpler test

b) Pupillary reflex

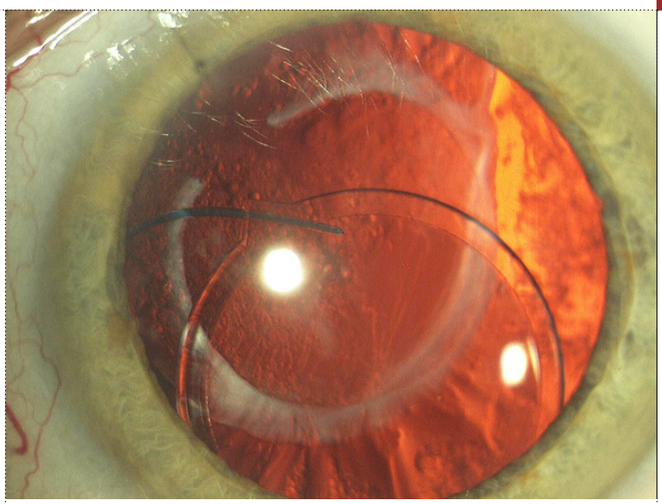
c) Stereopsis

d) Visual field test

e) Pin hole test

Answers

1. Answer – d)
The optic neuritis treatment trial, a well performed randomised trial assessing the use of steroids on the ultimate visual outcome in optic neuritis, did not show any benefit in their use at the five year mark. The use of methotrexate in this condition has not been assessed.



2. Answer – a)
The Schmidt-Rimpler test involves asking a patient to look at their own hand, held up in front of them. Those with normal proprioception should be able to do this easily with their eyes closed although patients feigning visual loss tend to purposefully look away from their hand when asked to look directly at it. The pupillary reflex should be normal and easily demonstrated and although both stereopsis and visual field test would also be expected to be normal quite bizarre results can be achieved when the patient tries to second-guess what the assessor is expecting to find. The pin hole test is used to diagnose those with uniocular simulated loss of vision.

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INTRA-VITREAL INJECTIONS

A Silvester, M Batterbury

Abstract

Intra-vitreal injections can play a huge role in the management of important ophthalmological conditions. It has become a popular method to manage retinal diseases such as age-related macular degeneration (AMD), diabetic retinopathy and retinal vein occlusion. The development anti-VEGF agents has made a significant impact the management of AMD and other conditions associated with pathological neovascularisation.

Newer Intra-vitreal therapies are emerging, and this includes the dexamethasone Intra-vitreal implants used for the management of macular oedema and posterior uveitis. Awareness of the indications, contraindications, signs and symptoms of adverse effects and potential complications of Intra-vitreal therapies is important for the junior doctor. This review article aims to explain the background pathophysiology and mechanism of action of current Intra-vitreal therapies, the steps involved in Intra-vitreal injections, the potential side effects and complications of the procedure.

Clinical Scenario

A 65 year old Caucasian lady presented with a 2 hour history of sudden loss of vision in her left eye. She has a history of type 2 diabetes and is an ex-smoker.

What questions would you include in your history?

- *Is it a monocular or binocular loss of vision?*
- *Is the loss of vision transient, persistent or progressive?*
- *The severity of the visual loss*
- *Associated symptoms such as pain*
- *Any history of flashes or floaters?*
- *Any trauma?*
- *Any headache?*
- *Previous ophthalmic history*

What examinations would you perform on this patient?

- *Visual acuity*
- *Confrontation visual field testing*
- *Pupillary reactions*
- *Fundoscopy or slit-lamp examination*
- *Tonometry*
- *General physical examination*

Examination of the patient revealed a severe reduction in visual acuity of the right eye to finger counting with a marked afferent pupillary defect. Slit-lamp examination of the right eye revealed widespread retinal haemorrhages with dilated tortuous veins (Figure 1). Examination of the left eye showed the presence of hard exudates with micro-aneurysms and engorged tortuous veins with scattered flame-shaped haemorrhages.

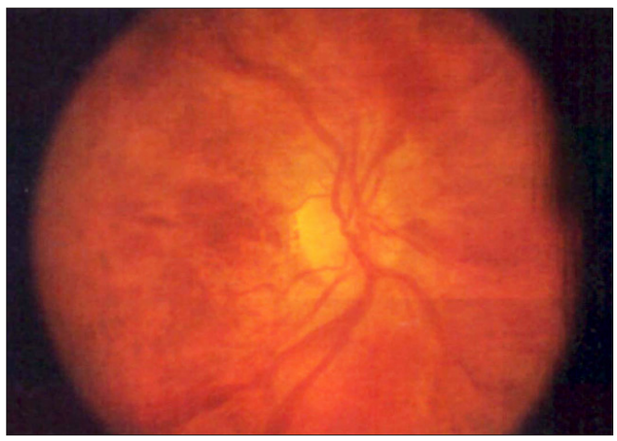

Figure 1: Widespread retinal haemorrhages and dilated, tortuous veins in central retinal vein occlusion. Image reprinted with permission from Medscape.com, 2012. Available at: http://emedicine.medscape.com/article/1223746-overview.

INTRA-VITREAL INJECTIONS

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This lady had an ischaemic central retinal vein occlusion (CRVO) of the right eye causing an acute painless loss of vision. Her left eye showed features consistent with non-proliferative diabetic retinopathy.

How would you manage this patient?

Treatment for CRVO is currently inadequate and much of the management involves identifying and treating systemic medical problems to reduce further complications (1). Some treatment options include the following (1):

- *Laser-induced chorioretinal venous anastomosis*
- *Infusion of tissue plasminogen activator (t-PA) into the vein*
- *Optic nerve sheathotomy*
- *Intra-vitreal injection of ranibizumab*
- *Intra-vitreal injection of triamcinolone*
- *Intra-vitreal injection of bevacizumab*
- *Dexamethasone Intra-vitreal implant*
- *Pan-retinal laser photocoagulation*
- *Radial optic neurotomy*
- *Vitrectomy*

The patient attended a follow-up clinic 6 months later. Examination of her right eye revealed signs of retinal neovascularisation. Fluorescent angiography was carried out which revealed evidence of macular oedema (Figure 2). A decision was then made to give the patient a dexamethasone Intra-vitreal implant (OZURDEX, Allergen). Before obtaining consent, the patient was given an explanation of the procedure and the potential adverse reactions. The risk associated with Intra-vitreal injections includes raised intraocular pressure, vitreous haemorrhage, retinal tear, retinal detachment or endophthalmitis. The patient should also be informed of probable repeated injections in the future and the frequency at which these will be required and for how long. The procedure is carried out under controlled aseptic conditions in theatre or in a dedicated room in outpatients.

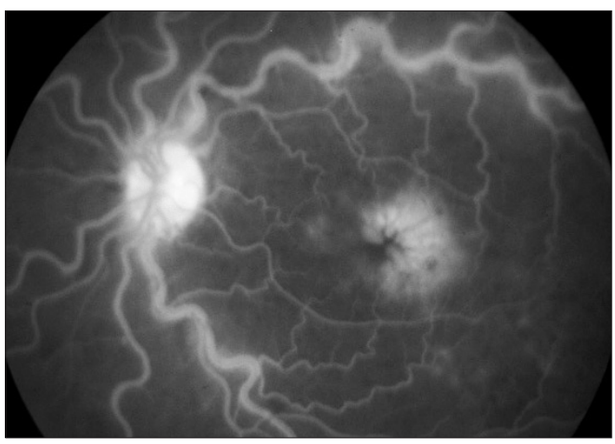

Figure 2: Fluorescent angiogram showing perifoveal leakage in a cystoid pattern in the late phases of angiogram. Image reprinted with permission from Medscape.com, 2012. Available at: http://emedicine.medscape.com/article/1223746-overview

Before the procedure, it is important to make sure that the patient is assessed for any risk of infection and also for contraindications. The following questions may be included in your 'risk assessment' (2):

- *Is there active blepharitis (a contraindication to Intra-vitreal injection)?*
- *Is the patient able to cooperate during the procedure and not interfere with sterile technique?*
- *Will the patient be able to use or receive antibiotic drops during the post-injection period?*
- *Will the patient be able to recognize symptoms of endophthalmitis should they develop?*
- *Is the patient likely to return for follow-up appointments?*
- *Has the patient's intraocular pressure been measured and recorded prior to the procedure?*

Summary of the procedure[3]

• Check pupillary dilation
• Apply topical anaesthetic
• Instill 5% povidone iodide (or chlorhexidine) on to the ocular surface
• Clean the periocular skin, eyelid margins and eye lashes with 5-10% povidone iodine
• Dry the skin and apply the drape
• Insert eyelid speculum
• Instruct the patient to direct gaze away from the site of injection
• Mark the scleral injection site using the mm gauge (the entry site of the needle should be 3.0-3.5 mm from the limbus in aphakic/pseudophakic patients, and 3.5-4.0 mm in phakic patients). Avoid the horizontal meridians of the globe; although the infero-temporal quadrant is often used, any quadrant can be used and may be changed in rotation.
• Using forceps to steady the eye (if necessary), the needle is inserted perpendicular through sclera with the tip aimed towards the centre of the globe (to avoid any contact with the posterior lens).
• Inject appropriate volume (maximum 0.1 ml) of therapeutic agent slowly and carefully
• Remove needle carefully
• Discard syringe and needle appropriately
• Apply 1-2 drops of single use antibiotic
• Check that the patient is able to see objects immediately after injection

INTRA-VITREAL INJECTIONS

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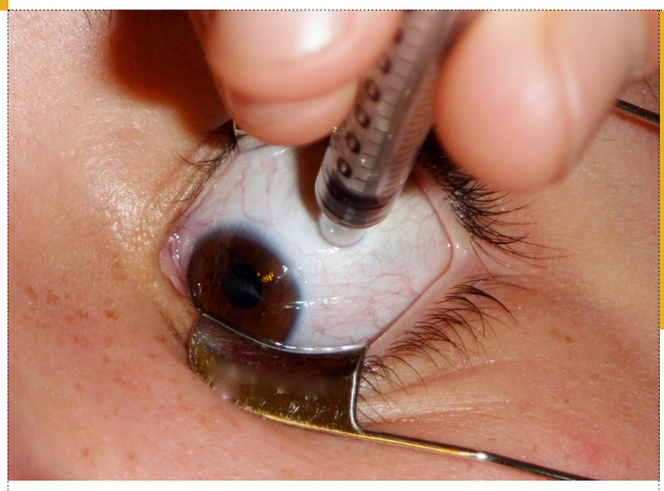


This case reflects the use of Intra-vitreal therapies in ophthalmological conditions. Intra-vitreal drug delivery has become a popular method to manage retinal diseases such as age-related macular degeneration (AMD), diabetic retinopathy and retinal vein occlusion. The efficacy and the relative ease of performing the procedure make the procedure both appealing and practical for the ophthalmic surgeon and patient. Intra-vitreal injections are usually performed by an ophthalmic surgeon or by a trainee under supervision.

