The Newcastle upon Tyne Hospitals NHS Foundation Trust

Eye Emergency Department (EED) Guidelines

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Ratified By:	Miss Lucy Clarke, Head of Emergency Eye Department

1. Aim

The aim of these guidelines is to provide a basic management plan for a range of conditions likely to be seen in EED. In particular they are meant to outline what investigations may be appropriate from EED and provide guidance for appropriate referral on of the patient. They are not meant to replace textbooks, which will contain far more detail, but should be used in conjunction with them.

2. Principles

EED is the appropriate setting for patients with urgent or acute eye problems. It should not be replacing outpatients. Patients with chronic or non - urgent conditions should be investigated and treated from clinic not casualty.

a) Patients should only be followed up a maximum of once in casualty and then referred to clinic if necessary.
b) Think carefully if a patient needs to be followed up at all or simply advised to return if the condition is not settling.

3. General guidance

Clearly it is hoped that all patients will be accurately diagnosed and appropriately treated, however that does not mean that every last detail needs to be sorted out in casualty. The important step is to distinguish serious pathology from more minor conditions. Remember many conditions are not sight threatening and are self-limiting.

- Always check visual acuity.
- If the visual acuity is reduced always try and explain why.
- Have a low threshold for a dilated fundal examination.
- Have a low threshold for glucose and BP measurements.
- Ask for help when needed, but bear in mind need for urgency, i.e. now, today, in outpatients.
- Try and make a diagnosis and or action plan.
- Write this clearly on the casualty card.
- Sign legibly and use stamp

CONJUNCTIVITIS

Precautions

1. Steps must be taken to make sure you do not catch infection yourself or transmit it to other patients.
2. Do not applanate – if you do, by mistake, wash the tonometer head under the tap (when you wash your hands!) **before** placing it in hypochlorite solution.
3. Wash your hands.
4. Dispose of minims and tissues.
5. Wipe down chin and head rest of slit lamp and any other surfaces that may have become contaminated with tears with disinfectant spray and or wipes.
6. Warn patient not to go swimming and to use their own towel until the episode has cleared.

Swabs

Will it change my management?
Not indicated unless:

- No improvement and diagnosis unclear, e.g. to distinguish between adenoviral, chlamydial and Thygeson keratitis

- Rarely: If possible outbreak suspected for infection control.

Viral Conjunctivitis:
There is no indication for routine swabbing for viral conjunctivitis.
As from 1st August 2005, the Health Protection Agency at NGH will move from a culture based to a PCR based identification technique for adenovirus.
PCR (compared to culture) is more sensitive, faster and more expensive.

Chloramphenicol

- Evidence for aplastic anaemia due to absorption is minimal in adults.
- There is no logical basis for qds prescribing of drops. Antibiotic drops should be administered hourly for two days rather than qds for one week. Ointment qds is acceptable but bioavailablility is less predictable.

CONJUNCTIVITIS - Diagnosis

Aetiolgy	Condition	Onset/ Duration	Symptoms	Conjunctival Response	Preauricular Lymph-adenopathy	Discharge
Bacterial	Bacterial	Acute Usually one eye	FB sensation Purulent discharge Lid Crusting Tearing	Diffuse hyperemia Papillae	Occasional	Purulent
Viral	Adenoviral	Acute Usually bilateral	Tearing Gritty discomfort Lid crusting on waking	Diffuse hyperemia Follicles Petechial hges Pseudomemb	V Common	Watery Serousmucoid
	Herpetic	Acute	Tearing	Diffuse hyperemia Follicles	Occasional	Watery Serousmucoid
Allergic	Seasonal	Seasonal /recurrent	Itching Tearing	Mild hyperemia Mixed follicles/ papillae	Unusual	Mucoid
	Vernal	Seasonal/chronic	Itching Mucus discharge	Giant papillae (tarsal) Trantas dots (limbal)	Unusual	Ropey Mucoid
	Giant Papillary	Acute/ Chronic	Itching Contact lens intolerance Mucous discharge	Giant papillae – check for shield ulcer	Unusual	Mucoid
Chlamydial	Chlamydial	Acute/ Chronic	Tearing	Diffuse hyperemia Giant follicles (predominantly inferiorly)	Occasional	Mucoid

CONJUNCTIVITIS - Management

CONDITION	MANAGEMENT	FOLLOW UP
Bacterial Conjunctivitis	Start topical antibiotic: 1st line: Chloramphenicol. 1st line: Fucithalmic **(*Do not use* fluoroquinolone antibiotics such as oflocaxin or ciprofloxacin.)** Educate patient about cleaning and contact	No routine follow up. Advice to return if not better.
Adenoviral Conjunctivitis	Cold compress **Acute**: Topical antibiotic only if secondary bacterial infection **Late with subepithelial punctate keratitis**: Vision unaffected : no treatment Photophobia and reduced vision: no Rx or predsol 0.5% 4/3/2/1/d each week plus clinic follow-up. Educate patient about cleaning and contact	**Acute:** No routine follow up. Advise symptoms can last for a month. Re-attend if vision becomes affected **Late**: Clinic follow up
Allergic Conjunctivitis	Identify/remove allergen (consider regular eye drops) Cold compress Educate patient Opatanol BD Cetirizine 10mg po OD (N.B.: Remember Contact lens)	No routine follow up. Advice to see GP for persistent chronic symptoms
Chlamydial Conjunctivitis	See following section	See following section

Management of Chlamydia Conjunctivitis

Diagnosis
Persistent follicular conjunctivitis, usually unilateral, often quite productive of mucus and discharge.

Investigations
The nursing staff will take a conjunctival swab (fornix scrape) with the specific kit and send to microbiology.
A patient contact telephone number should be documented on the EED card.
The patient does not need to be told to return, they will be informed of any positive result that affects their management.

Management
A member of EED staff will contact the patient with a positive result and ask them to attend EED. As a policy we do not give out test results over the phone.

The patient should be informed of the importance of:
• Treatment to avoid long term complications
• Partner notification and abstaining from unprotected sexual intercourse until they and their partner(s) have been treated
• Attendance at a Sexual Health / GUM clinic for full screening to exclude other STIs

Ideally the patient should be asked if they consent to referral to health advisor team at the New Croft Centre, who will then be contact them directly. This will then allow them to have full STI screening, appropriate antibiotics and partner notification.

Email: tnu-tr.newcroftsexualhealth@nhs.net A scanned copy of the cas card or a letter with the positive result documented, clearly marked FAO: Health Advisor Team
Telephone: 0191 229 2914 (Health Adviser team) for queries.
There are information cards with directions of how to reach the New Croft Centre in the “info card” drawer in EED.

Patients who refuse referral, are unable to attend, or require urgent treatment should be given oral antibiotics. There is no effective topical cure, lubricants may provide symptomatic relief.

Antibiotic therapy:
- 1st line: Azithromycin 1g po stat single dose
- 2nd line: doxycycline 100mg BD po 7 days
- 3rd line: erythromycin 500mg BD 14 days or ofloxacin 200mg BD 7 days

Caution with all during pregnancy and breastfeeding – seek advice from New Croft Sexual Health Service ‘Duty Dr’ via 0191 229 2999

Meibomian gland dysfunction/marginal keratitis

Meibomian gland dysfunction.
Explanation that this is a chronic relapsing condition.
Lid hygiene & viscotears - patient information leaflet.
Discharge **no follow up**.

Marginal keratitis.
Confirm lesions are concentric with limbus and document clear interval.
Document corneal sensation.
G. Maxitrol qds for 10/7
Treat meibomian gland dysfunction as above.

- Discharge no follow up unless numerous relapses or significant underlying MGD – refer to **general clinic routine**

Herpes simplex keratitis

Dendritic ulcer (epithelial disease).
Oc acyclovir 5 times daily maximum 2 weeks
Discharge **no follow up**.

Disciform keratitis (stromal &/or uveitis)
Oc acyclovir 5 times daily maximum 2 weeks **ONLY** if any associated epithelial disease

- Oral acyclovir 400mg po BD.
- Add topical steroid betnesol 0.1% QDS once epithelial defect resolved if present.
- Consider increasing steroid frequency if significant uveitis and no epithelial defect
- Refer to **corneal clinic soon/1 month** (corneal fellow clinic if available) – continue treatment unchanged until then (to go to GP for repeat prescription).

Herpes Zoster Ophthalmicus

Document corneal sensation and IOP
Oral acyclovir 800mg x5 day for 1 week, if rash onset <5 days.
Consider additional occ acyclovir x5/d **ONLY IF SIGNIFICANT** ocular surface involvement, eg. Extensive epithelial keratitis rather than just SPEs
Advise lubricants and oral NSAIDS for analgesia with GP f/u to discuss neuralgia
Cautious treatment of uveitis: betnesol 0.1% QDS with oral ACV cover
Raised pressure: timoptol 0.25% BD, **casualty follow up < 1week** to ensure improved
Refer to **general clinic soon/1 month** if corneal or intra-ocular involvement.

Bacterial Keratitis

For full document Mangement of Keratitis

These cases should ideally be reviewed by the EED registrar if they are being brought back to EED for review.
Document: contact lens use (type/duration of wear/last worn), preceding trauma, past corneal disease or surgery, topical drop use, systemic antibiotic or immunosuppressive.

Microbiology samples
Send contact lenses & case if possible, with consent from patient.
Wipe pus/mucus/debris with a sterile C&S swab & send for culture.

Scrape kits are kept in the fridge in EED and on ward 20. Use a green needle or 15 blade, fresh for each plate or slide. Obtain samples from the advancing edge of the ulcer. Inoculate the surface of plates over the central cross and the slides in the etched circle.
Kit in order of inoculation:

1) chocolate agar
2) 1st slide (gram stain) labelled on frosted end with pencil
3) blood agar
4) Sabouraud's

Then if acanthamoeba is suspected:

5) 2nd slide (calcofluor for acanthamoeba/fungal) – discard if not used
6) Acanthamoeba agar

Ensure each sample is labelled with a patient sticker. The microbiology request form should have clear documentation of the contact telephone number for gram stain results. All samples to be sent as urgent.

Treatment
G. Ofloxacin 0.3% hourly daytime for 48hrs. If ulcer <1mm, off axis, no atypical features then instruct the patient to **return only if no improvement**. Otherwise they do not have to return, can reduce their drops to 2hrly for the week. No CL wear for further week without treatment, then see their own optician before resuming CL wear.
If ulcer >1mm or any atypical features, scrape, EED reg to see then EED follow up 24-48 hours.
Consider additional cyclopentolate 1% TDS and oral NSAIDS for analgesia.
Depending on severity and difficulty of drop administration consider admission plus hourly G. ofloxacin 0.3% and G cefuroxime forte hourly, alternating half hourly. In any doubt refer to the full protocol online, seek senior &/or corneal specialist advice.

For severe or atypical ulcers that WILL require corneal input, refer for review at the first visit. Get the ball rolling so that the patient isn't seen in EED several times before they are referred. Call the fellow, or speak to the corneal consultant on the rota for advice

Chemical injury

Aims are to ensure immediate and adequate lavage of injured eyes and to identify those which may require more aggressive treatment.

Initial management
Anything other than CS gas irrigate eye with at least 1000 ml of normal saline.

- Check & document the pH of both eyes before commencing
- Use topical anaesthetics liberally to facilitate irrigation and examination
- Irrigate the superior and inferior cul-de-sacs and remove any foreign bodies that may act as a depot for the chemical. Intravenous tubing connected to a container of normal saline makes an ideal irrigation tool. A lid speculum greatly facilitates irrigation.
- Continue irrigation with sterile water or normal saline solution for at least 20 minutes.
- Evert the eyelids, especially if plaster or cement to remove particulate debris
- Five to 10 minutes after stopping irrigation, touch dry litmus paper to the inferior cul-de-sac to test pH.
- Continue irrigation until pH is normalised

Minimum data set
Nature of chemical and duration of contact.
Extent and timing of previous irrigation, and that required to normalise the pH.
pH with litmus paper.
Visual acuity.
State of lids and adnexae.
Extent of conjunctival / limbal blanching (as clock hours or degrees)
Corneal epithelial loss (as approximate % of total corneal surface).
Clarity of stroma. Lens involvement. IOP.

> 50% epithelial loss
>90^0 of limbal blanching
Any stromal involvement

→ **admit**; inform on call SPR & Consultant.
All drops preservative free:
G chloramphenicol QDS, G predforte 1-2 hourly; G.Vitamin C drops 2 hourly; G citrate hourly; G cyclopentolate TDS. Systemic Vitamin C 1g po BD, Doxycyline 100mg po OD

<50% epithelial loss
< 90^0 limbal blanching
No stromal whitening

→ Rx g chloramphenicol QDS + G predforte QDS. **casualty follow up 24hrs**

For very mild cases/non alkali injuries with no limbal ischaemia or significant corneal involvement use G predforte and occ chloramphenicol QDS for 1 week, no follow-up unless persistent or deteriorating symptoms.

Ocular surface chemical injury severity - The Roper Hall Classification

Grade	Cornea	Conjunctival Limbus	Prognosis
I	Epithelial damage	No limbal ischaemia	Good
II	Hazy but iris detail seen	<1/3 limbal ischaemia	Good
III	Total epithelial loss, stromal haze, iris detail obscured	1/3 – 1/2 limbal ischaemia	Guarded
IV	Opaque. No view of iris or pupil	>1/2 limbal ischaemia	Poor

- **CS gas (***Give advice maybe no need to come to eye department*)

Wear gloves, apron and mask
Do not irrigate eye.
Put patient in well ventilated area and use a fan to evaporate residual chemical.
Remove contaminated clothing and put outside to air, or bag.
Main problem is lacrimation and can induce bronchospasm (consider medics).
IOP can be raised in short term.

Protocol for Main ED treatment of chemical injuries

Initial management
Triage for immediate assessment/treatment
Anything other than CS gas irrigate eye with at least 1000 ml of normal saline.