In this particular scenario, the patient received a dexamethasone Intra-vitreal implant which has been approved by NICE in 2011 for the treatment of macular oedema following central or branch retinal vein occlusion(4). The dexamethasone Intra-vitreal implant is a biodegradable implant that contains a potent steroid which suppresses inflammation by inhibiting capillary leakage, oedema, fibrin deposition and phagocytic migration(5).

It also suppresses angiogenesis by decreasing the synthesis of vascular endothelial growth factor(5). According to NICE, the dexamethasone Intra-vitreal implant is also used for macular oedema secondary to branch retinal vein occlusion only when treatment with laser photocoagulation has not been beneficial or is not considered suitable due to the extent of macular haemorrhage(4).

The dexamethasone Intra-vitreal implant may also be used for the treatment of non-infectious uveitis affecting the posterior segment of the eye(5). Injections are given every 6 months and common adverse reactions are increased intraocular pressure, conjunctival haemorrhage, vitreous detachment and haemorrhage, posterior subcapsular cataracts and endophthalmitis(4).

Intra-vitreal Injections Good Clinical Care=

Other Intra-vitreal therapies include the anti-vascular endothelial growth factor (anti-VEGF) injections, which are becoming increasingly important for the treatment of exudative AMD, macular oedema due to background diabetic retinopathy and retinal vein occlusion, and also for other conditions associated with pathological neovascularisation like retinopathy of prematurity(6).

Angiogenesis is a complex process involving many growth factors and several biochemical processes. VEGF is one of the important growth factors that plays a key role in physiological and pathological angiogenesis.. Decades of research into the role of angiogenic factors has identified VEGF (initially termed vascular permeability factor) as a critical rate-limiting molecule in angiogenesis, making it a target for the development of anti-angiogenic drugs(6).

Tissue hypoxia due to vascular occlusion or anaerobic tumour metabolism is the main stimulus for VEGF synthesis. In the eye, VEGF is produced by retinal pigment epithelial cells, neurons, glial cells, endothelial cells, ganglion cells, Muller cells and smooth muscle cells(6).

VEGF targets the vascular endothelial cells to undergo mitosis and stimulates vasodilatation, as well as activating matrix metalloproteinases to increase vascular permeability, leading to a breakdown of the blood-retinal barrier and capillary leakage into the intercellular matrix(6).

Pathological neovascularisation can lead to chorioretinal diseases such as wet AMD and diabetic retinopathy, which are the leading causes of blindness in industrialised nations. Knowledge of the underlying physiological mechanisms of angiogenesis and research into the detailed biochemistry of VEGF has led to the development of intra-ocular anti-VEGF injections such as pegaptanib, bevacizumab and ranibizumab.

INTRA-VITREAL INJECTIONS

A Silvester, M Batterbury

AMD remains one of the most common causes of blindness in the above-60 age group and is the main driver for the development of ocular anti-VEGF drugs. Exudative or 'wet' AMD accounts for only 10% of patients with AMD and can cause severe loss of vision due to focal ischaemia and inflammation of the outer retina, which induces neovascularisation from the choriocapillaris into the subretinal pigment epithelium or sub-photoreceptor space(6).

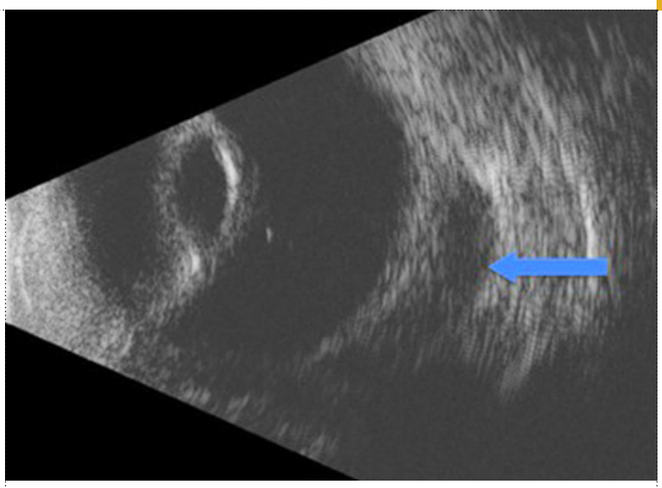
Pegaptanib was the first anti-VEGF drug developed for intra-vitreal injections in 2004(6). Research has shown that when compared with sham injections, intra-vitreal injections of pegaptanib every 6 weeks led to a significant decrease in the average 1-year vision loss due to AMD (loss of 7 letters vs. loss of 15 letters, respectively) (6). However, the use of pegaptanib has decreased substantially with the development of bevacizumab and ranibizumab.

Bevacizumab is a full-length murine-derived antibody which targets VEGF binding sites and was initially approved for the treatment of advanced adenocarcinoma of the colon in 2004(6). Bevacizumab was not originally considered appropriate for intra-vitreal use, despite its widespread off-label use amongst retinal surgeons, as there were concerns over adverse reactions when injecting full-length antibodies(6).

Due to these concerns, bevacizumab was cleaved into smaller Fab fragments with enhanced affinity for VEGF binding sites, hence forming ranibizumab. In addition to these drugs, a new anti-VEGF agent derived from an engineered protein called aflibercept is currently moving through clinical trials for intra-ocular use and has shown to have even greater affinity for binding on to VEGF receptors compared to ranibizumab and will likely receive approval soon for intra-ocular injections (6).

In diabetic retinopathy, increased VEGF synthesis is associated with the formation of free radicals and oxidative stresses due to prolonged hyperglycaemia, leading to a VEGF-mediated breakdown of the blood-retinal barrier. Furthermore, vascular ischaemia in diabetic retinopathy increases the production of VEGF, leading to neovascularisation (proliferative diabetic retinopathy) and further complications such as traction retinal detachment and vitreous haemorrhage.

Complications such as diabetic macular oedema (DMO) may also occur due to the diffusion of serum proteins into the interstitial space which impairs visual acuity (6). Although laser photocoagulation is commonly used for DMO and proliferative retinopathy, randomized controlled trials has shown that the use of Intra-vitreal ranibizumab for DMO has produced more letters of visual improvement than laser photocoagulation alone(6). Similar conclusions were also found when comparing the use of bevacizumab injections with laser photocoagulation(6).



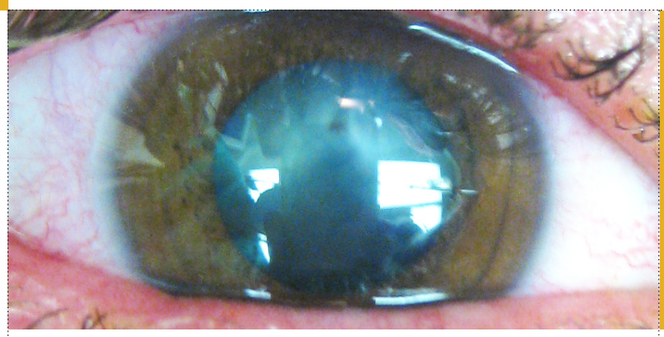
Other conditions that can be managed by Intra-vitreal anti-VEGF agents includes retinopathy of prematuritiy (bevacizumab has been shown to be superior to laser photoablation (6), juxtafoveal telangiectasia and radiation retinopathy(6). Anti-VEGF injections can also be used for the management of trabeculectomy bleb failure and neovascular glaucoma following retina vein occlusion, as well as ocular surface disorders such as corneal neovascularisation (6).

Intra-vitreal techniques have also been used for administering chemotherapeutic drugs to treat retinoblastoma(7). Intra-vitreal chemotherapy for retinoblastoma was first introduced more than half a century ago but is still controversial due to concerns of extraocular spread of tumour cells and seeding along the needle tracks(7). Currently, Intra-vitreal chemotherapy is only used for globe salvage in selected patients with advanced retinoblastoma or patients who have failed systemic chemotherapy(7). Nevertheless, a study has found that injective a bleb of subconjunctival carboplatin prior to drug delivery can mitigate the risk of orbital tumour seeding in advanced stage retinoblastoma (8).

In conclusion, this article has shown how Intra-vitreal therapies can play a huge role in the management of important ophthalmological conditions. The lady in this case scenario may benefit from the use of Intra-vitreal therapies such as the dexamethasone implant injection and also anti-VEGF agents to prevent the complications of her diabetic retinopathy. The development anti-VEGF agents has made a significant impact the management of AMD and other conditions associated with pathological neovascularisation. Despite the invasiveness of the procedure, Intra-vitreal injections have become a popular and effective method for delivering drugs to treat intraocular problems.

INTRA-VITREAL INJECTIONS

A Silvester, M Batterbury



Newer Intra-vitreal therapies are emerging, and this includes the dexamethasone Intra-vitreal implants used for the management of macular oedema and posterior uveitis. For the junior doctor, it is crucial to know how to explain the procedure and respond to the patient's concern regarding Intra-vitreal injections and also to be aware for signs and symptoms of potential side effects and contraindications of Intra-vitreal injections.

Multiple-choice questions (MCQs)

1. A 67-year old woman who was diagnosed with age-related macular degeneration underwent Intra-vitreal anti-VEGF injection of her right eye 2 days ago. She presents today complaining of increasing pain, redness and reduced vision in her right eye. What is the most likely diagnosis?

a. Anterior uveitis

b. Endophthalmitis

c. Acute angle-closure glaucoma

d. Conjunctivitis

e. Scleritis

2. The following diseases can be treated by Intra-vitreal injections except:

a. Retinal vein occlusion

b. Posterior subcapsular cataract

c. Age-related macular degeneration

d. Endophthalmitis

e. Retinoblastoma

Fitness to drive
Good Clinical Care

3. Which of the following investigations are not indicated in the diagnosis of central retinal vein occlusion (CRVO)?

a. Full blood count

b. Urine analysis

c. Intraocular pressure

d. Random cholesterol

e. Clotting studies

4. Which of the following is not a complication of Intra-vitreal injections?

a. Retinal detachment

b. Cataract

c. Raised intraocular pressure

d. Endophthalmitis

e. Cystoid macular oedema

5. Which of the following is not a risk factor for CRVO?

a. Diabetes

b. Hypertension

c. Cataract

d. Ocular hypertension

e. Blood dyscrasia

INTRA-VITREAL INJECTIONS

A Silvester, M Batterbury

MCQs: Answers

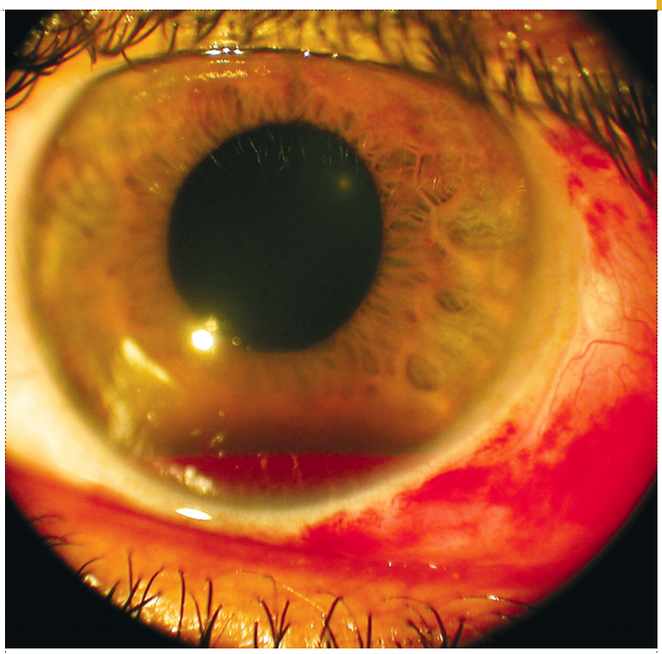
1. b. All except e)
can cause redness of the eye, pain and reduced vision. Endophthalmitis is more likely due to her recent Intra-vitreal injection.

2. b
There is no Intra-vitreal therapy for cataracts. Intra-vitreal injection of chemotherapy for retinoblastoma is used for globe salvage in selected patients with advanced retinoblastoma

3. d
It should be a fasting lipid profile, not a random cholesterol.

4. e
Cystoid macular oedema

5. c
Risk factors for CRVO includes arteriosclerosis, hypertension, diabetes and polycythaemia. Incidence of CRVO increases with age.



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LOW VISION AND REHABILITATION SERVICES: AGE-RELATED MACULAR DEGENERATION

E Cabourne, A Weston, K Cobb, A Koukkoulli

Abstract

Background: The ageing UK population brings an increasing demand on our healthcare system. Age-related macular degeneration (AMD) is an eye condition affecting the elderly with few treatment options, and is a major contributor to the number of people who are registered blind. Consequences of poor vision, such as falls and low mood, give rise to a high morbidity associated with the condition and the UK Vision Strategy has recognised that the provision of support services for these patients is inadequate. Clinicians should be able to recognise and refer patients who require support to the appropriate services.

Methods: A review and discussion of visual support has been carried out to understand the needs of our population with the view to introducing low vision rehabilitation to a higher proportion of visually impaired patients.

Results: There is a growing amount of evidence showing that the provision of emotional and financial support, encouragement, visual aids and guidance can improve the quality of life of many visually impaired patients. These services need to be offered from primary care, secondary care and from private consultations in order to improve the quality of life of visually impaired patients.

Conclusion: Optimising the patient's visual condition needs to be a key focus in the management of AMD, involving health care professionals and community services as a team to provide holistic and patient-centred care.

Key Words: Age-related Macular Degeneration, Visual Rehabilitation. Low vision aids.

Low Vision and Rehabilitation Services: Age-related macular degeneration
Teaching & Training

Introduction

The UK population over 75 is anticipated to increase from 4.7 million in 2007 to 8.2 million by 2031 (1). The UK Vision Strategy also predicts that by 2021, 40% of the population will be over 50 year old (2). The significance of our ageing population is the increasing burden of age-related health conditions on the National Health Service (NHS), which as a result demands reform and adaption.

Sight loss is a major health issue negatively impacting on a person's physical, social and mental well-being. Visual impairment currently affects two million British people, predominantly within the elderly population (3). Thus, with a predictable increase in the prevalence of visual impairment over time, ophthalmic support services need to be optimised.

The Royal National Institute of Blind People (RNIB) estimates that approximately 980 000 people have a certifiable visual impairment that is mostly attributed to conditions such as age-related macular degeneration (AMD), diabetic retinopathy and glaucoma (2). In 2008, 50.5% of those registered as severely sight impaired were due to AMD, which is recognised as the leading cause of irreversible blindness in the elderly in the western world(3). This article will consider age-related sight loss, particularly in AMD, and will explore the current services available and highlight the importance of adequate support. All healthcare professionals, including junior doctors, are expected to recognise visual impairment and offer appropriate guidance and support.

Psychosocial impact on the patient

Sight loss has proven to be a condition more feared than cancer (2). Adapting to life with visual impairment can dramatically reduce a patient's quality of life as a result of the barriers created by dependence, social isolation, fear and depression (3). It has been estimated that severe AMD can cause up to 63% decrease in quality of life, comparable to that experienced with advanced prostate cancer, severe stroke and uncontrollable pain (4).

LOW VISION AND REHABILITATION SERVICES: AGE-RELATED MACULAR DEGENERATION

E Cabourne, A Weston, K Cobb, A Koukkoulli

Studies have shown that severe visual loss is strongly associated with psychological symptoms and reduction in social participation, with 13.5% of those affected having depression, compared to 4.6% in those with good vision(5). In addition, over half of AMD patients are widowed and often live alone, which may further exacerbate depression and isolation (6). RNIB found that 48% of people with sight loss felt 'moderately' or 'completely' cut off from the people and things around them yet only 8% of patients were offered formal counselling . Therefore it is important for clinicians to account for the psychosocial effects of visual impairment and refer such patients when necessary.

Physical impact on the patient

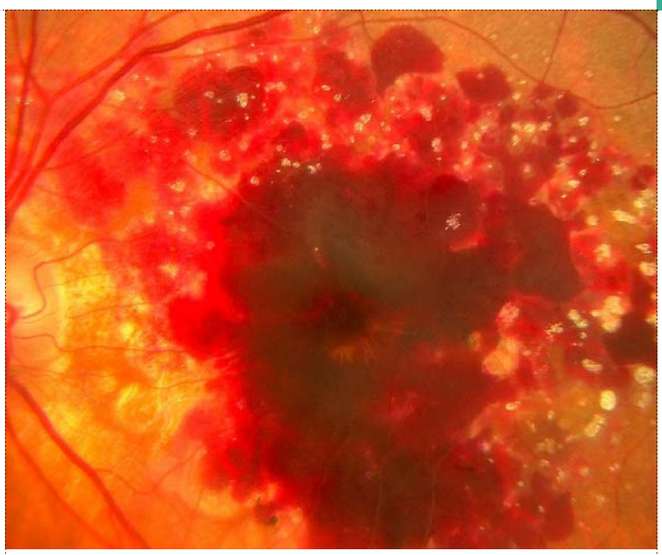
The elderly often have other medical ailments, which can make the adjustment to visual impairment even more difficult. The majority take multiple medications and reading the instructions can be virtually impossible with the risk of serious errors. Dual sensory loss in this group of patients is common, with a high proportion also having hearing impairment. Recognising this will allow referral to a sensory disability team that provides services to patients with hearing and visual impairment.

Mobility and orientation are highly impaired by sight loss as over 80% of information from our surrounding environment comes from sight. Older people with sight problems are 1.7 times more likely to have a fall and are at risk of suffering burns from cooking appliances(5). Further, blindness can cause disturbance of the normal sleep-wake cycle, as circadian rhythm directly relates to light perception (7). Most clinicians often fail to recognise this and the fact that it could further exacerbate depression.

Charles Bonnet Syndrome is a disorder of visual hallucinations that can occur in elderly people of sound mind who suffer from significant visual impairment (8). The hallucinations are varied from simple patterns to detailed pictures and can last from a few minutes to several hours. The patient often retains insight but is reluctant to talk about their symptoms due to fear of being labelled mentally ill. Reassurance and education seems to be the most effective treatment to date as it improves their ability to cope with the hallucinations and reduces anxiety (8).

Financial impact on the NHS

The annual healthcare costs for a patient with AMD is sevenfold higher than for a patient of the same age with no AMD (5). The economic burden of visual impairment can be compared to that of cancer, dementia and arthritis. As mentioned, visual impairment can also predispose patients to depression and increased risk of falls. Scuffham et al identified that of the 2.35 million falls that occur each year, 189,000 involve individuals with visual impairments costing the NHS £269 million (9). This highlights that both direct and indirect costs attributed to visual impairment are significant and warrant the need for change and sustainable improvement, particularly in current times when funding is restricted.