- Check & document the pH of both eyes before commencing
- Use topical anaesthetics liberally to facilitate irrigation and examination
- Irrigate the superior and inferior cul-de-sacs and remove any foreign bodies that may act as a depot for the chemical. Intravenous tubing connected to a container of normal saline makes an ideal irrigation tool. A lid speculum greatly facilitates irrigation.
- Continue irrigation with sterile water or normal saline solution for at least 20 minutes.
- Evert the eyelids, especially if plaster or cement to remove particulate debris
- 10 minutes after stopping irrigation, touch dry litmus paper to the inferior cul-de-sac to test pH. Testing any sooner would mean that irrigating fluid and not the tear film was being tested.
- Continue irrigation until pH is normalised (compare to the other eye if unaffected)

Referral
Each case should be considered on its merits with a high index of suspicion if industrial accident, assault, intoxicated patient with limited reliability, or high pH (>=8) on arrival.
Refer urgently:

- If prolonged irrigation (>2 litres) does not lead to normalisation of the pH. ***Further irrigation of these patients should not be interrupted but continued in the ED until either eye casualty or the on-call SpR is confirmed available and ready to receive the patient.***
- If clinical assessment indicates a severe injury with significant reduction of corrected visual acuity (with topical anaesthetic instilled and pinhole if glasses not available), widespread injection with staining epithelial loss.
- Careful examination to identify a severely blanched conjunctiva “white eye” or total corneal epithelial loss.

All other chemical injuries to be seen by ophthalmology the following day, in eye casualty Monday – Saturday (no need to telephone the on call doctor for permission), or ward 20 via the on call SpR on a Sunday.

CS gas (*Give advice maybe no need to come to eye department*)
Wear gloves, apron and mask. ***Do not irrigate eye.***
Put patient in well ventilated area and use a fan to evaporate residual chemical.
Remove contaminated clothing and put outside to air, or bag.
Main problem is lacrimation and can induce bronchospasm (consider medics).
IOP can be raised in short term – ophthalmology review next day if out of hours

Acute glaucoma
Gonioscopy of other eye can help with diagnosis. Consider other diagnoses to acute angle closure, i.e. Rubeosis, hypertensive AAU, drug induced
Treatment of acute angle closure glaucoma:
Admit under the consultant on call
Diamox I.V. 500mgs stat then 250mgs qds
Pilo 2% stat; one hour later, then qds
Predforte 2 hourly
Timolol 0.5% bd (unless contraindicated)
Unaffected eye: pilo 2% qds
If IOP not settled within 6-12 hours consider Mannitol I.V. 20% 2g/kg – ensure a complete medical clerking has been undertaken and cardiac risk factors taken into consideration. If necessary discuss with medical registrar on call.
YAG iridotomy to both eyes before discharge. Review at 24 hours – either yourself or make specific arrangements on call team. If the iridotomy is patent stop pilocarpine, continue predforte for ten days, and reduce other medication depending on IOP. Review SOON in consultant on call's clinic, or refer to glaucoma team at their discretion.
High Intraocular pressures (IOP)
Exclude risk of angle closure taking into account: AC depth, Van Herrick, goniscopy and refractive status. Ensure relevant tests (ie. visual fields) required are booked with follow-up.
Ocular hypertension (healthy looking discs):
IOP 22-30 & no risk of angle closure: Refer routinely to glaucoma diagnostic clinic or appropriate peripheral clinic.
IOP 30-35 or risk of angle closure: Refer routinely glaucoma consultant clinic
Open angle or secondary glaucoma: IOP 22-35
Obtain Humphrey VF & commence topical treatment if positive. Refer routinely glaucoma consultant clinic
ANY IOP >35: start topical treatment
If patient is young or IOP above 40: ***discuss with the EED registrar*****.** Do not send home with IOP over 40mm Hg. Refer to glaucoma consultant clinic for SOON follow up
Normal tension glaucoma: suspicious discs & IOP <22
Exclude vertical midline field loss with fields to confrontation. No treatment. Refer to routinely to glaucoma diagnostic clinic.

Glaucoma drop intolerance
Patients telephoning EED with questions or concerns about their drops can be forwarded to the glaucoma secretaries, they do not routinely have to attend EED or have their IOP checked.
Patients attending are instructed to stop the drop that is suspected to be causing topical irritation for 1-2 weeks. If necessary more than one drop can be stopped then each reintroduced individually to see which the offending medication is. Don't just add lubricants.
The patient MUST contact their consultants secretary to inform them of their medication problems and receive advice about further prescriptions or medication changes.

NEURO-OPHTHALMOLOGY

<u>To arrange an urgent scan Mon – Fri 9am – 5pm:</u>
contact the ATTENDING NEURORADIOLOGIST RVI DECT 23933 or ext 23648

Neuro-ophthalmology section covers:
Papilloedema
Optic neuritis
Transient monocular visual loss
Giant cell arteritis
Double vision

PAPILLOEDEMA
Papilloedema is bilateral optic disc swelling due to raised intracranial pressure. The visual function (visual acuity, colour vision and visual field) are NORMAL in papilloedema

History
Headache
Transient visual obscurations (blurring or blacking out of one or both eyes for *seconds*)
Pulsatile tinnitus

Examination: Blood pressure (exclude malignant hypertension)
Urinalysis
Visual acuity
Colour vision
Visual fields
Look for other neurological signs
Photography/OCT if available

NOTE: *VI nerve palsy can occur in raised intracranial pressure as a false localising sign*

Action
Patient needs a scan (a CT will do) same day to rule out space occupying lesion. **To arrange an urgent scan Mon – Fri 9am – 5pm contact the ATTENDING NEURORADIOLOGIST RVI DECT 23933 or ext 23648**
Refer to neurology – if CT normal will need MRI and LP
Consultant on call should be informed of ALL patients who require a scan or who are referred to neurology.

What to do if patient is <u>asymptomatic</u> and referred to ask for an opinion on whether discs are swollen

1. check visual function (acuity, colour vision and visual fields) is normal
2. check there are no orbital signs and no RAPD
3. look for optic disc signs of swelling
    a. hyperaemic disc

b. dilated capillaries on disc surface
c. thickened nerve fibre layer around disc, i.e. area of elevation extends across the disc margin to the peripapillary retina
d. haemorrhages or cotton wool spots on the disc
e. absence of venous pulsation (absent in 30% of the population anyway)
f. macular star of exudates

4. consider other tests to look for true disc swelling
    a. optical coherence tomography of disc on Cirrhus machine
    b. ultrasound (to look for disc drusen)
    c. fluorscein angiogram (to look for disc leak)

If in doubt ask consultant on call.

OPTIC NEURITIS

Optic neuritis is the term for any optic nerve inflammatory condition: it does not indicate the aetiology and can be due to demyelination, sarcoid, infection and autoimmune conditions amongst others. Of these, only demyelinating optic neuropathy will show spontaneous visual improvement after about 2 weeks and it is this improvement that confirms a demyelinating aetiology.

NOTE*: not all young adults with optic neuropathy have optic neuritis.*
A diagnosis of optic neuritis can only be made in retrospect once spontaneous visual recovery has started – it is best to tell the patient they 'probably have inflammation of the optic nerve' and need to be followed up in clinic to confirm the diagnosis. Optic neuritis and the link with MS can best be discussed in the clinic setting.

History:
Pain behind the eye, usually worse on eye movements
Acute visual loss: worsens for up to 2 weeks then stabilises. Spontaneous recovery starts at about 2 weeks
Visual photopsias may occur
Associated neurological symptoms or past history of neurological symptoms e.g. numbness, weakness of arm or leg, and vertigo

Examination:
Visual acuity (can be 6/6 to no perception of light)
Colour vision
RAPD testing
Visual field. If vision is worse than 6/18 do confrontation fields in the affected eye and formal perimetry in the unaffected eye.
Typical field defect is central scotoma but field loss can be of any type
Optic disc normal or mildly swollen
Dilated fundoscopy to exclude other cause of reduced vision

Atypical cases – consider **non**-demyelinating cause in any patient who has

1) severe pain
2) bilateral at onset
3) atypical fields e.g. hemianopic field defect
4) atypical age for first onset <16 or >50
5) very swollen disc (optic neuritis produces none or mild disc swelling)
6) macular star of exudates

Treatment

Over 90% of patients will regain vision of 6/12 or better
There is no treatment which will improve the final visual outcome
Steroids will hasten the visual recovery and should be considered in patients with very poor vision or who require good visual acuity for employment. Discuss with consultant on call if steroids to be used
ALL patients who are given steroids MUST have MRI to exclude other pathology – the natural history of the disease will be affected by the steroids.

Action

Patient should always be seen by ST3 Registrar or above
See consultant on call in 2 weeks then refer to neuro clinic -: vision should have started to improve by then – if it hasn't then patient needs MRI of optic nerves and brain with contrast.

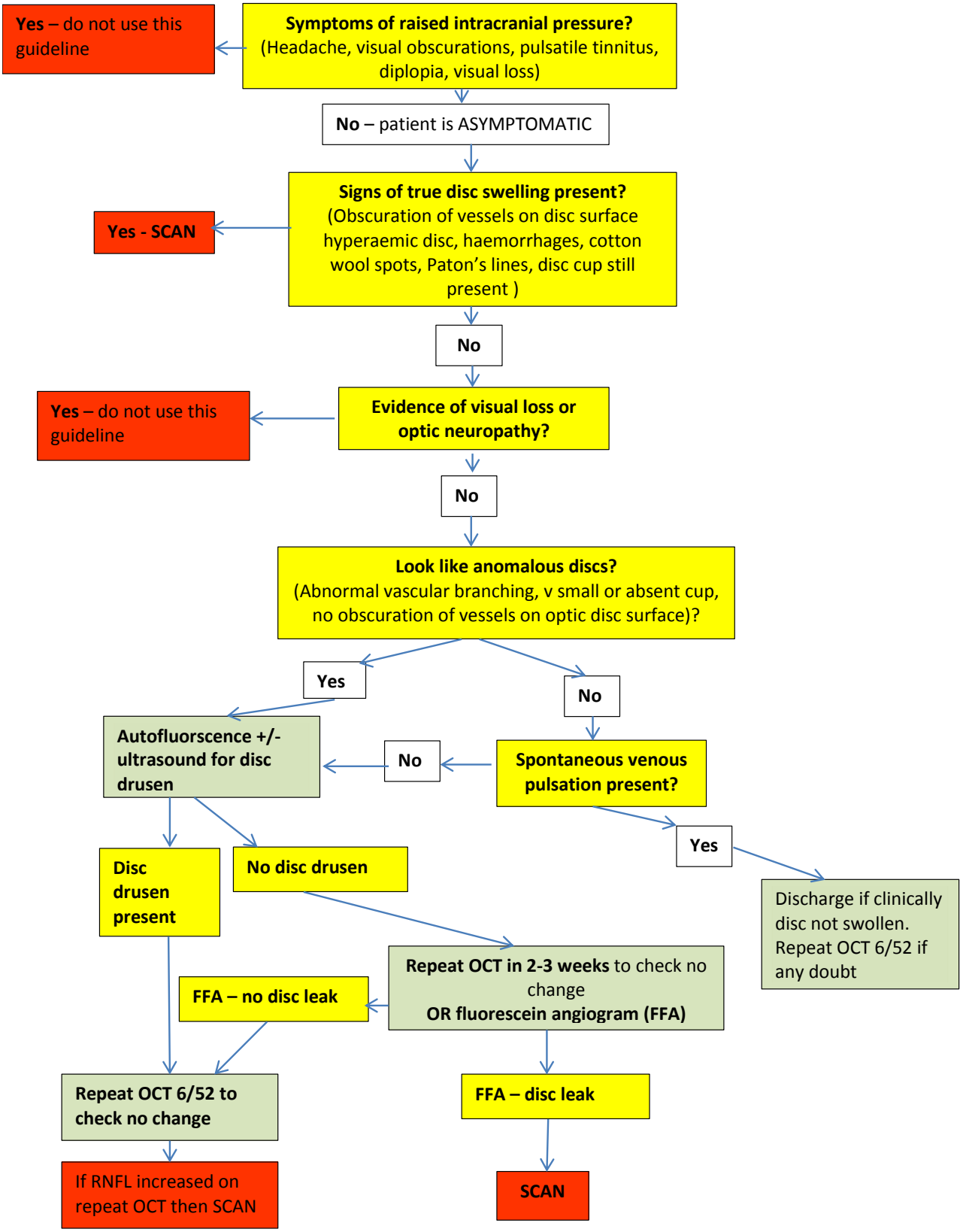
RAPID ACCESS NEUROLOGY CLINIC REFERALS

1. Discuss with neurology registrar on call to confirm appropriate referral
2. Email clinical details to: tnu-tr.neurology-secretaries@nhs.net

   Include a patient telephone number
3. Inform the patient that they will hear direct from the neurology department with an appointment, usually by telephone
4. Give the patient the follow up safety net leaflet of who to call if they haven't received an appointment within a specific timeframe

   amend the contact telephone number to *0191 2829316

GUIDELINE FOR MANAGEMENT OF
ASYMPTOMATIC PATIENTS WITH
EQUIVOCAL OPTIC DISC APPEARANCE
and **BORDERLINE OCT**
ie difficult to tell if there is mild disc swelling
NB this is a guideline only and does not replace clinical judgement



MONOCULAR TRANSIENT VISUAL LOSS (TIA) (AMAUROSIS FUGAX)

Causes

Embolic (from carotid arteries or heart)
Patients with transient monocular blindness are at risk of hemispheric stroke (3-6% per year) and premature death from myocardial infarction. We therefore have a duty of care to recognise and appropriately investigate such patients.
Vasculitic (especially giant cell arteritis)
(less commonly thrombophilia or carotid artery dissection)

History

Sudden onset of *severe* visual loss over seconds to 1 minute
Affects entire field of vision of the eye
Usually resolves fully within 10-15 mins (rarely lasts up to 1 hour)
No other neurological symptoms
Ask about giant cell arteritis symptoms
Loss of vision in both eyes is not due to emboli from the carotid artery and reflects posterior cerebral circulation problems
Transient monocular blindness in younger patients with break-up of the visual field into pieces and positive visual phenomena is not likely to be embolic.

Examination

Ocular exam including dilated fundoscopy – usually normal but may see retinal emboli
Pulse rhythm and rate (look for atrial fibrillation)
Cardiac & carotid examination (murmurs/ arrhythmia/bruits as source of emboli)

Investigation

Blood pressure
BM
Urgent ESR, CRP to exclude giant cell arteritis if clinically indicated
Full blood count, U+E and blood glucose if referred to the RVI TIA clinic (state on request form ‘referred to TIA clinic’)
If referred to non-NUTH TIA clinic then they will do the bloods themselves (as per TIA referral proforma)

Management

If giant cell arteritis likely then treat appropriately (see giant cell arteritis section)
If embolic cause likely:
a. Give oral Aspirin 300 mg once a day (maximum duration 14 days from the latest symptom onset) followed by 75 mg once a day until seen in TIA clinic with appropriate gastric protection if required.[1]
b. Refer urgently to TIA clinic according to instructions on TIA referral proforma.
c. Notify patients that DVLA rules state no one may drive within a month of TIA or stroke and for 1 year for HGV drivers who need to notify DVLA.