The importance of low-vision rehabilitation

Rehabilitation offers an important facility for patients with all degrees of sight loss and therefore should be available and easily accessible. With particular focus on AMD, many patients have no definitive cure and rely on hospital follow-up and community services to provide the support they need to adapt and optimise their mental, physical and social functioning.

The UK vision strategy recognised that the provision of eye health and sight loss services was unfit to meet current and future needs and thus outlined proposals, in response to the World Health Assembly Resolution of 2003 (2). This recognition of need has slowly made improvements within the public sector through the introduction of initiatives to improve equality and service provision to those with visual impairment.

Low-vision rehabilitation teams aim to provide help and support, by encourageing individuals to accept and adapt to life with reduced vision as early as possible and assist them to regain function and normal lifestyle. An evidence-based review has shown that input from low-vision rehabilitation can have a positive impact on the quality of life as it offers a combination of social, educational and vocational care (10).

Introduction of rehabilitation can be made initially in the outpatient department, although more emphasis is being made on alternative points of access such as outreach teams and community based services. There has been an explosion of support from the voluntary sector with services such as RNIB, Macular Disease Society (who offer local peer support groups and telephone counselling services), voluntary support centres and other national lottery funded projects. Information about UK groups can be found by contacting the RNIB helpline on 0303 123 9999. Carrying out an internet search for 'AMD support services' displays 14,100 hits in 0.23 seconds, all of which offer help and information to those who need it.

LOW VISION AND REHABILITATION SERVICES: AGE-RELATED MACULAR DEGENERATION

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Low Vision and Rehabilitation Services: Age-related macular degeneration
Teaching & Training

Low vision aid (LVA) services

LVA services provide a range of interventions, including equipment and training, to allow patients to make full use of their remaining sight and also provide a point of contact for follow-up support. Xuan-Nguyen et al found statistically significant improvement in the reading time of all study patients with reduced visual acuity due to AMD, when they were provided with appropriate visual aids (13).

Eye Clinic liaison officers work within certain eye clinics to provide information and guidance to patients about support they can receive practically, emotionally and financially from a wide variety of sources. Referral for this service can be directly from primary care, secondary care or even from private consultations. Introducing this facility nationwide would increase the number of patients receiving support, although individual trust funding will determine whether such a role is possible or not.

Registering provides patients with access to further means of support that includes concessions and benefits for a variety of services, clearly ensuring that a patient has the financial support to reduce the burden of their disability (table 1). Furthermore, the register is an official record of those with poor visual function and thus can serve epidemiological functions such as trend monitoring and audit collection, which can allow a better understanding of the provisions that are needed. Although the epidemiological role is important, problems may arise with accuracy because of the strict criteria that is required in order for a patient to be registered (box 1). The proportion of eligible patients registering is very low, with one study showing that less than half of those eligible register (14). It has also been observed that the number of people seeking visual aid services is higher than those that are registered, implying that more patients are requiring help than is estimated by the visual impairment registers.

Rehabilitation workers and social services work together within the patients' own environment to provide practical solutions and home adaptations to minimise their disability. Adaptations in the house include contrast enhancement, good lighting, tactile markers on applications and reading stands, and task lighting to aid the use of magnifiers. The mobility assessment and training will take into account the routes the patient regularly travels, their level of vision, mobility aids, physical fitness, preparation for guide dogs and use of sighted guide.

BENEFIT	CRITERIA
Blind persons personal income and tax allowance	Person must be registered as blind. In addition to usual personal tax allowances.
50% reduction on television licence	Person must be registered as blind.
Transport help	The Blue Badge Scheme (car parking), discounted rail travel with Disabled Person's Railcard and local bus pass.
Free postage	Items must be marked 'articles for the blind' and include books, papers, letters, Braille items, computer discs and CDs.
Electrical Items on loan	Certain local authorities provide those who are eligible with free permanent loan of radios, cd/cassette players.
Free NHS sight test	
Leisure concessions	Cinemas/exhibitions/theatres/concerts.
Council tax reduction	Depending on needs to accommodate for disability.
Attendance Allowance (AA)	Registration does not mean automatic qualification but those aged 65+ can apply for this benefit.
Disability Living Allowance (DLA)	Registration does not mean automatic qualification but those aged 18 – 64 can apply for this benefit.
Carers Allowance	Claimed if partner, relative or friend is eligible and spends time looking after patient.
Employment and Support Allowance (ESA)	Claimed if person is assessed as having limited capability for work.
Tax credits	Income-based benefits on top of income (wages and benefits). Working Tax Credit tops up wages if working 16+ hours a week.
Pension credit	Aged 60+. Guaranteed Pension Credit if weekly income at low level Aged 65+. Savings Pension Credit if patient has modest retirement income or savings.
Housing benefit and council tax benefit	Income based benefits to help people on low income with council tax and rent.
Exemption from 'non-dependants' deductions	A reduction in benefit if living with a non-dependant.
VAT relief	On products designed for those with visual impairments (such as daily living aids). Also, if an adaptation to the house is made for disability reasons, the person is entitled to VAT exemption.
Free Directory Enquiries Service	Exemption from BT Directory Enquiry charges.
Disability Discrimination Act	Protection under the DDA Act, 1995 - Gives rights to people with sight problems who have been treated unfairly.
Help with telephone costs	Help with telephone installation charges and line rental if the person is considered eligible.
Digital Switchover Help Scheme	Help with switching from analogue to digital TV.

Table 1: A table to show the available allowances and benefits that patients may be entitled to when registered blind or partially sighted.

LOW VISION AND REHABILITATION SERVICES: AGE-RELATED MACULAR DEGENERATION

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To be registered as *severely sight impaired* or *blind* and thus gain access to the linked services, a patient must fall into the following categories:

- visual acuity of less than 3 / 60 with a full visual field
- visual acuity between 3 / 60 and 6 / 60 with a severe reduction of field of vision, such as tunnel vision
- visual acuity of 6 / 60 or above but with a very reduced field of vision, especially if a lot of sight is missing in the lower part of the field.

To be registered as *sight impaired* or *partially sighted*, a patient must;

- visual acuity of 3 / 60 to 6 / 60 with a full field of vision
- visual acuity of up to 6 / 24 with a moderate reduction of field of vision or with a central part of vision that is cloudy or blurry
- visual acuity of up to 6 / 18 if a large part of your field of vision, for example a whole half of your vision or your peripheral vision is missing.

Box 1: The visual requirements a patient must have in order to be legally eligible for registration.

Who can currently register patients?

In accordance with the Royal College of Ophthalmologists, a consultant ophthalmologist is the only certified practitioner who can register a patient sight impaired or severely sight impaired by signing the Certificate of Visual Impairment (CVI). The CVI is a nationally recognised document that has been formed with input from the government, healthcare professionals and voluntary services.

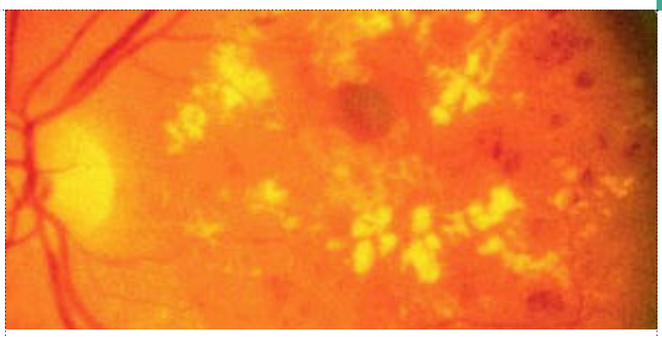
There are other forms that also alert social services of the patient's needs. An optometrist can give a Low Vision Leaflet (LVL) to a patient so that they can refer themselves. Alternatively, Referral of Visual Impairment (RVI) can be completed by hospital staff as to notify social services. Once these forms are received, the local authority has a legal duty to advise the patient on the range of services available and carry out an assessment of their needs.

Conclusion

There is a growing amount of evidence to support the use of low vision services as a successful tool to help patients with AMD to manage their sight loss in order to maintain a reasonable quality of life. Of course measuring quality of life can be very subjective, but studies have proven that simply providing visual aids, emotional support and rehabilitation can help adaptation and have a positive impact on the daily lives of patients.

Currently, services in the UK are fragmented and inadequate, which leaves a significant proportion of patients with limited access and at an increased risk of both depression and falls. Further, a large number of eligible patients remain unregistered, indicating that current services are not reaching patients. Clinicians should be able to recognise and refer patients who require support to the appropriate services, and improving staff awareness through training can help to achieve this.

With increasing focus on introducing eye care into primary care and the community, patients will no longer be relying only on their nearest hospital for information and support. Consistent pathways that connect healthcare, social services and community support will ensure that a holistic service is provided.



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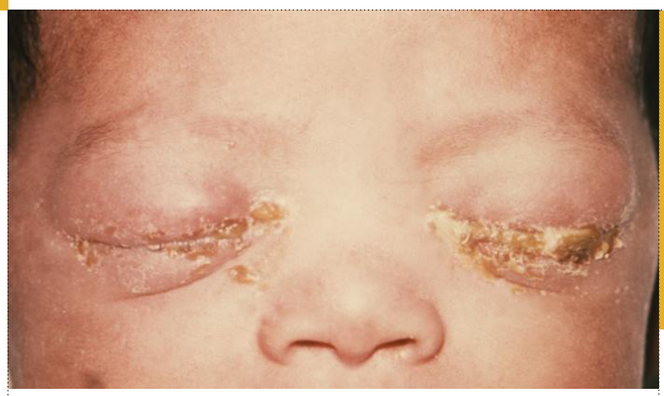
OPHTHALMIA NEONATORUM

MB Elahi, T Parmar, L Butler



Abstract

Ophthalmia neonatorum (ON) is defined as neonatal purulent conjunctivitis occurring within the first month of life. It is usually a result of direct spread of infection from mother to baby during vaginal delivery. Conjunctivitis in neonates may be caused by a variety of pathogens. The time of onset after birth and clinical characteristics of conjunctivitis are helpful adjuncts in reaching a tentative diagnosis. As a junior doctor, one should be very careful in dealing with newborns with conjunctivitis. It is therefore imperative to recognise and treat the condition early on, to prevent potentially sight threatening and systemic complications. Many NHS Trusts have local guidelines for the management of ophthalmia neonatorum.