Reference:

1- https://www.nice.org.uk/guidance/cg68/chapter/1-Guidance#rapid-recognition-of-symptoms-and-diagnosis

GIANT CELL (TEMPORAL) ARTERITIS

Sight threatening but treatment can also have serious side effects so every effort should be made to clarify diagnosis.

A careful history is required as headaches are common.

History

General
- Age >50
- New headache
- Jaw claudication (jaw pain which develops with and gets worse on chewing)
- Scalp tenderness
- Symptoms of polymyalgia rheumatica
- Malaise, weight loss

Ocular
- Amaurosis fugax
- Double vision (transient or persistent)
- Visual loss

Examination
- Anterior ischaemic optic neuropathy (AION)*
- Central retinal artery occlusion artery occlusion
- Retinal cotton wool spots
- Signs of ocular or orbital ischaemia
- Ocular motility abnormality
- Tender temporal arteries

Cilioretinal artery occlusion or branch
- Scalp tenderness (rarely scalp necrosis)

*(**Non** arteritic AION is more common than arteritic, characteristics include: younger age group, vascular risk factors, altitudinal field defects, often less severe visual loss, crowded disc – check other eye: if disc not crowed then suspect arteritic.

Tests
- **ESR** (usually high but *can be normal therefore need to do CRP as well*
  (Remember other causes of high ESR: e.g. cancer, myeloma; infection; endocarditis)
- **CRP**
- **FBC** (normocytic, normochromic anaemia, high platelets)
- **U+E and glucose** (baseline tests as patient may need steroids)
- **Baseline blood pressure**
- **Temporal artery biopsy**: don't delay treatment to do biopsy. Can get a positive result up to 4 weeks after starting steroids, but the sooner the better. A positive result is very useful in guiding future treatment, as it rules out diagnostic doubt, and the patient may need to be on steroids for 2 years or more.

Treatment

GCA with AION is an emergency – risk of visual loss in 2nd eye within 24h

GCA with AION

1. Admit

2. iv steroids (e.g. iv methyprednisolone 250mg qds for 3 days then prednisolone 80mg po and slow taper over months. If methylprednisolone not available then use hydrocortisone 100mg iv). *START IMMEDIATELY*
3. aspirin 75mg od po unless contraindications
4. lansoprazole 30mg od po
5. bone protection

GCA without visual symptoms but with jaw claudication
Prednisolone 60mg po

GCA without visual symptoms or jaw claudication
Prednisolone 40mg po
GCA without any ophthalmic symptoms should not usually be accepted to EED & needs to be referred to rheumatology for management

Consultant on call should be informed of all patients commenced on steroids or requiring a temporal artery biopsy for suspected giant cell arteritis.

Precautions with steroids:
Monitor Blood pressure and BM
CXR to rule out old TB
Dipstick urine +/- MSU
Advise against smoking, increase Vit. D, calcium
Low sugar diet.
Rheumatology referral for osteoporosis management, follow up, bone densitometry etc.

GCA rheumatology referrals

Specific referral forms can be found on the intranet under (use this scan whether duplex specifically required or not)

Clinical Directorates>Musculoskeletal>services Rheumatology
GCA Fast Track Path Referral To Rheumatology for TA Duplex Scan
Or use the hyperlink:
http://nuth-intranet/cms/ClinicalDirectorates/Musculoskeletalservices/Rheumatology.aspx

1. UPLOAD this referral form to the eRecord Document Store via the upload tool http://documentstore.app/uploaddocument

2. Let the Rheumatology Department know that there is a referral waiting in eRecord, by EMAIL to Karolyn Houghton on Karolyn.houghton@nhs.net AND by PHONE to the Rheumatology on-call registrar on Dect Phone 39964.

DIPLOPIA

MONOCULAR DIPLOPIA: Persists with monocular occlusion
Cause is ocular or refractive

Refer to clinic appropriately or discharge

BINOCULAR DIPLOPIA: Due to abnormal ocular motility – see below

Double vision can be due to disease of:

Extraocular muscles (inc myasthenia, myositis)
Orbit (inc thyroid eye disease, space occupying lesion, inflammatory)
Cranial nerves
Brain

History

Monocular or binocular diplopia
Sudden or intermittent onset
Change with gaze direction or distance? (horizontal diplopia for distance is VI palsy unless proven otherwise)
Diurnal variation?
Pain?
Associated symptoms – headache, neurological symptoms
Past history: vascular risk factors, cancer, smoking

Examination:

Visual acuity
Eye movements
Look for anisocoria (dilated pupil in III palsy suggests aneurysm, Horner's syndrome in VI palsy suggests cavernous sinus lesion)
Ptosis (III palsy, myasthenia, Horner's syndrome) or lid retraction
Orbital signs
Cavernous sinus signs: Horner's syndrome, reduced corneal sensation
Cranial nerves (including corneal sensation)
Visual field to confrontation
Ocular examination
Dilated fundoscopy

Blood pressure
BM
ESR and CRP for giant cell arteritis if indicated
Orthoptic assessment if diagnosis uncertain

Always consider the possibility of giant cell arteritis

Further management
ALL patients with diplopia should be seen by ST3 Registrar or above.

Blenderm one lens of patient's glasses (orthoptics can supply plano spectacles with blenderm if required).

Patients should be warned not to drive with one eye occluded until they have had a 'period of adaptation' (usually 6 weeks) and not to drive with double vision

Consultant on call should be informed of ALL patients referred for scan or neurology opinion.

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- **3rd Nerve palsy:** 10-20% due to aneurysm
  III palsy cannot be said to be pupil sparing unless it is a COMPLETE III ie TOTAL palsy of SR, IO, IR and MR.
  Pupil involvement or pain suggests aneurysm

ALL III palsies need an MRAngiogram or CTAngiogram scan the same day whether pupil sparing and painful or not (this has been agreed with the clinical director of neuroradiology Jan 2012). To arrange an urgent scan Mon – Fri 9am – 5pm contact the ATTENDING NEURORADIOLOGIST RVI DECT 23933 or ext 23648

- **Microvascular 3rd, 4th or 6th nerve palsy (unlikely under 50yo)**

  FBC, U+E, glucose, cholesterol, ESR
  Start aspirin 75mg od po unless contraindicated
  Refer to adult motility clinic with OD for follow-up within 1 month

- **Diplopia plus disc swelling or other neurological involvement**:

  Discuss with Consultant on call.
  Consider referral to Neurology unless obvious orbital cause.

- **Bilateral sixth nerve palsies** – consider raised intracranial pressure.
- **Check blood pressure + urinalysis (exclude malignant hypertension)**
- **Discuss with neurology**

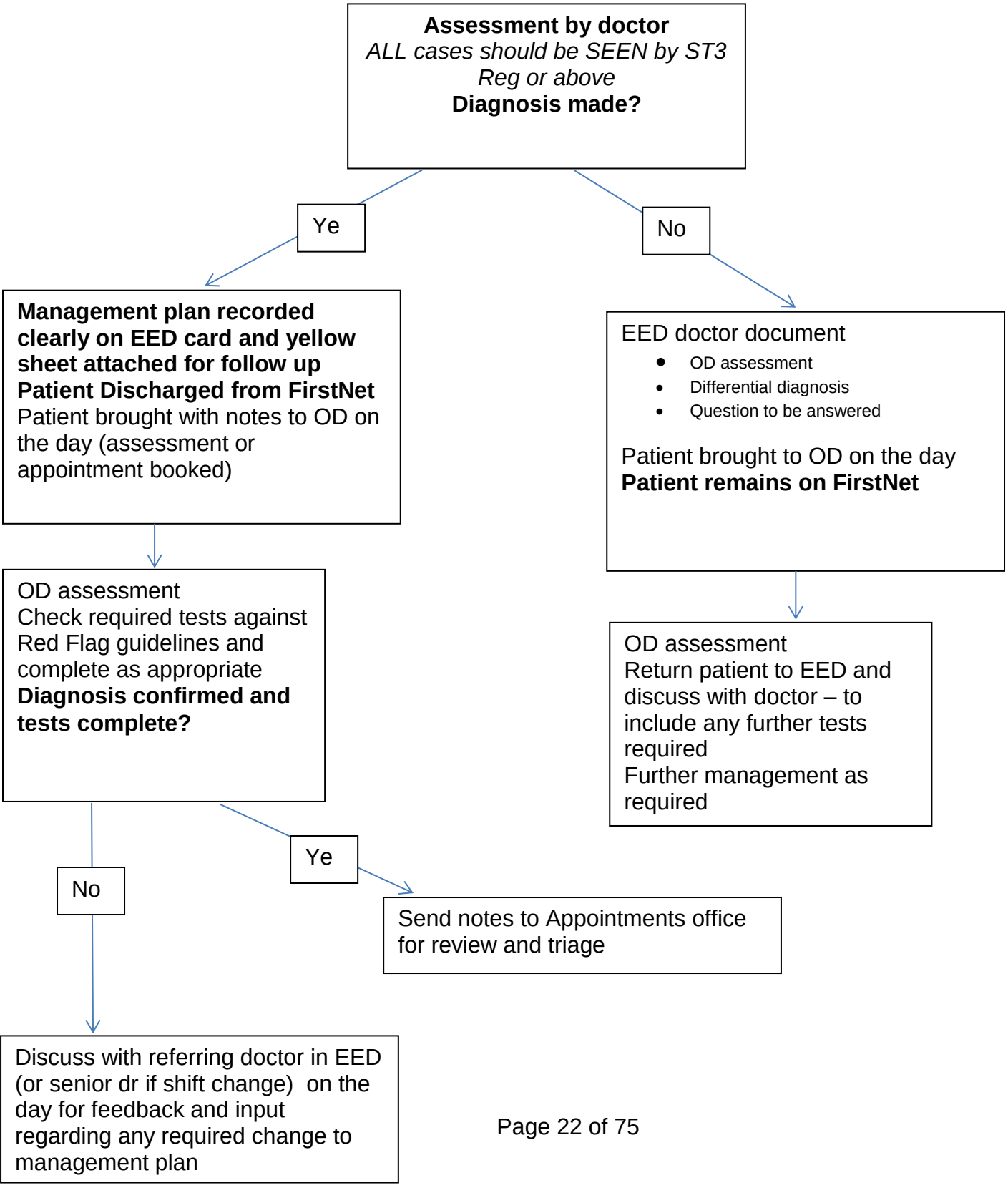
ALL cases of binocular diplopia should be SEEN by ST3 Registrar or above

Guidelines for orthoptic referrals from EED

Assessment of diplopia

There is no standing arrangement for patients with double vision to be sent straight to Orthoptics without assessment by a doctor first.
ALL cases of binocular diplopia should be SEEN by (not just discussed with) ST3 Registrar or above

Patient Pathway for Binocular Diplopia in EED



Children's VA assessment

- Pre-verbal infants
- Young children whose assessment is difficult or compromised in EED

(Please note that we have Kays pictures for testing pre-literate children's VA in EED)
Note vision assessment may not be necessary in all, eg babies with sticky eyes

Blow-out fractures/max-fax referrals

Max-fax refer directly to OD for assessment on their pro-forma. As part of this Max-Fax require a dilated fundus examination. Every effort is made to incorporate these requests into a doctor-led clinic however; occasionally this will need to be completed by an EED doctor.

For blow-out fractures that present urgently to EED, likely to require surgical intervention, every effort will be made to see these in OD to complete pre-operative assessment on the day.

PTOSIS

Can be due to disease of: Eyelid/ levator
Myasthenia/ myopathies
III nerve
Brain
Horner's syndrome – brain/ neck/ upper chest

History

Ptosis: Duration
- Constant or intermittent?
- Diurnal variation/ absent on waking? (myasthenia)

Associated symptoms:
- Double vision
- Pain
- Myasthenic symptoms (shortness of breath, limb fatiguability, bulbar symptoms such as problems swallowing or chewing or change of voice on talking)

Past history: Trauma or recent head or neck surgery (carotid dissection/ Horner's)
- Cancer

Examination

Visual acuity
Eye movements (myasthenia, III Palsy)
Anisocoria (III palsy, Horner's syndrome)
Ptosis: measurement
- Levator function
- high skin crease? (aponeurotic ptosis)
- fatiguability (myasthenia)
- orbicularis oculi weakness? (myasthenia)

Orbital signs

Management

Depends on suspected diagnosis

Suspected myasthenia gravis – discuss with neurology
Third nerve palsy – see third nerve section above
Horner's syndrome –
Apraclonidine 1% test to confirm diagnosis: instill in both eyes, remeasure pupil size after 45 mins. Apraclonidine dilates Horner's pupil but not normal pupil. May be negative in very recent onset Horner's syndrome.
- **Horner's syndrome with neck or facial pain** – suspect carotid dissection and refer neurology urgently – needs MRangiography.
- **Horner's syndrome with VI palsy** – suspect cavernous sinus lesion: requires imaging.
- **Isolated Horner's syndrome** – discuss with neuro-ophthalmology or neurology re: appropriate investigation

Oculoplastics

This section covers:
Acute orbit
Peri-ocular lesions
Lid malposition
Lacrimal
Orbital fractures
All eyelid / conjunctival lacerations and orbital injuries

Acute orbit

History:
- Symptoms of acute infection, fevers, rigors
- History of sinusitis
- Any history of thyroid dysfunction or systemic symptoms associated
- Previous history of orbital inflammatory disease
- Systemic symptoms of Wegeners / vasculitis
- Known systemic malignancy esp. lymphoma

Examination:
- Document soft tissue involvement – mark extent if suspicion of cellulitis and document any necrosis, fluctuant swelling, crepitus or skin anaesthesia
- Ptosis or lid retraction
- Extraocular movements
- Optic nerve function: Acuity, RAPD, colour vision
- Exophthalmometry
- Auscultate if any signs of CCF

Investigations:
- Systemic observations – temperature, BP, pulse
- FBC, ESR, CRP, U&Es and consider TFT+ TBII if suspicion of TED
- CT orbits with contrast

Action:
- Discuss with the EED registrar
- Consider admission
- If suspected orbital cellulitis follow protocol in paeds section
- Do not give steroids without consultation with oculoplastics consultants unless emergency situation with optic nerve compromise
- Consider NSAIDS whilst awaiting test results/definitive diagnosis
- **Urgent** discussion with oculoplastics consultants

Peri-ocular lesions

History:
- How long have they noticed ANYTHING at that site?
- Is it growing? Slowly or fast?
- Any pain / tenderness / anaesthesia
- Medications – any aspirin /clopidogrel / warfarin

Examination:
- Palpate and stretch skin to assess **true** size then measure
- Relation to other structures, i.e. lacrimal drainage, lid margin
- Evidence of tissue destruction – loss of lashes, cicatricial ectropion
- Evidence of deep fixation or orbital involvement (e.g. restricted eye movements)

Action:
- Photograph if at all possible

Definite chalazia, small skin tags etc refer NP cyst list, (but remember BCCs frequently cystic)
BCC refer to oculoplastics routine (AJD or EB)
Any other lesions suspicious of malignancy discuss **urgently** with oculoplastic consultant (remember 2 week rule). Take patient telephone number and explain they may require a biopsy.

Lid malposition
Lower lid
Only refer if the patient is symptomatic and keen for surgical intervention
- **Routine** referral to oculoplastic clinic

Entropion: if the cornea is severely affected, give lubricants, advice about taping and consider referral to **next available** Botulinum clinic, as well as oculoplastics clinic.
If cornea no significant concern then refer to oculoplastics clinic or discuss with team for prompt surgery (ask if patient is on warfarin or anti-platelet treatments) NB. Short waiting list: most patients do not need botox.

Upper lid
See neuro-ophthalmology section to exclude neurological causes. Ask about diplopia.
Evert upper lid and palpate orbit to exclude a mechanical cause.
Aponeurogenic ptosis: slow history; good levator function; high skin crease; often have evening fatigue
- **Routine** referral to oculoplastic clinic

Lacrimal
Acute: treat ocular surface or allergic symptoms.
Chronic: not appropriate for EED management or investigation (ie syringing)
- **Routine** referral to lacrimal clinic or LC oculoplastic clinic

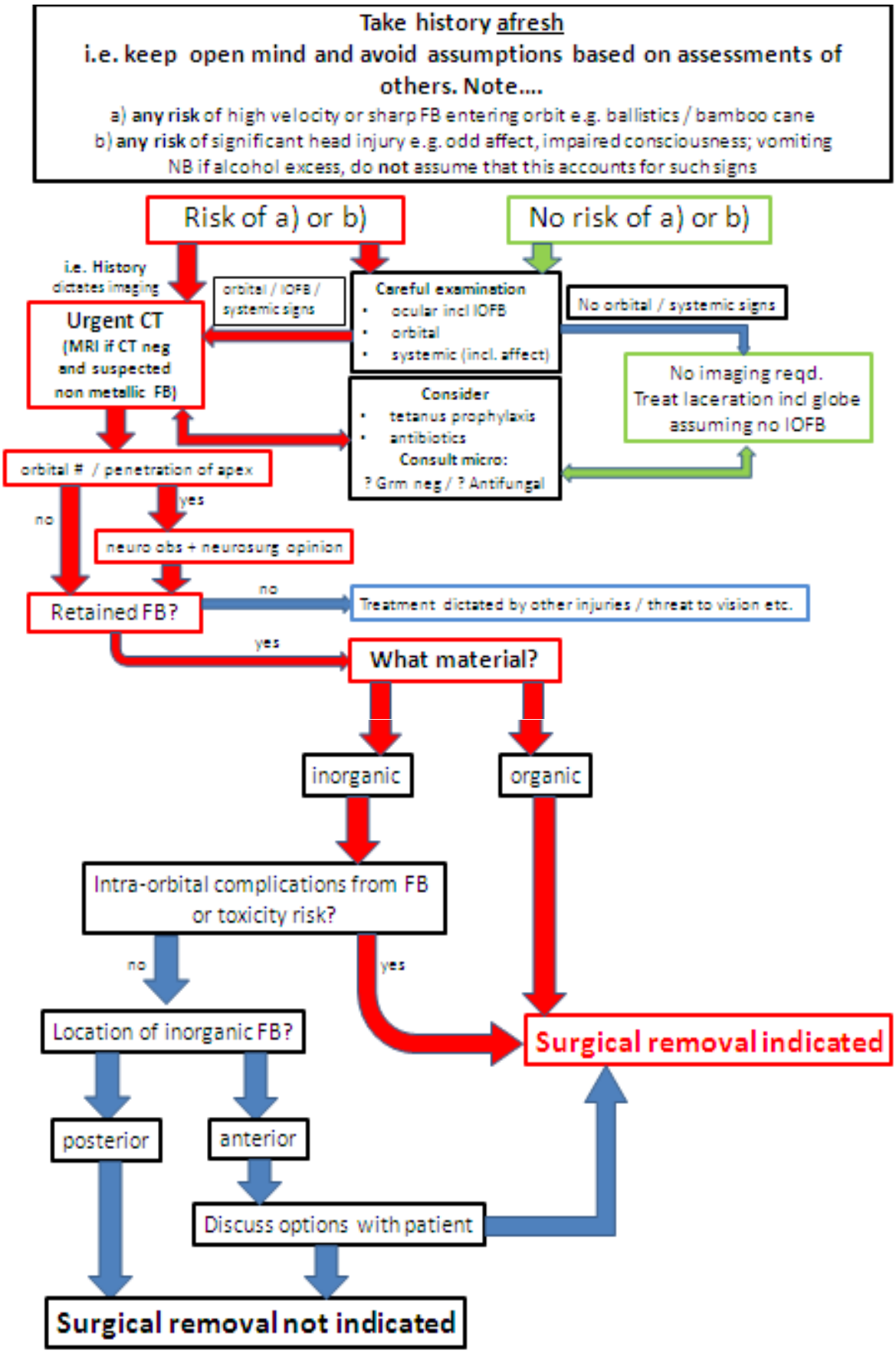
Dacryocystitis: treat with warm compresses and oral antibiotics (co-amoxiclav 325mg TDS 10 days if not allergic)
- Soon referral to lacrimal clinic or LC oculoplastic clinic. Treatment of choice is an endoscopic DCR so notes to consultant ASAP

Lacrimal gland swelling:
Unilateral or bilateral?
Any signs of infection?
Dacroyadenitis – treat with oral antibiotics (co-amoxiclav 325mg TDS 7 days if not allergic)
Inflammatory – investigations for sarcoid: CXR and serum ACE
Neoplastic – consider lymphoma
- **Soon** referral to oculoplastic clinic

Orbital fracture
Examination:
Restricted elevation
Enophthalmos
Surgical emphysema
NB. Severe pain or gaze evoked vasovagal symptoms esp in young person requires urgent surgery – discuss with max fax.
Action:

Orthoptic assessment if available
Fundus examination
Maxfax referral if other facial fractures found/suspected (eg. zygoma)
CT orbits if significant ocular motility restriction or enophthalmos, or child with vaso vagal symptoms
- **Soon** referral to LC oculoplastic clinic with OD on arrival

Protocol for management of ALL eyelid / conjunctival lacerations and orbital injuries



Eye Emergency Department (EED) Guidelines : Paediatric Section

Neonatal Conjunctivitis (Ophthalmia Neonatorum)

Defined as conjunctivitis occurring in the 1st month after birth.

History taking to include:

Method of delivery
Onset of symptoms (N Gonorrhoea more likely 1st 24 – 48 hours post-partum)
(Chlamydia 5 – 14 days post-partum)
Child's general health – feeding/ respiratory / temperature
Parental GU symptoms

Symptoms Purulent discharge
Conjunctival chemosis
Lid swelling

Management

Swab both eyes – send for Gram stain, M, C + S including Chlamydia for PCR
Check temperature – assess general state of child (Chlamydial pneumonia)
Discuss with paediatrician on call

Urgent microscopy to exclude N Gonorrhoea

If gonorrhoea +ve refer directly to paediatricians for IV therapy
Else treat with po erythromycin suspension 12.5mg/kg/day qds for 14 days.

Parents to attend GUM clinic if swabs positive
Chlamydial conjunctivitis is a notifiable disease

Follow-up

Refer **SOON** to paediatric ophthalmology clinic

Orbital Cellulitis

In preseptal cellulitis, the infection is limited anterior to the orbital septum. True orbital cellulitis implies infection that has penetrated the septum. The distinction needs to be made as orbital cellulitis has potential life threatening sequelae.

History to include:	Preceding anterior lamellar lesion or trauma (source) Fever Headache Sinus problems Surgery / trauma
High risk features:	State of health, feeding, drowsiness Immunosuppression
Signs	Decreased visual acuity Reduced optic nerve function (colour vision/pupils) Lid swelling and erythema Conjunctival injection / chemosis Restricted EOM movement/diplopia Proptosis

Management

Check temperature
Assess optic nerve function – VAs, RAPD, Ishihara (paediatric/innumerate charts available in orthoptics), Field to confrontation
Assess eye movement
Assess for proptosis
Fundal examination - assess optic disc

If pre-septal cellulitis suspected – treat with po Augmentin review in eye cas next day or SOS sooner. Consider ciprofloxacin and clindamycin if penicillin allergic

If orbital cellulitis suspected (or pre-septal cellulitis not responding to po therapy)

Requires admission for IV Abx,
Contact paediatricians/ ENT
CT orbit, paranasal sinuses and immediate brain parenchyma
FBC, U+E, ESR, CRP, Blood cultures

Follow up
Inform consultant on call

Paediatric Leucocoria

Defined as a white pupil. Variety of causes at different ocular levels.
The most important cause to exclude is retinoblastoma.
Other potential causes include corneal opacities, cataract, uveitis, vitreous opacities, PHPV, Coats' disease, retinal detachment

History taking
- Duration
- Family history
- Maternal health during pregnancy - TORCH
- Parental concerns about vision – note may be asymptomatic
- Other medical problems – Down's / metabolic disorders

Signs
- Leucocoria
- Absent / decreased red reflex

Management

Assess both eyes – may be bilateral
Assess for RAPD – cataract does not cause an RAPD
Assess for other ocular abnormalities
Determine at what ocular level leucocoria is present – e.g. lens / retina / cornea

Dilated fundal examination mandatory (Age <6/12 use cyclopentolate 0.5%, else 1%)
May need speculum examination
If retinoblastoma suspected **immediate** referral to consultant
Else prompt referral to paediatric ophthalmologist

Follow up

Urgent paediatric ophthalmology clinic
IF THERE IS ANY SUSPICION OF A TUMOUR OR FUNDAL LESION YOU MUST SPEAK TO THE PAEDIATRIC CONSULTANTS OR CONSULTANT ON CALL BEFORE THE CHILD LEAVES THE HOSPITAL.

Paediatric Glaucoma

Elevation of pressure associated with optic nerve damage. 80% of primary congenital glaucoma presents within 3/12.

Suspicious symptoms in history taking

	Epiphora Photophobia Rubbing of eyes Enlargement of eyes Comparison of photos if possible Family history Aphakia / Intraocular surgery e.g. cataract extraction
Signs	Blepharospasm Corneal enlargement / opacification, Haab's striae Buphthalmos (large eyes, thin sclera) Optic disc cupping Reduced acuity

Management

Urgent
Attempt IOP measurement – optician can undertake non contact tonometry
Diamox 5mg/kg/qds, **do not** use brimonidine (respiratory depression)

Follow up
Urgent paediatric ophthalmology clinic for consideration of surgery – discuss with consultant

Acute sudden onset squint

Most commonly esotropia. Need to exclude nerve palsy (commonly VI) and fundal pathology.

History taking
- Birth / peri-natal history
- Family history
- Actual duration of symptoms
- Evidence of longstanding squint
- Constant / Intermittent symptoms
- General health – temperature, rash, vomiting, drowsiness, poor feeding, headache

Signs
- Eye misalignment
- Limited eye movements
- Pupillary assymetry
- Ptosis

Management

Examine eye movements – cover test if possible
Orthoptic assessment if unclear
Assess pupillary reactions
Cycloplegic refraction
Dilated fundus examination – assess for papilloedema, retinal pathology

If cranial nerve palsy / papilloedema – liaise with paediatrician
Urgent imaging – see neuro-ophth guidelines
Inform consultant on call

If fundal pathology deal with as appropriate

Conjuctival / Corneal abrasion including chemicals

Depending on the age of the child determine VA if possible. Try to find out the nature of the substance causing the abrasion / burn.

History taking
- Witnesses (especially in case of preverbal children)
- Nature of substance (acid / alkaline, liquid / solid / gas)
- Duration of symptoms

Signs
- Lid involvement (chemosis, burn)
- Intense photophobia + blepharospasm
- Corneal involvement (epithelial, stromal)
- Conjuctival adhesions / scarring
- Anterior chamber inflammation

Management

Examine anterior segment (may prove challenging)
Determine extent of epithelial defect / burn with fluorescein staining
Assess pupillary reaction
Dilated fundus exam
Involve consultant on call if extensive chemical burn and /or signs of limbal deficiency to consider admission (liaise with Paediatric ophthalmologist)
Involve Paediatric ophthalmologist if examination difficult and / or unsure of findings
Treatment and follow up according to extent and cause of damage (see corneal guidelines) *remember drops can be quite difficult to instil in a child in pain*. No need to routinely follow up simple corneal abrasions – clear SOS advice

Ocular trauma: Penetrating + Perforating, Blunt trauma

It is very important to determine and document VA asap since it is the most reliable indicator of visual prognosis. Orthoptists are available to help in hours
Be aware of child abuse on every case of child trauma if the history and physical finding do not fit.