Conjunctivitis in neonates may be caused by a variety of pathogens. The time of onset after birth and clinical characteristics of conjunctivitis are helpful adjuncts in reaching a tentative diagnosis. A neonate presenting with conjunctivitis is often daunting to the general practitioners and A&E doctors as well as concerning to the parents. It is therefore imperative to recognise and treat the condition early on, to avoid increasing anxiety and to prevent potentially sight threatening and systemic complications.

Definition

Ophthalmia neonatorum (ON) is defined as neonatal purulent conjunctivitis occurring within the first month of life. It is usually a result of direct spread of infection from mother to baby during vaginal delivery(3). Prompt diagnosis is essential due to the seriousness of the condition owing to lack of immunity and immaturity of the ocular surface(1). Delayed treatment may rapidly lead to blindness.

Background

Before the use of Crede's prophylaxis (silver nitrate), which was introduced approximately 100 years ago, there was widespread visual loss due to corneal infection by Neisseria(8). With routine use of silver nitrate drops at the time of birth the incidence of neonatal eye infection caused by Neisseria species has been greatly reduced. However nowadays silver nitrate drops is not commonly used in the UK because of limited use and potential side effect of chemical conjunctivitis.

Ophthalmia Neonatorum
Good Clinical Care

Epidemiology

The prevalence of ophthalmia neonatorum varies among different countries and is related to socioeconomic background, knowledge of general health and standards of maternal healthcare. Ophthalmia neonatorum affects up to 12% neonates in western world and up to 23% in developing countries.

Classification based on causes:

1. Gonococcal neonatal conjunctivitis

2. Chlamydial neonatal conjunctivitis

3. Herpes Simplex Virus neonatal conjunctivitis

4. Other bacterial neonatal conjunctivitis

5. Chemical conjunctivitis

Gonococcal neonatal conjunctivitis

Neisseria Gonorrhoea usually causes a bilateral, hyperacute infection within 1-3 days of birth (Table 1). There is a profuse purulent infection with lid oedema, conjunctival membrane or pseudo-membrane formation and chemosis (swelling).

There is risk of severe keratitis and corneal scarring leading to visual loss (3). Conjunctival swabs for gram staining as well as for culture should be performed urgently. If Gram negative diplococci are found the most likely diagnosis is gonococcal conjunctivitis.

Urgent treatment is indicated. The baby should be admitted and treatment started without waiting for results of culture. There is a risk of developing corneal ulceration and even perforation if appropriate treatment is not instituted. Treatment is Ceftriaxone 50mg/kg/day, intravenous for 1 week (table 2). Frequent saline irrigation of the ocular discharge helps to clean the eyes. The mother also needs to be referred to a GUM clinic or sexual health clinic for assessment and treatment. Contact tracing will be done through the clinic

OPHTHALMIA NEONATORUM

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Chlamydial neonatal conjunctivitis

C. Trachomatis is the most common cause of Ophthalmia Neonatorum in developed countries (5). It may be associated with pneumonitis, otitis and rhinitis (9,10). It may be responsible for third to a half of all neonatal conjunctivitis (8). The incubation period for Chlamydia is usually 4-28 days (Table 1). It presents as unilateral or bilateral conjunctivitis of mild to moderate severity. The discharge may be blood stained.

It has papillary rather than follicular reaction due to delayed development of palpebral lymphoid tissue. There may be an associated preseptal cellulitis. Basophilic intracytoplasmic inclusion bodies in conjunctival epithelial cells, polymorphonuclaer leukocytes or lymphocytes may be seen on Giemsa stain. The best way of making the diagnosis however is by PRC (7). Treatment involves oral Erythromycin 25mg/kg, twice a day for two weeks (10) (table 2). The mother also needs to be referred to a GUM clinic or sexual health clinic for assessment and treatment. Contact tracing will be done through the clinic.

Time of onset and cause of Ophthalmia Neonatorum

Time of onset of conjunctivitis in neonates holds an important clue to diagnosis:

Chemical	<2 days
Gonococcal	1-3 days
Other bacterial	2-5 days
Herpes simplex	1-14 days
Chlamydial	4-28 days

Table 1: Usual timing of presentation of ON.

Treatment Summary

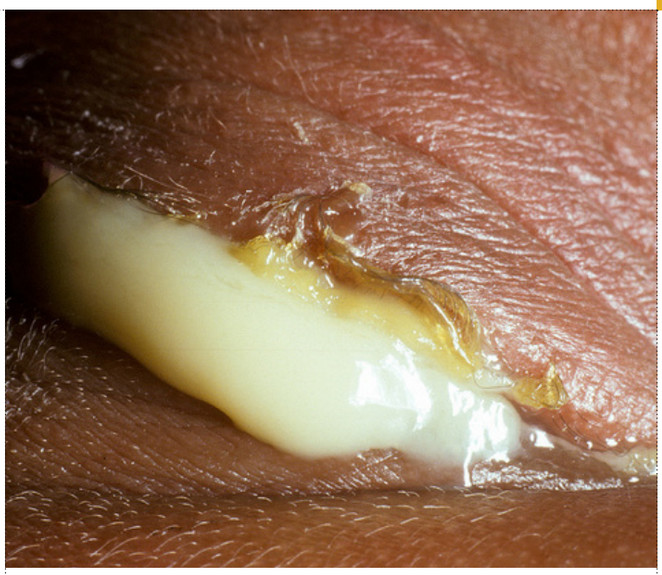
Gonococcal: *I.V. Ceftriaxone 50mg/kg OD for 1 week, + frequent irrigation of discharge from the eye with saline until resolved.*

Chlamydial: *P.O. Erythromycin 25mg/kg BD for 2 weeks, (or 3 weeks if systemic illness, e.g. pneumonia). Occ. erythromycin 0.5% or tetracycline 1% eye ointment may given as an adjuvant therapy, but must not be used as a sole treatment.*

Herpes simplex: *Occ. Aciclovir 5x for 1 week ± I.V. aciclovir 10mg/kg for 10 days in systemic infection*

Other bacterial: *Gram +ve (occ. Chloramphenicol or occ. Erythromycin qds according to sensitivities), Gram –ve (occ. Tobramycin qds).*

Table 2



Herpes Simplex Virus neonatal conjunctivitis

Although viral causes of neonatal conjunctivitis are uncommon, they can be associated with serious ocular and systemic complications. Herpes Simplex usually presents within 1 to 14 days of birth (Table 1). Corneal examination with fluorescein may reveal dendritic ulcers or microdendrites. Typical herpetic vesicles are usually seen on the eyelid margins.

Other ocular findings may include anterior uveitis, cataract, retinitis and optic neuritis. The baby may also develop systemic problems including jaundice, hepatosplenomegaly, pneumonitis, meningoencephalitis and disseminated intravascular coagulopathy. Diagnosis is best made by PCR. Giemsa stain will show multinucleated giant cells (7). The baby should be admitted for systemic treatment with intravenous acyclovir 10mg/kg, three times a day for 10 days. Topical acyclovir 3%, five times a day for seven days is usually given as well (table 2).

Other bacterial neonatal conjunctivitis

Staphylococcus aureus is a frequent cause of nasocomial infection in the newborn (2). Streptococcus pneumoniae and viridans, Escherichia coli and Haemophilus influenza may also cause conjunctivitis in this period (4). Pseudomonas aeruginosa is a rare but serious cause of neonatal conjunctivitis primarily occurring in premature infants (11). It can rapidly lead to keratitis and septicaemia that may be life threatening.

The organism can be detected on gram staining if an urgent diagnosis is required with culture and sensitivities providing subsequent information. Treatment involves hospital admission only if the baby is systemically unwell or if Pseudomonas is suspected for Gram negative organisms topical tobramycin, four times a day should be used. For gram positive organisms, topical chloramphenicol or ofloxocin four times a day are the usual first line treatments (table 2).

OPHTHALMIA NEONATORUM

MB Elahi, T Parmar, L Butler

Chemical neonatal conjunctivitis

Chemical conjunctivitis may be seen within a few hours of instilling silver nitrate drops. It usually does not last more than 24 to 36 hours (Table 1). It is rarely seen now as silver nitrate drops are rarely used (3).

Clinical approach to Ophthalmia Neonatorum

As a junior doctor, one should be very careful in dealing with newborns with conjunctivitis. Any conjunctivitis in first month of life is ophthalmia neonatorum. A baby with ophthalmia neonatorum, who requires admission, should be admitted under joint paediatric and ophthalmic care. There may be systemic features requiring parenteral treatment. An urgent opinion from a Paediatric Ophthalmologist should be sought if possible. Urgent gram staining and liaison with Microbiology on-call team should be considered. Neisseria gonorrhoea and pseudomonas can cause rapid corneal involvement and lead to permanent visual damage. Swabs for bacterial culture & sensitivity as well as swabs for PCR for Chlamydia and HSV should be taken. Treatment can be changed based on the outcome of these results. Daily ophthalmic review is required. Many NHS Trusts have local guidelines for the management of ophthalmia neonatorum.