History: Dynamics of trauma
Type of objects causing trauma (sharp / blunt)
Potential for penetrating injuries (projectile)
Possibility of contamination
Eye problems prior to trauma (amblyopia, squint ect)

Signs: Proptosis
Enophthalmos
Eyelid involvement (oedema, laceration, ptosis)
Restriction of eye movements
Corneal / scleral laceration
Hyphema
Shallow anterior chamber
Pupillary reaction
Dislocated lens
Vitreous haemorrhage

Management

Asses both eyes (often there is damage to both)
Penetrating + perforating trauma involve consultant on call (admit)
Blunt trauma with hyphema and raised IOP may need admission depending on age of child (involve consultant on call)
A dilated fundus exam is mandatory in all cases B scan should be performed in those cases when fundus evaluation is not possible because of media opacities (hyphema, cataract, vitreous haemorrhage)
Gonioscopy should be performed before fundus dilation comparing to the other eye

Follow Up

Depending on severity of trauma may be appropriate to involve paediatric ophthalmologist (seek consultant on call advice)

Post-operative squint surgery

Most of the cases will be post-operative inflammation but be aware of potential complications such as granuloma or cyst formation. Preseptal or orbital cellulitis is rare but well documented. Anterior segment ischemia is uncommon but still possible especially in redo operations. Endophthalmitis is extremely rare in squint surgery but has the same devastating effect as in intraocular procedures.

History	Systemic symptoms (fever, malaise) Temporal relation of symptoms onset to squint surgery Sudden onset of diplopia following initial satisfactory outcome
Signs	Lid swelling Conjuctival chemosis Eye misalignment disproportionate compared to type of surgery Ciliary injection AC inflammation

Management
Involve paediatric ophthalmologist in all cases of serious complications above mentioned. A slipped or lost muscle is easier to fix the sooner the patient is brought back to the operating theatre. However muscles can slip even many years after squint surgery. Hence temporal relation of symptoms points to the correct level of urgency for every individual patient.

Suspected NAI retinal screening

The referring paediatrician contacts the ophthalmology registrar on call, they see the child and if they are happy they have performed a satisfactory examination and there are no haemorrhages, no further action is taken.

If there are haemorrhages or the examination is incomplete, then they contact Mr Clarke or Mr Shafiq either that day, or the next working day and they will review and arrange imaging if necessary. Any positive findings are telephoned to the referring consultant.

UVEITIS IN ADULTS

Practical EED AAU management guide
Please see the full uveitis guidelines for further details

Assessment

Document grade of inflammation, pupil/synechiae after dilation, IOP & and fundus findings on every patient.

Treatment

Fresh synechiae need to be broken in EED if at all possible. Intensive dilating drops (handed over to the co-ordinator), local warming and subconjunctival injection are available. If the patient has to go home before the dilation is completed then they should be seen again within 24hrs to assess the result.

Hourly predforte 1% plus cyclopentolate 1% for 3-7 days until significant improvement in symptoms. Can be adjusted according to the severity of the presentation but caution regarding underestimating & undertreating, particularly with known or suspected HLA B27positive patients.

The cyclopentolate can be halted and the steroids start to be weaned once the hallmarks of acute inflammation, of pain and redness have resolved. If the pain and redness doesn't resolve or reoccurs the patient should re-attend EED rather than attempt to manage their drops.

The steroids are normally weaned over 1-2 months. A suggested regime that is easy for the patient to remember is to halve the dose every week. Give all patients an information leaflet with their drop regime on the back with safety net advice.

Follow up

Not everyone has to be followed up. We have a nurse-led uveitis follow up clinic weds PM for those that do, with booked appointment slots – this is preferable that asking patients to re-attend EED.

It is ideal to follow-up newer sufferers, rebound cases or anyone who has had problems with compliance or understanding. Patients with a good understanding of their disease, no history of complications, who understand what to expect for recovery can be given written instructions on the clear understanding that if they are not progressing as expected to return to EED for further assessment.

General Comments about Terminology & Diagnosis

Uveitis refers to the presence of intraocular inflammation. Every effort should be made to establish a specific diagnosis, though this may not always be achieved at the first visit. A specific diagnosis will inform on prognosis, guide management, avoid mis-management, and establish a relationship (if any) to systemic disease.

The anatomical and clinical classifications of uveitis are the cornerstones of diagnosis.

ANATOMICAL CLASSIFICATION

This defines the **predominant** anatomical location of inflammation in the eye.

- Anterior uveitis: Anterior chamber
- Intermediate uveitis: Vitreous cavity
- Posterior uveitis: Retina and / or choroid
- Pan-uveitis: All above locations equally involved

CLINICAL CLASSIFICATION

This defines the clinical association or aetiology of the inflammatory process.

- Non-infectious with no known systemic association (e.g. isolated AAU; birdshot retinochoroiditis)
- Non-infectious with known systemic association (e.g. ankylosing spondylitis; sarcoidosis)
- Infectious (eg ARN; TB)
- Masquerade – can be non-malignant (e.g. chronic RD) or malignant (e.g. intraocular lymphoma)
- Medication induced (e.g. rifabutin)

Please refer to SUN 2005 definitions / terminology / grading in uveitis

DOCUMENTATION

A thorough history and examination are essential, and a meticulous documentation of the findings at the first visit is crucial in the process of diagnosis. It is often at presentation that clinical signs are at their most meaningful, before time and treatments alter them. Lack of good documentation at this stage cannot be rectified later – so your notes are vital.

Good drawings speak volumes. Please include presence of globe injection; the character & distribution of KPs; other corneal changes where relevant; AC cells & flare, fibrin or hypopyon; the state of the iris and pupil, lens changes; the state of the vitreous cavity; and a fundus drawing.

New presentation (i.e. new patients or existing patients with first casualty visit with flare up of symptoms) should be assessed before tonometry and mydriasis in order to properly document all anterior segment findings (e.g. redness, corneal sensation, iris details, pupil size & shape, posterior synechiae) should be assessed before tonometry and mydriasis in order to properly document all anterior segment findings (e.g. redness, corneal sensation, iris details, pupil size & shape, posterior synechiae) as well as after mydriasis to document persisting posterior synechiae and to allow for examination of the posterior segment.

RECURRENT UNILATERAL (NON-GRANULOMATOUS) ACUTE ANTERIOR UVEITIS ("AAU")

Acute anterior uveitis (AAU) that is unilateral at presentation and non-granulomatous is the commonest form of uveitis in adults and is strongly associated with HLA B27 antigen. **We use the term AAU to refer to a particular clinical syndrome described below, rather than its strict semantic definition.**

Features of the syndrome of Acute Anterior Uveitis

1. Anterior uveitis
2. Acute course
3. Unilateral
4. Onset with redness, discomfort, pain, photophobia
5. Recurrent unilateral attacks (affecting either eye)
6. Age at onset of 1st attack < 40 years
7. No granulomatous features (KPs no more than dust, no iris nodules)
8. Posterior synechiae, anterior chamber fibrin, or hypopyon may be present in severe attacks
9. Significant vitritis, cystoid macular oedema, or disc swelling may occur in severe attacks
10. Ankylosing spondylitis or another HLA B27 associated disease (e.g. psoriatic arthritis, Crohn's disease, ulcerative colitis, Reiter's disease) present in only about 50% cases
11. Clinical features (ocular or systemic) do not suggest another diagnosis

Explanations

Anterior Uveitis

The initial and primary location of inflammation is in the anterior chamber. In severe inflammation, vitreous cells and vitreous haze may be observed but this is not the initial or primary focus of inflammation. Optic disc swelling and cystoid macular oedema may occur in severe disease. Posterior uveitis (retinitis, choroiditis, or retinal vasculitis) is not consistent with “AAU” and suggests a different diagnosis. **Note: Dilated examination of <u>both</u> eyes is essential to exclude posterior segment inflammation.**

Acute course
An acute attack is one that is sudden in onset (developing over hours to days) and limited in duration (usually less than 3 months). The onset is characterised by discomfort, pain, photophobia, and redness and is soon followed by blurred vision. Untreated, the symptoms may escalate rapidly over 1 – 3 days.

Unilateral
The vast majority of attacks are unilateral at onset, but subsequent attacks may affect either eye. The fellow eye may become involved before the inflammation in the first eye has settled, but bilateral simultaneous involvement is rare, and should raise suspicion of alternative diagnoses.

Recurrent unilateral attacks affecting either eye
The disorder is characterised by repeated episodes of AAU, separated by periods of inactivity without treatment. Chronic uveitis (i.e. persistent uveitis greater than 3 months duration with prompt relapse after discontinuation of therapy) is rare in this disorder and should raise suspicion of alternative diagnoses. It is not possible to predict the timing of an attack or the natural history of a given attack. There are no clear precipitating factors and no disease modifying therapy is routinely available to reduce attack frequency.

No granulomatous features
Keratic precipitates are fine (no larger than dust and cannot be easily individualised). Medium, large, mutton fat or stellate KPs do not occur in this condition, and nor do iris nodules.

Age at onset of 1st attack < 40 years
The majority of patients present with their first attack between 16 – 40 years. The range in this department for presentation with a first attack is 10 – 82 years but such outliers are rare.

Up to 80% of patients with the syndrome of AAU are HLA B27 antigen positive (depending on study), and a significant minority of these have HLA B27-associated systemic disease, most commonly ankylosing spondylitis. Some of these patients will already be aware of this. For the remainder, the early identification of such disease and its treatment may substantially affect their quality of life and long term outcome. An important role of the ophthalmologist is to identify associated systemic disease in

these patients and to arrange appropriate referral (refer to spondyloarthritis screening tool in eye casualty).

TREATMENT – General Principles

There is a tendency to under-estimate the severity of attacks, and a tendency to under-treat attacks even when severe. A casual approach to treatment even when the severity at the first visit appears to be mild, can lead to inadequate control of inflammation.

Be aware of:

- The inherent nature of HLA B27-associated AAU is rapid escalation of severity over 1 -3 days (it is not uncommon for a transformation from mild uncomplicated disease to blinding and painful disease to occur in this time frame). Occasionally this escalation occurs later.

- The general principal therefore is to treat aggressively early, observe improvement, and only then to slowly taper treatments.

- Persisting redness, photophobia, pain, involuntary eye closure – these usually indicate problems with uncontrolled inflammation in the absence of another confirmed explanation.

- 1st attack patients. The first attack is frequently the most severe and prolonged for several reasons (late presentation, unfamiliarity with drop instillation, tendency to relax therapy when symptoms better). Greater vigilance required in these patients.

- Poor compliance or likelihood of poor compliance. Consider medical and social factors that may lead to difficulties with self-medication. Greater vigilance required in these patients. Consider ward admission (see below)

Topical & Sub-conjunctival Corticosteroid

Intensive (hourly, sometimes more frequently) topical steroid using a strong preparation such as g.prednisolone acetate 1% (g.PredForte 1%) is essential in the early stages. If compliance with this very frequent regimen cannot be guaranteed or if the attack is very severe at onset, sub-conjunctival corticosteroid (e.g. betamethasone 4mg or dexamethasone 4mg), repeated if necessary, is important.

Topical Cycloplegic Mydriasis

The importance of this cannot be overstressed. Effective cycloplegic mydriasis achieves pain relief and will shorten the duration of an attack. The persistence or progressive accumulation of posterior synechiae is associated with several complications (persistent inflammation, macular oedema, cataract, glaucoma, complicated future cataract surgery). Every effort should be made to produce maximal mydriasis at the first visit, and the state of the pupil and the end of the consultation should be documented.

In the acute situation, patients who have not responded to one application of topical mydriatic should be given intensive g.cyclopentolate 1% and g. phenylephrine 2.5% (one drop of each every 5 minutes for up to 30 minutes). Placing the patient supine or semi-recumbent, keeping the lids closed in between drops, the application of local heat over closed lids, and pressure over the lower punctum and canaliculus will help.

Note: g.phenylephrine 10% is specifically contraindicated in children, elderly patients, and patients with hypertension or other cardiovascular disease. It has significantly greater systemic side-effects and offers little advantage over 2.5% as a mydriatic.

Sub-conjunctival Mydricaine

Use if intensive topical mydriasis fails to achieve good mydriasis. The injection can be repeated at daily intervals if required.

Mydricaine No 2 is the formulation for adults, aged 16 – 75 years. It comes as 0.5ml ampoule. It contains atropine 1mg, adrenaline solution 1 in 1000 0.12ml [=0.12mg], and procaine 6mg.

Mydricaine No 1 is the formulation for children and adults over 75 years. It comes as 0.5ml ampoule and contains the same constituents at half the doses.

Acute and transient anxiety, tremor, pallor, tachycardia, and hypertension are not uncommon after Mydricaine injection, and patients may need to keep lying down for a while post-injection. Rarely, cardiac arrhythmias occur (call on-call medical SpR in this event).

Following these acute treatments, mydriasis should be maintained with anti-cholinergics such as g.cyclopentolate 1% qds until the next review.

When to consider ward admission:

We probably do not admit severe cases early enough or often enough. Ward admission for nurse administered drops or overnight frequent drops can often rescue uncontrolled and blinding inflammation without the need for systemic steroids.
Consider if:

- Severe disease at onset with hypopyon and / or intense vitritis
- Escalating inflammation despite intensive therapy
- Uncontrolled pain
- Problems with compliance
- Lower threshold for admission in 1st attack patients

In some patients with exceptionally severe and blinding disease, systemic steroid will be necessary.

INTRA-OCULAR PRESSURE

Most patients with AAU will have a normal or low IOP at presentation (the IOP then rising as inflammation is controlled). A minority will have high IOP at presentation (causes include marked fibrin or “plastic” aqueous, accumulated PAS, or rarely pupil block). The principles of treatment of the AAU are the same in these patients and the IOP should be controlled medically as needed. In patients who are “steroid responders”, never undertreat with steroid for fear of an IOP rise – treat the inflammation as needed; review soon & treat IOP on its merits (consider loteprednol if inflammation not too severe and IOP proving difficult to control – but these instances are unusual in the acute setting).

FOLLOW-UP OF AAU

- **In most patients, management of the entire acute episode should occur within eye casualty.** All patients should be reviewed soon in eye casualty (time interval is a clinical judgement, but in general should not exceed one week). This is because of the inherent tendency of inflammation to escalate early during an attack.

- All 1st attack patients **after initial management in casualty (this may mean a few visits**) - refer to the uveitis service

- Patients already under **active** clinic follow-up – refer back to uveitis service after any urgent problems have been addressed.

- Patients who have had recurrent attacks, who know their disease well **and** whose AAU is settling well or has settled at casualty follow-up visits:

- Discharge with a decreasing scale of treatment over several weeks and advice to return if further problems

- If there is concern of an undiagnosed systemic association, refer to uveitis service

- Patients with typical recurrent unilateral non-granulomatous AAU who have had uncomplicated disease and who know their disease & symptoms well are advised to keep a bottle of topical steroid (e.g. g.PredForte 1%) and mydriatic (e.g. g.cyclopentolate 1%) unopened at home. They should dilate their pupil and use the steroid drop frequently in the event of a flare up when they are sure this has occurred. Such early treatment will enhance recovery**. However, this advice is not a substitute for ophthalmological diagnosis and supervision, and these patients are also advised to attend eye casualty within 24 hours. This advice should not be given to those who have not had recurrent attacks or those who are not very familiar with their disease & symptoms.**

SIMULTANEOUS BILATERAL ANTERIOR UVEITIS

- All patients should be seen by the SpR.
- Treat as necessary with topical steroid and mydriatics.
- Refer all patients to the uveitis service

CHRONIC ANTERIOR UVEITIS

- All patients should be seen by the SpR.
- Treat as necessary with topical steroid and mydriatics.
- Refer all patients to the uveitis service

INTERMEDIATE AND POSTERIOR UVEITIS

- All patients should be seen by the SpR.
- Refer all patients to the uveitis service

NOTE

- Certain forms of posterior segment intraocular inflammation are very urgent (eg macular threatening toxoplasma retinitis, CMV retinitis, acute retinal necrosis syndrome, infective endogenous endophthalmitis). These cases must discussed with Mr Pandit / Dr Innes or if not available or out of hours with the consultant on call.

SUMMARY OF CONDITIONS TO REFER TO THE UVEITIS SERVICE

- **AAU – 1st attack patients; severe or complicated disease; suspicion of underlying systemic association**
- **Any granulomatous form of uveitis**
- **All chronic forms of uveitis, regardless of anatomic location**
- **All intermediate uveitis**
- **All posterior uveitis**
- **All panuveitis**

These guidelines are not for childhood uveitis. Children presenting with uveitis should be discussed with consultant on-call or the paediatric ophthalmology service.

Uveitis in Adults, Eye Emergency Department Guidelines written by Mr R J Pandit, Consultant Medical Ophthalmologist, Revised March 2015.

Toxoplasma Retinochoroiditis (Adults)

This document should not be considered to contain all the necessary information for the safe treatment of toxoplasma retinochoroiditis. The responsibility for drug safety is that of the prescribing doctor.

Patients with seen in normal working hours should be discussed with the available Uveitis Service Consultant. This information provides guidance for EED staff out-of-hours.

BACKGROUND

Toxoplasma retinochoroiditis (TRC) is the commonest cause of posterior uveitis world-wide. It is the result of infection *Toxoplasma gondii*, a protozoan that is able to infect almost all mammals and birds. Cats are the definitive host in who the sexual cycle occurs, and excrete oocysts which are ingested by any intermediate host (including humans), so entering a wider food chain. Human infection is usually caught by (1) ingestion of tissue cysts in undercooked meat (especially pork) or unpasteurised cheese or milk; (2) ingestion of oocysts in contaminated water that is then used for drinking or cooking, or by inadvertent eating of soil or unwashed vegetable matter contaminated by cat litter; or (3) by transplacental transmission of trophozoites if the mother acquires primary infection during pregnancy.

Most people acquire toxoplasmosis by early adulthood. The initial infection is usually asymptomatic. In a minority, it produces a non-specific flu-like illness, a glandular fever syndrome with cervical lymphadenopathy, or very rarely meningo-encephalitis. After initial infection, the organism lies dormant in the form of tissue cysts containing bradyzoites in skeletal & cardiac muscle, the brain, and retina. After initial infection, a transient IgM positivity occurs followed by a lifelong IgG positivity.

About one-third of the world's population is infected, but there is great variation of prevalence between different countries rates depending on climate, dietary habits, hygiene standards, and presence cats in the community, especially around animal farms. Approximate seropositivity rates are:

West Africa / France / South America	80 - 90%
Sub-Saharan Africa	50%
USA	50%
Caribbean	35%
Indian sub-continent	10 – 20%
Caucasians and blacks born in UK	15%

CLINICAL FINDINGS

In the vast majority of patients TRC is a clinical diagnosis. It is usually a reactivation of dormant infection in an otherwise healthy adult, most commonly with 1st episode occurring aged 20 – 40 years. In immunocompetent patients, TRC is confined to the eye, has no systemic implications, and has no risk of spread to the fellow eye or other organs.

- Sudden onset unilateral blurred vision (clouding, floaters, scotoma).

- Pain and redness may occur with mild anterior uveitis, sometimes granulomatous and with elevated IOP. Elevation of IOP in TRC with anterior uveitis is common and can cause mild corneal decompensation even with only modest IOP elevation, contributing to reduced vision.

- Unilateral unifocal area of active retinitis adjacent to an old pigmented chorioretinal scar with overlying vitritis & vitreous haze (“headlight in fog”).

- Retinal periphlebitis, is common, and sometimes widespread

- Retinal arterial sheathing close to the active lesion may be seen.

Variations from this typical presentation are very wide and you should familiarise yourself with the spectrum of disease. Difficulties in diagnosis may occur when a scar is absent, or located far from the active lesion or in the fellow eye; in immunosuppressed patients when multiple, large, and / or bilateral lesions may occur; or in very elderly patients who may also have large fulminant lesions mimicking acute retinal necrosis syndrome.

The natural history of untreated TRC in an immunocompetent patient is resolution over 6 – 8 weeks, with the active focus of retinitis changing to an inactive chorioretinal scar with pigmented edge and leaving an associated absolute scotoma. The vitreous opacity may take longer to settle.

SEROLOGICAL TESTING

- Toxoplasma serology is not useful as a screening test for uveitis in general, because seropositivity is too common to be discriminatory.

- A positive IgG or IgM test confirms Toxoplasma infection has occurred at some time. It does not confirm the ocular diagnosis.

- However, serology should be performed, because negative serology rules out the diagnosis. Rare exceptions to this rule include primary infection with retinal involvement, before IgG or IgM positivity has occurred, or in exceptionally severely immunocompromised patients.

PCR TESTING ON AQEUOUS OR VITREOUS

This is only rarely required and generally reserved for atypical presentations where

the diagnosis is in doubt and the test result could substantially alter the initial management (eg atypical retinitis in immunosuppressed patient in whom CMV or other infections are in the differential diagnosis). Always discuss with consultant.

TOPICAL THERAPY

Anterior uveitis and raised IOP should be treated with topical steroid / mydriatic / pressure lowering drops as needed.

SYSTEMIC THERAPY

In immunocompetent patients:

- **An anti-microbial regimen PLUS**
- **Oral prednisolone**

In immunocompromised patients:

- **An anti-microbial regimen alone**

Additional treatments such as folinic acid (if pyrimethhamine chosen), gastro-protection & bone protection are given as needed

INDICATIONS FOR SYSTEMIC THERAPY

Not all *immunocompetent* patients with TRC require systemic treatment, which has significant side effects, bearing in mind that resolution is the norm in untreated disease in these patients. The purpose of treatment is to limit the extent of retinal damage and speed resolution of intraocular inflammation. The balance of risks versus benefits should be assessed for each individual patient.

Factors to consider include the location of the retinitis; the degree of vitreous haze; the stage of the inflammation (is the lesion already mostly healed?); the prospect of achieving better vision or stabilizing vision with treatment; pre-existing ocular morbidities / amblyopia / the state of the fellow eye; and the patient's wishes.

In general, treatment is given in the following situations:

- A focus inside or straddling the temporal vascular arcades.
- A juxtapapillary focus or a focus overlying the optic nerve head.
- Neuroretinitis
- A focus causing a retinal vascular occlusion.

- Substantial vitreous clouding that is itself producing severe visual loss or preventing fundus view to monitor disease

- Vitreous whiteout with a confirmed history of recurrent toxoplasmosis.

- Active lesion in any location in an immunocompromised patient.

INVESTIGATIONS FOR PATIENTS STARTING SYSTEMIC THERAPY

NB Treatment if indicated should not be delayed pending the results of these investigations. However, you must check the results as soon as possible and amend your treatment advice in their light if needed.

Blood pressure / Weight / BM

FBC
U&Es / LFTs
Glucose
HbA1c
Toxoplasma serology
Varicella zoster serology (due to oral steroid use)
Hepatitis B & C serology (due to oral steroid use)

CXR

ANTI-MICROBIAL REGIMENS (see Appendix 1 for doses & side effects)

- Anti-microbial treatment prevents protozoal proliferation and is given usually for 4 – 6 weeks and only discontinued when or after oral steroid treatment has been discontinued. It should not be discontinued before oral steroid therapy.

- Although the evidence base is weak, combination anti-microbial therapy remains preferred to monotherapy particularly for visually high risk lesions in otherwise healthy persons in who anticipated side effects are less than those with medical co-morbidities.

- The choice of regimen will depend on the visual risk of the individual case and the anticipated side effects, taking account the individual patient's medical history, allergy history, drug history, and medical co-morbidities.

- **It is the responsibility of the prescribing doctor to be familiar with drug side effects, drug interactions, and to have taken an appropriate medical history before prescribing a particular regimen.**

Standard Combination Regimens options:

- **Pyrimethamine / Sulphadiazine / Folinic acid (= classical triple therapy)**

- **Co-trimoxazole**

For visually very high risk cases, consider:

- **Pyrimethamine / Sulphadiazine / Clindamycin / Folinic acid (= quadruple therapy)**

- **Co-trimoxazole / Clindamycin**

Combination Regimens in Sulpha – allergic patients

- **Pyrimethamine / Clindamycin / Folinic acid**

- **Pyrimethamine / Azithromycin / Folinic acid**

Monotherapy Options

- **Clindamycin**

- **Azithromycin**

- **Atovaquone**

- **Spiramycin**

Oral Prednisolone 40 – 60mg OD (mane)

Rapidly effective and limits destructive retinitis. It is given at the above starting dose for 2 weeks if tolerated, then tapered over 6 - 8 weeks. With Ranitidine 150mg BD & Calcichew D3 forte 1 tab BD, and Alendronate 70mg once a week if no contra-indications.

INTRAVITREAL CLINDAMYCIN (1mg in 0.1ml)

The role of intravitreal clindamycin is unclear and is not considered as standard treatment. Reserve for cases in whom all systemic anti-microbials are either contra-indicated or carry serious risk, or in cases where there is progressive active retinal disease despite standard systemic therapies. Consider also in pregnancy if visually high risk lesion and patient does not wish systemic options (see appendix 1). May be combined with intravitreal dexamethasone 0.4mg / 0.1ml OR oral prednisolone as above. May need to repeat injection every 3 – 4 days until clear response (may need x3 – 4 injections in total often needed).

TREATMENT IN IMMUNOCOMPROMISED PATIENTS

Most patients in this group are either HIV/AIDS patients or transplant patients.

- These patients often have a number of medical co-morbidities and are on often on many medications. Their overall management is under infectious disease physicians or hematologists and treatment decisions should be made following

discussion with the respective medical teams.

- Diagnosis may be difficult due to atypical retinal presentations. Aqueous or vitreous sampling to rule out other infectious causes and to confirm toxoplasma infection by PCR may be needed, but should not delay starting empirical treatment before the results are available.

- The natural history of untreated disease is generally more aggressive and progressive, and any active focus will usually warrant treatment.

- Avoid systemic corticosteroid – treat with anti-microbials only (+ topical therapies as needed)

"NEVERS" IN TOXOPLASMA RETINOCHOROIDITIS

NEVER treat with systemic steroid alone (can lead to fulminant retinal necrosis)

NEVER give a peri-ocular injection of long-acting steroid such as triamcinolone or methylprednisolone (can led to fulminant retinal necrosis)

NEVER administer intra-vitreal steroid alone (can lead to fulminant retinal necrosis)

NEVER "taper" anti-microbial treatment – it should be discontinued abruptly when or after the oral steroid treatment is discontinued (not before).

- All patients should be referred to Uveitis Service – discuss with uveitis consultant on same day if in working hours; or on the next working day. Out of hours, discuss with consultant on call.

- Take OPTOMAP fundus photos and OCTs at earliest opportunity as a baseline;

- Investigate as outlined and start treatment if indicated WITHOUT DELAY in vision threatening cases;

- The choice of treatment choice is a clinical judgement based on visual risk and side effect profiling for the individual patient;

- Arrange for back up EED review WITHIN ONE WEEK to review clinical picture and monitoring bloods (FRB, U&Es, LFTs), BP & BM if systemic therapies and oral prednisolone have been started

Appendix 1: Anti-Toxoplasma Drugs

Drug	Adult dose	Adverse effects	Avoid if	Pregnancy	Monitoring
Pyrimethamine	50mg stat loading dose on day 1; then 25mg BD for 30 – 60 days **MUST BE USED WITH FOLINIC ACID 15mg three times a week (Mon / Wed / Thurs) to avoid bone marrow toxicity**	Bone marrow suppression (esp thrombocytopenia); nausea, vomiting, diarrhoea, glossitis, rash, fever	Known thrombocytopenia or leucopenia On lorazepam - hepatotoxic	Avoid if possible	Weekly FBC
Sulphadiazine	1g QDS for 30 – 60 days	Rash, Erythema multiforme, Stevens-Johnson's syndrome, nausea, vomiting, diarrhoea, fever, headache, tinnitus, bone marrow suppression, renal impairment; crystalluria	Known sulpha allergy Already on other sulpha drug (eg sulphasalazine or acetazolamide) Known renal failure	Avoid if possible	Weekly FBC, U&Es & LFTs
Co-trimoxazole (Trimethoprim TMP + Sulphamethoxazole SMX)	960mg BD for 30 – 60 days (= TMP 160mg + SMX 800mg)	Renal impairment (AKI with TMP) As for sulphadiazine	Known renal failure	Avoid if possible	FBC / U&Es / LFTs at 2 weeks
Clindamycin	300mg QDS for 30 – 40 days	Mild diarrhoea common (10%);	Elderly frail patients	OK	-

		pseudomembranous colitis rare (<1%) – but more common in elderly; if diarrhoea is severe, persistent or bloody, treatment should be stopped; arrange stool culture and commence Vancomycin 250-500mg QID and seek advice Rashes; nausea, vomiting			
Azithromycin	1g loading dose on day 1; then 500mg OD for 21 days	Well tolerated. Nausea, vomiting, diarrhoea, rash	On warfarin (↑↑INR) Known QT prolongation	OK	FBC / U&Es / LFTs at 2 weeks
Atovaquone	750mg TDS for 30 – 40 days	Rash, nausea, vomiting, diarrhoea, headache, insomnia, neutropenia, hepatotoxicity		OK	FBC / U&Es / LFTs at 2 weeks
Spiramycin (special order)	1g TDS for 21 days	As for azithromycin	As for azithromycin	OK (may reduce maternal-fetal transmission)	FBC / U&Es / LFTs at 2 weeks

Episcleritis and Scleritis

Episcleritis

Presentation
- Redness, usually in a wedge shape
- Discomfort/tenderness
- Rarely watering and photophobia
- Often recurrent
- Usually young – middle aged adults
- Self-limiting (up to a few weeks), this does not lead to scleritis

Examination
- Exclude uveitis
- Note extent, presence of nodules, limbal inflammation
- Dilated blood vessels are superficial & blanch significantly with topical phenylepharine 2.5%

Investigations
- Nil required

Management
- Topical lubricants, e.g. viscotears PRN

- Only if symptomatic:
- Oral NSAIDS, e.g. ibuprofen 400mg QDS
- If intolerant of oral treatment topical NSAIDS eg. ketorolac TDS
- Rarely topical steroids e.g. predsol 0.5% 4/3/2/1 per day each week

Scleritis

Presentation
- Deeper, more homogenous redness, with radially orientated vessels
- Very painful – deep/boring pain, can wake from sleep
- Pain on eye movements
- Can cause reduced VA
- Older patients (mean age 50's)
- Commonly associated with systemic inflammatory disorders
- **Caution** posterior scleritis can present with little or no redness; & scleromalacia can present with minimal symptoms

Classification
Posterior (10%) or anterior (90%)
Anterior subtypes

- *Diffuse anterior* - this is the most common (and benign) form, characterized by widespread inflammation of the anterior sclera. It accounts for about 50% of scleritis cases. It is not usually sight-threatening and tends to resolve

- *Nodular* - there are erythematous, tender, fixed nodules which, in 25% of cases, progress on to necrotizing scleritis. It commonly recurs.