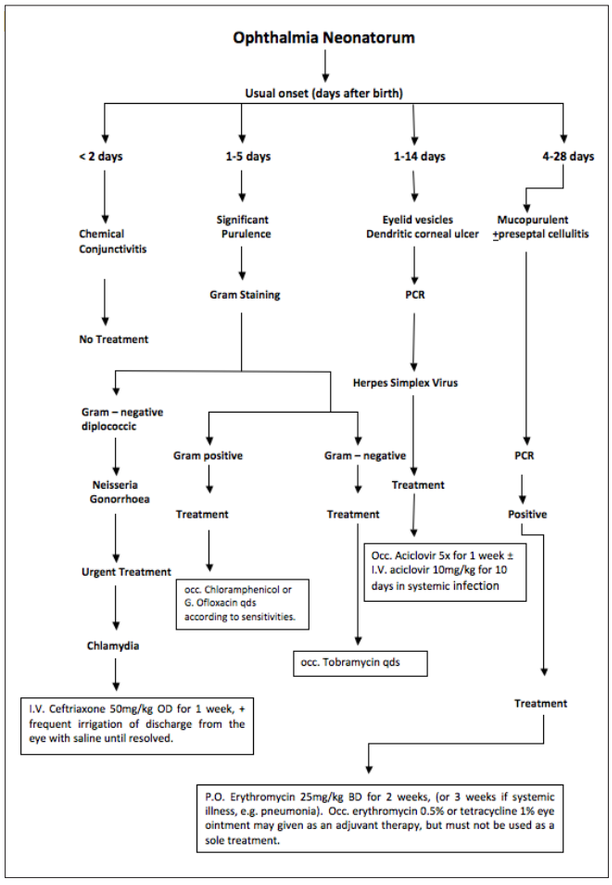
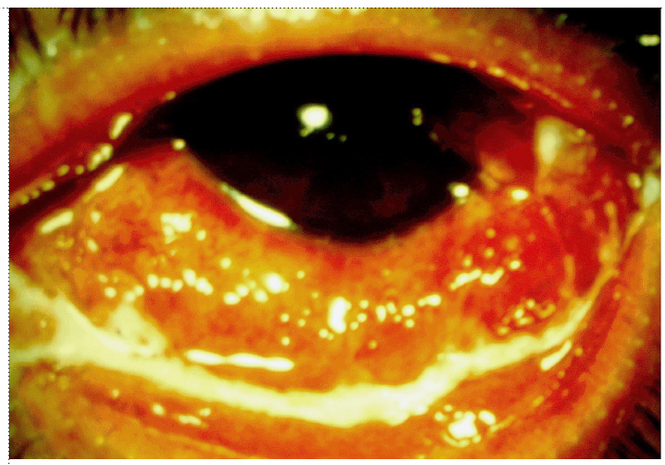


Figure 1: Management of Ophthalmia neonatorum.



MCQs

Choose the single best answer from the following:

Q1. What is the first most important diagnostic test for Ophthalmia neonatorum?

a. Swabs

b. Gram staining

c. PCR

d. ELISA

e. Corneal scrape

Q2. What is the most common cause of Ophthalmia neonatorum?

a. Gonococcal neonatal conjunctivitis

b. Chlamydial neonatal conjunctivitis

c. Herpes Simplex Virus neonatal conjunctivitis

d. Other bacterial neonatal conjunctivitis

e. Chemical conjunctivitis

OPHTHALMIA NEONATORUM

MB Elahi, T Parmar, L Butler

Q3. Which one of the following is associated with most serious systemic complications?

a. Gonococcal neonatal conjunctivitis

b. Chlamydial neonatal conjunctivitis

c. Herpes Simplex Virus neonatal conjunctivitis

d. Other bacterial neonatal conjunctivitis

e. Chemical conjunctivitis

Answers

1. Answer: b (Gram staining)

2. Answer: b (Chlamydial neonatal conjunctivitis)

3. Answer: c (Herpes Simplex Virus neonatal conjunctivitis)

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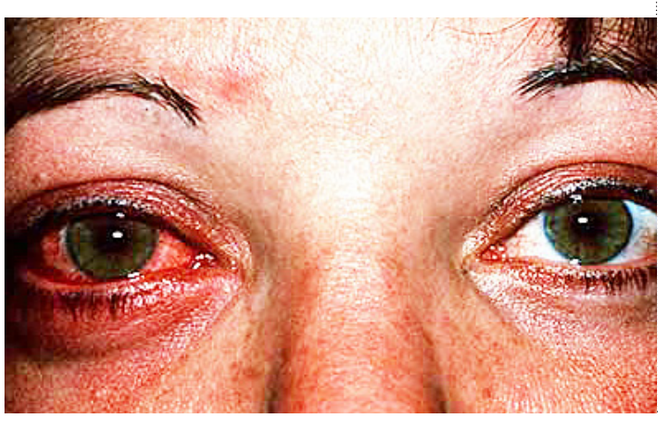

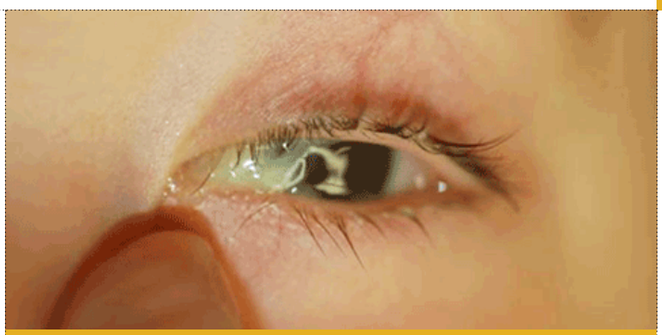

Ophthalmia Neonatorum
Good Clinical Care

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AN OVERVIEW OF HOW TO PERFORM AN ORTHOPTIC ASSESSMENT

S Mall, A Sharma

Abstract

A systematic method of examining patients with ocular motility disorders will be presented. This will be useful for all doctors including those in Ophthalmology training. We aim to outline an approach which will enable one to grasp the key pointers in the history, as well as ascertaining the relevant signs. A brief quiz has also been included to test your knowledge.

An orthoptic assessment is usually performed by an Orthoptist who is specialised in the diagnosis and non-medical management of strabismus (squint), amblyopia (lazy eye), and eye movement disorders.

It is important as a medical doctor to have an understanding of ocular motility assessment and reach a diagnosis. The findings from an orthoptic assessment are documented in an orthoptic report which has a standard format in ophthalmic departments throughout the UK.

A patient with an ocular motility disorder usually complains of diplopia (double vision), reduced vision, eye strain or headache. On examination one can often elicit one of the following signs: manifest strabismus, an abnormality of ocular movement, nystagmus (rapid involuntary movements of the eyes) or an anomalous head posture (AHP).

An orthoptic assessment begins with a good history, depending on the age of the patient the questions will vary.

An overview of how to perform an orthoptic assessment.
Practical Procedures

Case History for Children

History of Presenting Complaint

Are there any concerns regarding the vision?
In what direction are they noting the eye turn (in, out, up or down)?
Did it develop suddenly or gradually?
Which eye is involved or is it both eyes on separate occasions?
Is it increasing in frequency?
Is it more noticeable when tired or unwell?
Is it more noticeable for near and/or distance fixation?

Obstetric/development history

What was the birth weight?
Was it a normal vaginal delivery?
What was the gestational age?
Likelihood of gestational intake of excessive drugs or alcohol?

Family History (FH)

Is there any FH of strabismus/ refractive errors/hereditary eye conditions?

Past Ophthalmic History

Did the child have Retinopathy of Prematurity screening?

Case History for Adults or children old enough to complain of symptoms. In many cases adults present with acquired eye movement defects and are usually symptomatic with diplopia. It is essential to find and treat the underlying cause.

History of presenting complaint

Is the diplopia present when either eye is covered up?
Are the images sitting horizontally or vertically or both?
Is the separation bigger between the two
images greatest at near or distance fixation?
Has there been any recent head trauma?

AN OVERVIEW OF HOW TO PERFORM AN ORTHOPTIC ASSESSMENT.

S Mall, A Sharma

History of headache or blurred vision

Other symptoms: Has there been any unsteadiness of gait?
Any weight loss? Any change in appearance noted by the family?

Medical history

Specifically ask about cardiovascular risk factors:
Ischaemic Heart Disease/Diabetes/Hypertension
Drug History
Anticonvulsants can be the cause of defective ocular movement
Past Ophthalmic History
Childhood squint
Previous episodes of diplopia
Social History
Alcohol abuse can lead to thiamine deficiency
which is associated with Wernicke's encephalopathy
Cigarette Smoking

Observations

Observe any anomalous head posture (AHP) this is adopted by the patient to maintain binocular single vision (BSV) or to obtain better visual acuity (VA). Note any facial asymmetry or ptosis.

Ocular Examination

1. VA

The level of acuity in each eye can be tested individually and in some patients with both eyes open. A variety of methods are used in both adults and children dependant on their ability.

2. Cover Test

The purpose of this test is to elicit the presence of a manifest (heterotropia) or latent deviation (heterophoria). It also provides information about the type of deviation (eso, exo, hyper, hypo), the size of the deviation, and the speed of fixation (this indicates the level of vision). (1) This test is performed with and without glasses, and also with and without an AHP. It is performed at a distance of 1/3 metre and at 6 metres (Figure 1).

Figure 1: Cover test being performed at 1/3 m.

How to do a Cover test:

1. Use a target 1/3m away.
2. One eye is covered with an opaque occluder and the other eye is observed.
3. If the uncovered eye takes up fixation- a manifest deviation is present.

The eye moves out for an Esotropia (ET)
The eye moves in for an Exotropia (XT)
The eye moves up in a Hypotropia (HYPOT)
The eye moves down in a Hypertropia (HYPERT)

Table 1: Eye movement on cover test in relation to manifest deviation.

4. The movement of the covered eye is observed as the occluder is removed from the contralateral eye to observe for a latent deviation.
5. Then an alternate cover test is performed, in which you repeatedly cover each eye in turn for 2-3 seconds so that one eye is always covered.
6. The test is then repeated at 6 metres.