- *Necrotizing* - this is less frequent (10% of cases) and is characterized by extreme pain and marked scleral damage; it is usually associated with underlying systemic disease. Where there is associated corneal inflammation, this is also known as sclerokeratitis.
- *Scleromalacia perforans* - (5% of cases) this is also known as necrotizing anterior scleritis without inflammation and is notable for its lack of symptoms. It is bilateral and is only seen with advanced rheumatoid arthritis (usually in women)

Examination

Confluent deeper liverish redness, does not blanch significantly with topical phenylepharine 2.5%
Exquisite tenderness over inflamed area
Note extent, presence of nodules, corneal involvement
Look for associated uveitis
Check IOP
Scleral necrosis (yellowish nodules or patches) & thinning (blue-ish tinged/darker areas)
Rarely choroidal folds, disc swelling, proptosis with posterior involvement – look for the “T” sign on B-ultrasound

At least 50% of these patients have an underlying systemic disease and, in 15% of cases, the scleritis is the first presentation of a collagen vascular disorder, preceding it by one to several months.

- Rheumatoid arthritis is by far the most common.
- Wegener's granulomatosis,
- Relapsing polychondritis
- Systemic lupus erythematosus
- Reiter's syndrome
- Polyarteritis nodosa
- Ankylosing spondylitis.
- Gout
- Churg-Strauss syndrome
- Syphilis.
- Following ocular surgery (intense inflammation adjacent to the surgical site, usually within six months of the procedure
- Local infection (pse*udomonas aeruginosa*, *Streptococcus pneumoniae*, *Staphylococcus aureus* and varicella-zoster virus)

Investigations

Initial treatment will usually involve systemic treatments and it is therefore important that investigations are performed BEFORE these are started, otherwise tests will be difficult / impossible to interpret.

BP
BM
Weight
Urine dipstick and analysis

FBC, ESR
U&Es, LFTs, Bone, ACE, CRP, HbA1C

Immunoglobulins, autoantibodies, Rheumatoid factor, ANCA
Treponemal serology VZV serology

CXR

Management

Often requires urgent multidisciplinary input and referral to uveitis service is generally advised.

Intense topical lubricants, consider punctual plugs for RA patients with bad hands

If mild symptoms and non-nectrotizing disease only, oral NSAIDS, e.g. ibuprofen 400mg QDS or diclofenac 50mg TDS (check no contra-indications, and check BP and no concern re renal impairment)

Patients with severe pain, nectrotizing scleritis or posterior scleritis invariably require systemic steroid, with appropriate bone protection and gastro-protection. Systemic immune suppression may be required at a later date. These patients should be managed by the EED reg with discussion regarding treatment, the need for admission and follow up arrangements with the consultant on call

RED FLAGS

Always discuss these following cases with senior clinician:

- Patients with known systemic connective tissue disease: discuss with rheumatologist involved if possible
- Necrotizing scleritis – ADMIT
- Posterior scleritis – will invariably need oral steroid
- Raised BP - ? underlying systemic vasculitis with renal involvement / caution with oral NSAID
- Proteinuria / Haematuria on urinalysis - ? underlying systemic vasculitis with renal involvement – AVOID oral NSAID.
- Scleritis occurring in the context of systemic / constitutional upset, uncontrolled BP, and haematuria / proteinuria is a MEDICAL EMERGENCY and should be discussed with the consultant on-call and the duty medical registrar
- Associated corneal melt – ADMIT & involve corneal team

Acute Retinal Necrosis Syndrome

- **This document provides guidance for the non-specialist for the initial out-of-hours management of acute retinal necrosis syndrome.**

- **This document should not be considered *per se* to contain all necessary information for the safe and effective treatment of acute retinal necrosis syndrome.**

- **The responsibility for correct diagnosis, management, and drug safety is that of the doctors in charge of the patient's management.**

- **All patients with a suspected diagnosis should be referred urgently to the uveitis service in normal working hours.**

Acute retinal necrosis syndrome (ARN) is a retinal infection caused by herpesviruses. It may occur at any age, but most commonly in adults 20 – 50 years of age. Most patients are immunocompetent and otherwise healthy, and 30% of patients have had herpes zoster involving any dermatome within the preceding 6 months.

ARN can be rapidly progressive, leading to irreversible blindness within days. It usually presents unilaterally, but often becomes bilateral if untreated (30 – 50% fellow eye involvement within 3 months in untreated disease). Complicated retinal detachment is common. Early diagnosis and treatment are crucial to limit retinal destruction, prevent blindness, and reduce the risk of fellow eye involvement.

Symptoms

Onset usually unilateral. May start with pain, redness, photophobia from anterior uveitis, followed within days by floaters, cloudy vision, dark or blank patches in the vision; or start with floaters or visual loss, and subsequently develop redness and pain after days.

Signs

- Sharply demarcated areas of peripheral retinal whitening, often with scalloped posterior borders. Initially be small and round, or geographic before enlarging and coalescing.
- Circumferential spread of retinal lesions.
- Rapid progression in the absence of anti-viral treatment.
- Occlusive retinal vasculitis involving both arterioles and venules.
- Prominent anterior uveitis (may be granulomatous) and vitritis.
- Optic neuropathy / retrobulbar neuritis with associated pain common

Initial Management (All patients)

Baseline investigations:

Photography (if possible): Arrange for wide field (Optomap) fundus photography of both eyes at the earliest opportunity. Even in instances when fundus view is poor, Optomap imaging can often capture important retinal pathology. Ask for peripheral views all quadrants.

Bloods:
FBC; ESR
U&Es / LFT / CRP
Glucose
HbA1c

Treponemal serology (can mimic)
Toxoplasma IgG (can mimic in immunodeficiency & elderly)
Varicella zoster IgG
HIV serology

CXR

Initial Treatment

(1) Admit the patient and commence intravenous Aciclovir 5 - 10mg/kg TDS once creatinine known (lower dose in renal or hepatic impairment; caution with higher doses in very elderly). Each dose infused over one hour.

(2) If admission is not possible, or in the cases of very small lesions / localised peripheral disease diagnosed early, oral valaciclovir 1g TDS may be used, but daily review will be required.

(3) Treat anterior uveitis aggressively if necessary with intensive topical steroid and mydriasis according to its severity. Use sub-conjunctival Mydricaine if needed to achieve mydriasis.

- **The above constitutes the minimum essential management for all patients suspected of having ARN out-of-hours.**

- **Refer on to uveitis service the following day or Monday if after a weekend.**

- **Do not delay treatment until after ocular fluids sampling, which are or may not be necessary (see below).**

- **A common problem is that the diagnosis itself is simply missed, uncertain, or part of a wider differential. In the latter two scenarios, the advice is to treat for ARN as above, but additional investigations & treatments may be necessary to cover other possible diagnoses (eg toxoplasma, endogenous endophthalmitis, syphilis).**

Further considerations in out-of-hours management

Aqueous and / or vitreous sampling for PCR studies

If the diagnosis is clear-cut, it is <u>not</u> essential to undertake aqueous or vitreous biopsy out-of-hours

- IOP may be very high (>30mmHg) or very low in ARN (< 8mmHg), and in either case aqueous sampling may be hazardous or difficult. Do not attempt aqueous sampling in hypotonous eyes, particularly if phakic.

- Vitreous biopsy should not be performed out-of-hours by inexperienced operators or by simple needle and aspiration. Many patients are young and may not have a PVD, and ARN itself carries a very high risk of retinal detachment. Vitreous biopsy should be performed using a vitreous cutter by an experienced operator.

If the diagnosis is not clear and other real possibilities exist that would alter the immediate management, consult with VR team on call for consideration of aqueous and / or vitreous biopsy.

Aqueous or vitreous specimens should be sent to microbiology for PCR for herpesviruses: HSV 1 & 2, VZV, and CMV. Inform the laboratory & ensure specimen is collected promptly. If there is sufficient specimen, also request toxoplasma PCR, and routine Microscopy, C&S

Intravitreal Foscarnet

If vitreous biopsy undertaken, then administer foscarnet 2.4mg on 0.1ml at the end of procedure.

Even if no vitreous biopsy is performed, it is preferable to administer intravitreal foscarnet early because there will be some patients who cannot tolerate high dose intravenous anti-viral treatment, and intravitreal therapy will provide a short term beneficial effect.

Oral Prednisolone

After 48 hours, if there is substantial vitritis, commence moderate-dose oral prednisolone (40 – 60mg OD). Cover with Lansoprazole 30mg OD or Ranitidine 300mg OD

Oral Aspirin

Start aspirin 150mg OD if no contra-indications in patients who are not already on anti-platelet or anti-coagulation medicines.

Barrier Laser

If the fundus view permits and if the topography of the retinitis makes it practicable,

laser barrage should be performed posterior to all posterior edges of retinitis.

Vitreo-Retinal Referral

- if vitreous biopsy being considered (see above)

- if retinal detachment or retinal breaks present

Subsequent Management

- In typical fulminant disease, intravenous aciclovir is usually continued for 10 – 14 days, followed by oral valaciclovir 1g tds for at least a further 2 weeks before taper (total duration of anti-viral treatment is controversial).

- Check U&Es and LFTs daily; check results and reduce dose acyclovir if renal or hepatic impairment occurs.

- Prednisolone treatment is gradually reduced and discontinued after several weeks, depending upon response.

- Aspirin is discontinued as soon as retinitis stops extending.

- Topical steroid & mydriatic adjusted according to anterior uveitis activity.

- Barrier laser (as above) when fundus view permits.

- Later therapeutic vitrectomy if chronic vitreous opacification develops and contributory to visual loss.

FLASHES AND FLOATERS

History: Duration, nature of onset, description of symptoms (what kind of “flashes”, floaters, field defect), POH, PMH, FH

Features suggestive of increased risk of retinal tear and/or detachment:

- Flashes ***and*** floaters rather than just one symptom alone
- Multiple floaters
- Complaint of reduced quality of vision
- High myopia
- Previous retinal tear/detachment
- Predisposing syndrome (Sticklers, Marfans)
- Recent onset of symptoms (<2 weeks)
- Recent significant ocular injury (blunt, sharp iatrogenic or traumatic)
- Family history of retinal tear/detachment

Examination:

Acuity, RAPD, ***pigment cells in vitreous***, preretinal/vitreous haemorrhage, vitreous syneresis, Weiss ring, lattice degeneration, retinal tears, retinal detachment

Features suggestive of increased risk of tear and/or detachment

- Reduced corrected acuity
- RAPD
- ***Pigment cells in vitreous***
- Signs of previous vitreoretinal disease

Management:

The reasons for seeing patients with acute-onset flashes and/or floaters are:

- To identify retinal breaks before a retinal detachment occurs, and treat prophylactically
- To exclude a retinal detachment already present

It should be possible for the vast majority of these cases to be examined carefully in EED and discharged; if the EED practitioner does not feel confident to exclude a retinal break then the opinion of a more senior doctor (usually the EED registrar) should be sought at that visit, especially if pigment cells are seen in the vitreous.

Haemorrhagic PVDs where there is sufficient view of the fundus to exclude tears can be discharged with a PVD leaflet which has SOS symptoms for the patient to return if they deteriorate. If the vitreous haemorrhage is significant (there is some view of the retina and retinal tears cannot be ruled out with certainty due to poor view) the notes should be passed to the secretary of the VR consultant "on" for the week, who will arrange for appropriate clinic review. ***Do* not *give appointments at the time.***

If there is no fundal view due to vitreous haemorrhage then B scan should be performed to rule out RD, and the VR fellow or consultant should be informed.

NB There is no "PVD clinic" – a VR clinic slot may rarely be requested for further assessment of HIGH RISK PVD cases after every effort has been made in EED to exclude a tear/detachment. It is not a dumping ground for cases when the EED/on-call team does not feel inclined to perform an adequate examination.

Cases of blunt trauma should be handled by the consultant team on-call unless:

- ***A definite retinal break/detachment has been found***
- ***The senior member of that team does not feel able to exclude the above.***

Vitreo-retinal cases

On –call arrangements

RJH/KPS are on alternate weeks, subject to leave, rota in EED and on ward. VR fellow may be available. VR consultant available 09.00 Monday – 12.30 Friday.
There is a VR on call rota with Sunderland that covers weekends.
Bank holidays are covered on a regional basis.