The alternate cover test detects the total manifest and latent deviation by causing dissociation of BSV. It also allows assessment of any dissociated vertical deviation and manifest latent nystagmus which is common in infantile esotropia. (2)

3. Convergence

1. Ask the patient to look at a target 30cm away held perpendicular to nose.
2. Move this target slowly towards the bridge of the nose.
3. Measure the distance from the eyes at which the patient says the target becomes double.

This is the near point of convergence and it is usually around 10cm.

AN OVERVIEW OF HOW TO PERFORM AN ORTHOPTIC ASSESSMENT.

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An overview of how to perform an orthoptic assessment.
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4. Test for level of BSV

· Stereopsis - is the third and best level of BSV. This is regularly tested by using Randot stereogram, The Lang stereo test, Frisby test and TNO test (Figure 2). The normal level of stereopsis being 40 seconds of arc.

Figure 2: Tests used for assessing Stereopsis; TNO, Frisby and Lang.

5. Eye movements

· Smooth pursuit is tested by asking the patient to follow a light with their eyes without moving their head. It is essential that the patient is not wearing their glasses and any AHP is corrected. The purpose of this test is to determine if there is any limitation or restriction of any of the 12 muscles around the eye (Figure 3).

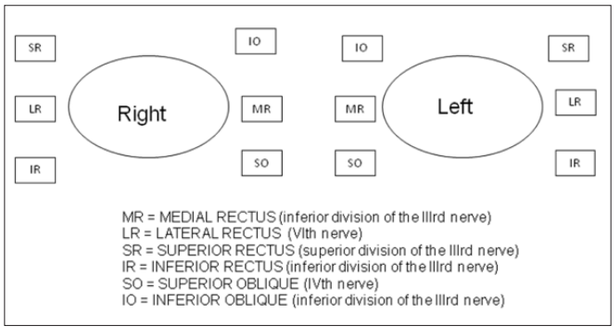

Figure 3: Diagram of the positions of the 12 muscles around the eyes and their innervation.

A duction is the assessment of one eye movement alone, and a version is the simultaneous movement of both eyes together (Figure 4) (3).In a nerve palsy the ductions are more than the versions and in a mechanical strabismus the ductions would equal the versions.

During this test patient is asked to report any diplopia and any overactions or underactions are documented.

Saccades

1. Hold two targets in front of the patient
30 degrees either side of the midline.

2. Ask patient to look rapidly at one target and then the other.

3. Repeat this several times in the horizontal plane
then test saccades in the vertical plane.

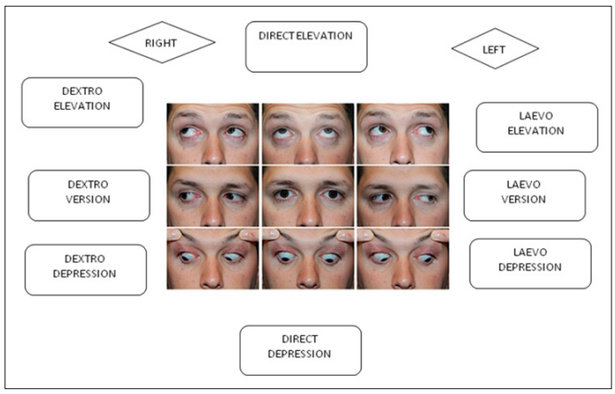

Figure 4: Diagram of the versions when moving the eyes in 9 positions of gaze from the primary position.

AN OVERVIEW OF HOW TO PERFORM AN ORTHOPTIC ASSESSMENT.

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Vestibulo-Ocular Reflex (VOR)

It is useful to examine this in a patient with partial or total ophthalmoplegia. In a supranuclear ophthalmoplegia the eye excursion elicited by VOR is greater than that on smooth pursuit or saccades.

1. First ask the patient if they have any neck problems - if they do avoid this test.
2. Horizontal VOR – ask the patient to keep looking at your nose and rotate their head from side to side.
3. Vertical VOR - asks the patient to keep looking at your nose and tilt their head forwards and backwards.

A normal response is for the patient's eyes to remain fixed on your nose.

Optokinetic Nystagmus (OKN)

This is a normal response of the ocular movement system; it is a form of jerk nystagmus which allows the eyes to follow an object in motion when the head remains stationary, for example it allows one to look at the view whilst travelling in a fast moving train.

OKN is a useful test if the patient is thought to have a non-organic cause for visual loss or localise the site of a lesion causing a homonymous hemianopia. (4) A rotating OKN drum is rotated to the right, left, up and down. An observation is made as to whether OKN is elicited or reduced (Figure 5).

Figure 5: OKN Drum.

6. Measurement of the deviation

The angle of the deviation can be measured using the prism cover test (PCT) and also the Hirschberg's test. The prism cover test is performed at 1/3m and 6 metres; it is also performed with and without spectacles and AHP.

1. A prism bar (Figure 6) is placed in front of the deviating eye in manifest deviations and before either eye in latent deviations.
2. The other eye is occluded.
3. An alternate cover test is performed by moving the occluder from one eye to the other while watching the movement of the eye behind the prism as it takes up fixation.
4. The prism strength is then increased gradually until the movement decreases and eventually there is no movement to take up fixation.
5. The prism strength is noted and is equivalent to the maximum measurement of the angle of deviation.

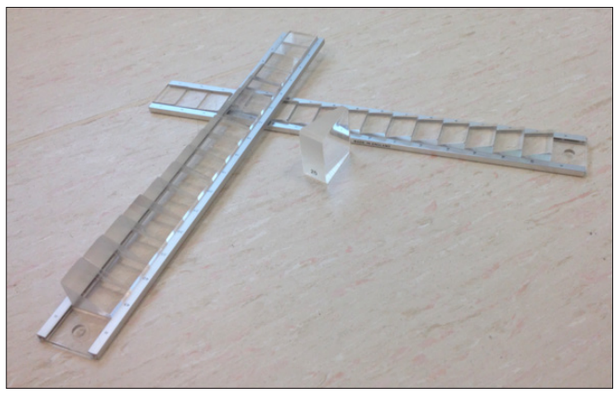
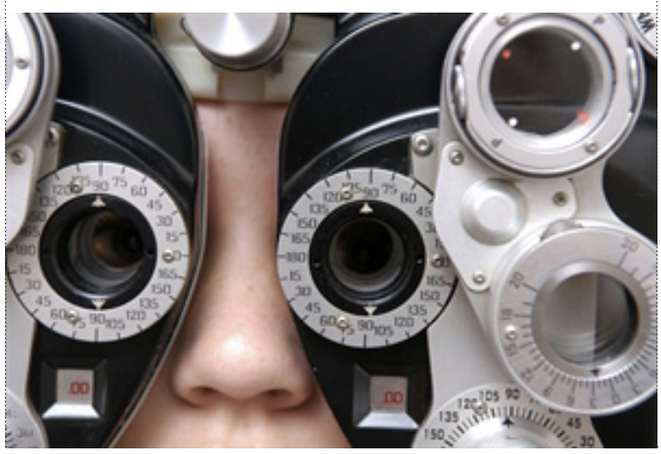


Figure 6: Horizonal and Vertical Prism Bars.

Hirschberg's test is based on the presence of comparable corneal reflections in the squinting eye with that of the fixing eye. This test can be very easily performed in an outpatient clinic or on the ward without any specialist ophthalmic equipment.

A spotlight is shone into the patient's eyes and an estimated measurement is taken as to the angle of deviation by observing the position of the corneal reflection of the deviating eye.

A 1mm displacement is equivalent to 15 prism dioptres.1Reflections displaced temporally indicate presence of an eso- deviation while nasal displacement indicates an exo-deviation (Figure 7).



AN OVERVIEW OF HOW TO PERFORM AN ORTHOPTIC ASSESSMENT.

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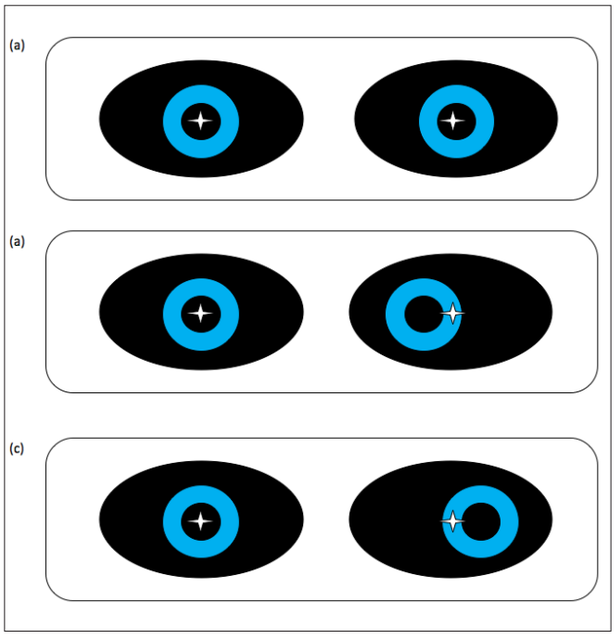

Figure 7: Hirshberg's test (a) Normal corneal reflection (b) Temporal displacement of corneal reflection in a left esotropia (c) Nasal displacement of corneal reflection in a left exotropia.

This completes the orthoptic assessment and a report is formulated (Figure 8).

An overview of how to perform an orthoptic assessment.
Practical Procedures

29/04/2012 – Aged 4 ½ Years

Referred by Local Optometrist

Mum concerned after playing a game and noticed white reflex in left eye. Mother concerned & took patient to local Optometrist and they noted a possible cataract in the left eye.

PMH – Good

Previous Ophthalmic History - Nil

Birth History – Full Term Normal Delivery, Birth Weight- 7lb

Family History – Nil

VA RE -0.100 [6/5] UNAIDED (U/A) LE 0.800 [6/36] LogMAR (Snellen)

Cover Test Near(1/3m) - Small esophoria (EP) going to a Small 10 -12 prism dioptres L esotropia (ET)

Distance (6m) – moderate EP with moderate recovery

Ocular Movements – Full

Convergence – Binocular to nose

Frisby – negative

DIAGNOSIS – LEFT Amblyopia

LEFT Esotropia, greatest at near

IMPRESSION – Left VA reduced. Possible congenital cataract

PLAN – Refraction, fundus and media examination.

Figure 8: Orthoptic report.

Quiz

1. Figure 9 shows a photograph of a patient with horizontal diplopia. Choose from one of the following options below which best explains the type of deviation;

(a) Esophoria
(b) Exophoria
(c) Exotropia
(d) Esotropia
(e) Hypertropia

AN OVERVIEW OF HOW TO PERFORM AN ORTHOPTIC ASSESSMENT.

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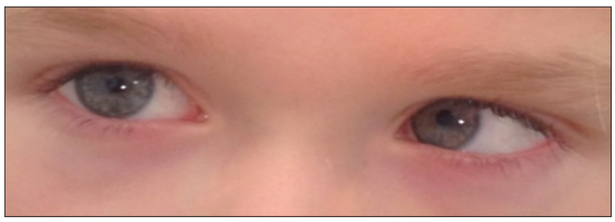

Figure 9: A patient with a manifest deviation.

2. Figure 10 shows a photograph of a patient with both horizontal and vertical diplopia. What is the most likely diagnosis?

(a) 7th nerve palsy
(b) Thyroid eye disease
(c) 6th nerve palsy
(d) 3rd nerve palsy
(e) Esophoria

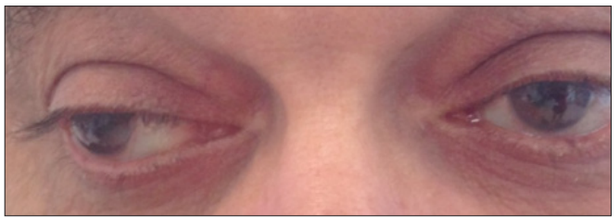

Figure 10: A patient with manifest deviation and right partial ptosis.

Answers

1. This figure shows a left eye esotropia (manifest deviation) in a four yr old patient. This can be further classified into accommodative and non-accommodative forms. The differential diagnosis would include a sixth nerve palsy, Duane's syndrome, esotropia associated with myopia and nystagmus blockage syndrome.

2. Right 3rd nerve palsy

For a pupil involving or partial 3rd nerve palsy the aetiology is often compressive and requires an urgent MRI with MRA. In children imaging should be performed regardless of the state of the pupil. Posterior communicating artery aneurysms require immediate transfer to neurosurgical unit for coiling or clipping.

The aetiology of a pupil sparing complete 3rd nerve palsy is often ischaemic. The systemic workup should include blood pressure, glucose, lipids, ESR, CRP and FBC. The patient needs to be monitored closely for the first week to ensure no pupil involvement. If the patient has symptoms of giant cell arteritis and a raised ESR and CRP, systemic corticosteroids should be initiated and a temporal artery biopsy performed. If a microvascular third nerve palsy shows no recovery at 3 months then further investigations should be performed including MRI and lumbar puncture.

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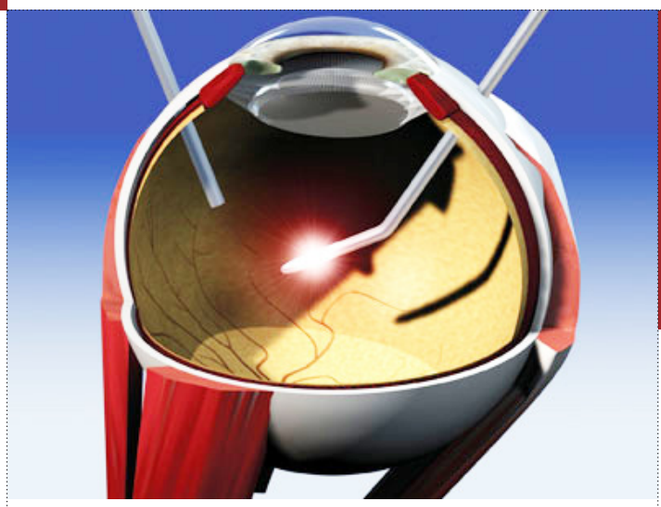
RESOLUTION OF VITREOMACULAR TRACTION & ASSOCIATED PIGMENT EPITHELIAL DETACHMENT FOLLOWING VITRECTOMY

GF McGowan, ZR Koshy



Resolution of vitreomacular traction and associated pigment epithelial detachment following vitrectomy
Patient Management

Abstract

Keywords: vitreomacular traction, pigment epithelial detachment, vitrectomy, distortion.

A sixty eight year old female presented with a three-month history of central distortion and reduced vision. Snellen visual acuity in the right eye was recorded at 6/18 and in the left eye 6/9. Fundal examination revealed vitreomacular traction associated with a pigment epithelial detachment (PED). These findings were confirmed by optical coherence tomography (OCT).

A 3 port pars plan vitrectomy with injection of C2F6 gas with no posturing resulted in release of the traction and resolution of the retinal distortion and PED (see figure). Vision in the right had recovered to 6/9 unaided with no subjective distortion at 6 weeks following surgery.

PED associated with vitreomacular traction has been reported in the literature. (1,2) This case besides highlighting the utility of OCT on surgical decision making also significantly, demonstrates the complete resolution of the PED and subsequent return of normal visual acuity following surgical release of vitreomacular traction.

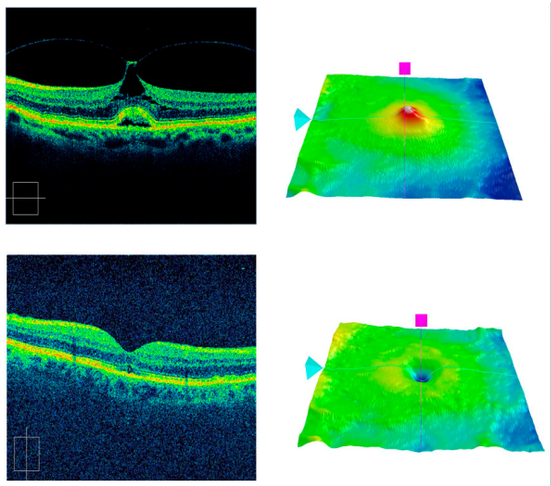


Figure 1: Pre and Post-operative OCT.

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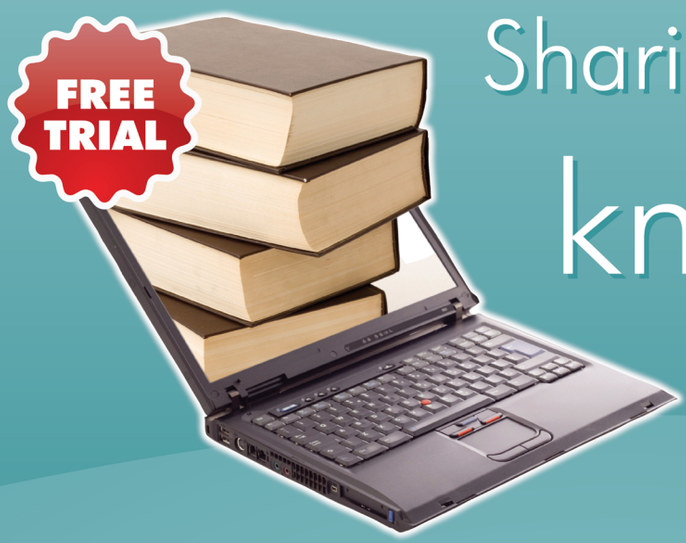

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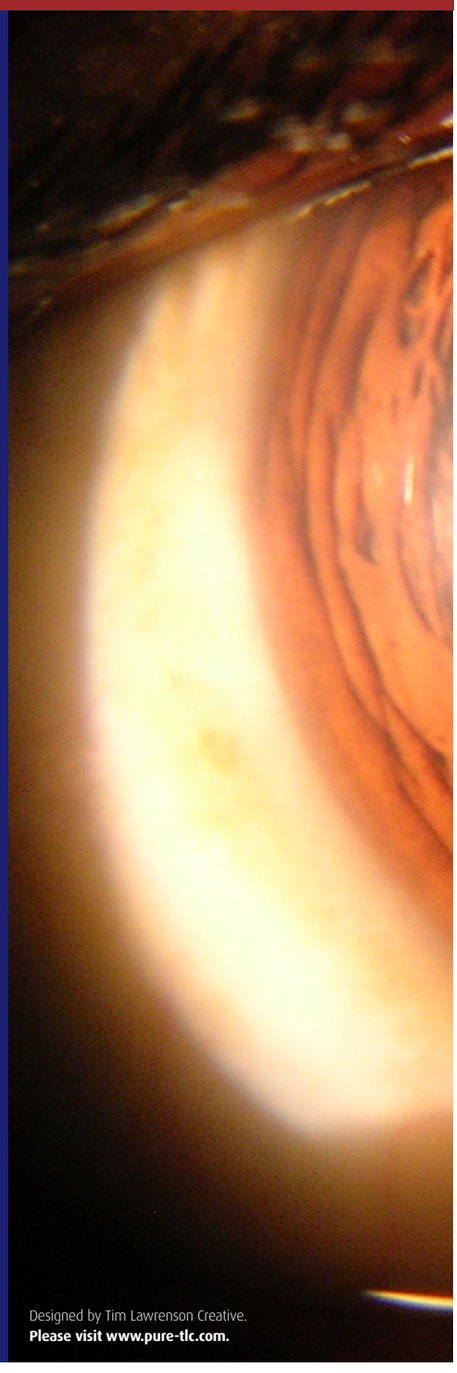

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