Retinal detachments

Inform VR team during working hours. After hours the patient can be admitted or sent home at the discretion of the on-call team and the VR team notified that day if possible, failing that early the next day.
If in doubt phone the on-call VR Consultant for advice.

Retinal tears

Initial treatment is the responsibility of the on call registrar. If the on call registrar is unable to perform adequate laser he/she should notify the VR fellow or on call VR consultant.

Acute spontaneous vitreous haemorrhages (with no or inadequate retinal view)

***Recurrent in diabetics*:** Early clinic appointment with their normal ophthalmologist if registered; or if not, the on-call ophthalmologist.

***First haemorrhage in diabetic*:** Perform US scan same day, to ensure retina flat then clinic as above.

In non-diabetic: Perform US scan same day, to ensure retina flat. US/VR clinic the following week.

Post-op endophthalmitis

Non-VR teams are encouraged to perform an urgent vitreous tap.

- Make up the antibiotics as per the instruction sheets in the dedicated boxes (kept in theatre)
- Make a scleral stab at the pars plana with a 23-gauge MVR blade (a small conj flap often makes this easier especially if the conj is oedematous)
- Insert a 23-gauge vitrector 1cm into the vitreous cavity, aiming at the centre of the eye
- Use the “VITBIO” setting on the Oertli machine.
- Use a 2ml syringe connected directly to the aspiration tubing on the vitrector handpiece, using a female-female luer adaptor if necessary, to gently aspirate vitreous whilst cutting.
- Take enough vitreous to fill the aspiration tubing
- Withdraw the vitrector from the eye and hold it with the port end up whilst aspirating the vitreous from the tubing into the syringe
- Cap the syringe and send it urgently to microbiology as the sample
- Instil antibiotics according to the current protocol

The VR team will happily give advice but this should not be allowed to delay prompt action.

Patients presenting with a vision of POL should be referred to the VR team ASAP. If unavailable, the above protocol should be followed and a VR consultant notified as soon as available.

IOFBs

The VR consultant on call should be notified in the same way as for detachments. In most cases CT scan should be performed. Any leaking wounds should receive primary repair. Intravitreal antibiotics should be considered.

MEDICAL RETINA

This includes common acute presentations:

- Age-related Macular Degeneration—Dry or neovascular; new or existing treated neovascular AMD
- Myopic Macular degeneration
- Retinal Vein Occlusion
- Intravitreal injection complications
- Central Serous Retinopathy
- Some retinal conditions that are non-MedRet

In general, diagnosis of medical retina conditions requires knowledge of likely symptoms, a fair amount of proficiency in examining the fundus by slitlamp biomicroscopy with indirect lenses (90, 78 or 60 dioptre) and some ability to interpret OCT scans.

DRY AGE-RELATED MACULAR DEGENERATION

Symptoms—reduced central visual acuity, difficulty with close work, inability to read even with reading glasses

Signs—can be few drusen or focal retinal pigment changes i.e. hyper or hypopigmentation or areas of geographic atrophy

Investigations—OCT to rule out exudative AMD if there is clinical suspicion

Management—explain condition to patient. Currently no treatment is available for dry AMD.

Explain regarding monitoring central visual acuity in **each individual eye**, particularly looking for central scotoma, distortion where straight lines appear wavy, difficulty seeing faces or reading text on TV etc. An Amsler grid is **not** necessary and everyday objects can be used to monitor changes in vision. Explain importance of reporting quickly to their optician or to EED in hours (*out of hours attendance not required*).

Information leaflet is available in the department; this includes advice regarding healthy diet with plenty of fruits and vegetables and smoking cessation advice.

Follow-up —If vision severely affected, may be eligible for registering as sight impaired, or may benefit from low vision aids, this can be achieved with an optician/LVA appointment without need for a dr clinic visit.

ECLO officers are often available on the day to help with support and practical advice if required, or twinned with an LVA appointment

NEOVASCULAR AGE-RELATED MACULAR DEGENERATION (EXUDATIVE OR WET AMD)

New presentation:

Symptoms— affects patients over 50 years age. Recent onset central distortion, duration of symptoms (this is usually in weeks or days), reduction in near vision, recent difficulty with close work, central scotoma or referred with changes seen by optician at regular sight test.

Previous ophthalmic history especially glaucoma or cataract surgery
Enquire about smoking (current or previous) and driving status

Signs—central macular haemorrhage which can be red intraretinal or dark subretinal, pigment epithelial detachment seen as a central raised area on slitlamp indirect ophthalmoscopy, cystoid macular oedema, exudates clustering around fovea with a suggestive raised lesion.
Similar signs may be seen in a peripapillary distribution with central intraretinal fluid
Orange-brown nodules of polypoidal choroidal vasculopathy may also be noticed
Signs of dry AMD as detailed above may also coexist in the same eye or the fellow eye.

Investigations—OCT to detect intra or subretinal exudation or PED. Please look at OCT and write down your impression. Ask a senior if unsure.

If vision worse than 6/60, a refracted logMAR is advisable to check whether eligible for treatment. NICE guidance is that vision should be 6/96 or better (i.e.25 letters on ETDRS chart or better) for treatment with intravitreal injections of antiVEGF.

Treatment - Seek advice from Medical Retina team which is doing a Macula clinic that session. (Ring MedRet Fellow DECT phone) They would advise about need for Fluorescein angiogram or possibility of commencing treatment right away. Give patients information leaflet regarding Macula treatment clinics if you are reasonably sure of diagnosis. Do not routinely book FFA from Eye casualty without consulting MedRet Fellow or other macula team member. Intravitreal injections should not be performed directly from EED, but should go via the Macula clinic (there is one every session of the week)

Give dietary and smoking cessation advice as above.

Advise regarding monitoring central vision in other eye as above.

Nutritional supplements may be used to reduce risk of other eye progressing to neovascular AMD—these are not prescription medicines but are available to buy at most chemists/ pharmacies.

If they do not meet treatment criteria, consider eligibility for certificate of visual impairment or requirement for low vision aids and refer accordingly to clinic (see follow up advice for dry AMD as above).

Patients known to have exudative AMD
Patients already diagnosed and receiving antiVEGF treatment may present with new symptoms due to worsening of their condition.

Symptoms and signs are the same as for new wet AMD as above.

History—ask regarding duration of symptoms, interval since their last injection

Look at Medisoft to get further details about their condition/treatment given (Lucentis or Eyelea).

Investigation—OCT to document any changes.

Use Medisoft and OIS to compare change in vision/ fundus picture/ macular thickness from previous.

Management—If the interval since their last antiVEGF injection is >4 weeks, they may warrant another injection. Seek advice from MedRet Fellow (ring DECT) regarding arranging further treatment.

If interval since injection is less than 4 weeks, check that they have their next macula clinic appointment and advise them to keep it. Pass notes and EED card to their consultant to consider sooner review. Seek advice from MedRet Fellow if unsure. Please record findings on Medisoft as a new attendance so this is visible to the Medical Retina team when they next see the patient.

For any other complications of injections see below in later section

MYOPIC MACULAR DEGENERATION
Signs and symptoms similar to exudative AMD, however the patients may be much younger and are usually highly myopic.

Investigation and management should be as for new exudative AMD (see above). **These OCT can be difficult to interpret** and so there should be a high index of suspicion when symptoms are significant in somebody with axial myopia even if there isn't fluid apparent on OCT. Looking at the entire OCT on the camera can give more information than OIS. Do consider referring to Macula clinic for treatment on the same day.

RETINAL VEIN OCCLUSION (Central, branch or hemiretinal)

Symptoms range from mild blurring to field defects to total loss of vision in one eye. This may also be noticed by their optician and referred to EED.

History—ask about duration of symptoms, past ophthalmic history especially glaucoma, medical history specifically regarding hypertension, diabetes and raised cholesterol. In younger patients(<45), ask regarding haematological problems or history of vasculitis

Signs—document best corrected visual acuity, intraocular pressure and look for afferent pupil defect. Look for rubeosis iridis preferably before dilating. Fundus examination will reveal superficial and deep retinal haemorrhages affecting the entire retina in CRVO or one sector in BRVO or one half (superior or inferior) in HRVO. Macular oedema, tortuous engorged retinal veins, cotton wool spots and swollen optic disc may be present.
Features suggesting ischaemic CRVO are vision <6/60, relative afferent pupil defect, numerous cottonwool spots and should trigger searching for new vessels on the iris, disc or retina.

Investigation—Blood pressure and blood glucose help to look for systemic associations which can be flagged up to their GP. A full blood count and ESR would also help. OCT should be done if macular oedema is suspected.

Management

Explain the condition and need for repeated review to the patient. Information leaflets are available

Of systemic associations--Any abnormal blood results should be communicated to the patient's GP. *Please do not advise aspirin in EED*. This is the responsibility of their physician as it requires various other bits of clinical information which would not be available in the eye department.

Of the eye—If there is evidence of glaucoma or ocular hypertension, pressure-lowering medication should be commenced right away. This is especially important for the fellow eye.

Macular oedema may need treatment with intravitreal steroid or antiVEGF—make a 'soon' referral to Medical retina clinic. If vision reduced due to macular oedema, seek advice from seniors in Macula clinic on the day about commencing urgent treatment. Mild BRVO may resolve spontaneously and may be observed depending on the overall clinical picture.

Rubeosis or new vessels on the disc or retina needs urgent panretinal photocoagulation—preferably by the oncall registrar that day if there is reasonable view of the fundus.

Follow up - All cases of retinal vein occlusion need to be reviewed at the Medical retina clinic for possible sequelae.

INTRAVITREAL INJECTION-RELATED COMPLICATIONS

Innocuous—the following need only reassurance. Injection information leaflets are available to explain.

- Conjunctival hyperaemia
- Sub-conjunctival haemorrhage
- Few floaters or visible air bubble/ steroid pellet
- Ocular surface features of dry eye—lubricants may be given.
- Post-injection corneal abrasion--Treat as any other abrasion.

Follow up for all above — keep their scheduled review appointments. Care should be taken not to give the patient the impression that they have developed an 'allergic reaction' to any of the drops or drugs unless this is truly the case.

Major—sight-threatening

Post-injection endophthalmitis

Symptoms—Rapid blurring or reduction of vision within 48-72 hrs of injection, numerous floaters, severe pain persisting to next day, sticky discharge.

Signs—Reduced VA from previous visit, cloudy cornea, flare and cells in anterior chamber, vitreous haze or cells or poor view of fundus.

Management - These need to be treated as infective and need **urgent** vitreous biopsy and intravitreal antibiotics. The oncall registrar and consultant have to be informed, as also theatre staff and microbiology (see endophthlamitis guidelines).

Post-injection raised pressure

Measures to lower IOP (see acute glaucoma section), ensure no features of endophthalmitis

CENTRAL SEROUS RETINOPATHY

Symptom--disturbance in the central vision, which is variously described as a central cloudy/ grey/ brown/ blurred patch, remains in the same location. Ask regarding steroid treatment—any current or previous use in any form including non-prescription for body building, inhaled for asthma, nasal sprays for allergies.

Signs—Reduced visual acuity, may improve with +1D lens, Ring reflex around fovea, Raised area in macula with loss of foveal light reflex, RPE changes in same or fellow eye indicating previous episodes

Investigations - OCT helps to document subretinal fluid.

Management—no treatment required in the majority of patients. Explain self-limiting nature of this condition (4—12 weeks).

Information leaflets are available.

Follow up—routine referral to Medical retina clinic.
There are some conditions affecting the macula which do not need attention in the medical retina clinic but have been added here due to popular misconceptions

- Macular hole—either full-thickness or lamellar
- Epiretinal membrane
- Vitreomacular traction

These are all *not medical retina* and these are all **not urgent**. If further management warranted, routine referral should be made to the vitreoretinal team.

RETINAL ARTERY OCCLUSION

This can be either central or branch retinal artery occlusion.

Symptoms range from a small field defect in BRAO to sudden painless total loss of vision in CRAO.

Asymptomatic incidental arteriolar emboli do not require any investigation or management from EED. If incorrectly referred by opticians, they are be referred back to the GP for assessment of vascular risk factors.

History—ask about GCA symptoms if of appropriate age, hypertension, diabetes, cholesterol and smoking.

Signs
Signs of CRAO–afferent pupil defect, no perception of light or just PL/ HM vision, cherry red spot on fundus examination, discontinuous segments of the blood column in retinal arteries (cattle trucking).

Signs of BRAO—Retinal ischaemia in one quadrant or smaller area

Management—this should be treated as a TIA and the stroke risk needs to be evaluated. A local TIA clinic referral should be made, there are proforma sheets for North of Tyne region patients. Patients should be advised not to drive for a month or at least until they are seen there.

In acute RAO (<8 hours), measures to re-establish circulation can be tried, such as IV acetazolamide 500mg, rebreathing into a paper bag, globe massage or paracentesis.

If risk of GCA check inflammatory markers and treat accordingly (see neuro-ophthalmology section).

Follow up—this is not a medical retina problem. This needs a ***routine general clinic*** *review* to ensure the patient has had review at the neurovascular clinic and that their stroke risk has been assessed. This also helps to check that the patient has access to low vision services or ECLO if required.

Acute management of CRAO following facial filler injection

Because of the extensive anastomoses of facial vessels it is possible to inject intra-arterial embolus of filler inadvertently in almost any region of the face. Highest risk areas are injections around the glabellar, nasal dorsum, nasojugal & nasolabial folds.

Cosmetic fillers are most commonly hyaluronic acid (HA) based but can also be autologous fat. The patient may not know exactly what type of filler has been used but will know if they have had liposuction to harvest autologous fat.

The referring practitioner should be able to give accurate information about the type, quantity and location of filler.

Patients complain of sudden unilateral visual loss at the time of injection or immediately afterwards. This is almost always total loss of vision down to hand movements or worse. There may be accompanying headache or periocular pain.

In all cases where suspected CRAO has occurred acutely following injection of facial filler that is NOT autologous fat, it should be assumed to be hyaluronic acid based; and the following emergency measures[1] instituted **within 4hrs[2]** of the acute visual loss:

- Ocular massage
- Rebreathing into a paper bag, limited by symptoms of dizziness
    - These can be done after diagnosis whilst other measures are being prepared
- **Inferotemporal quadrant retrobulbar hyaluronidase injection 1500 IU in 10 ml saline over 2 minutes[3]**
    - Hyalase is now kept in the drugs cupboards in EED, ward 20 and the “antidote cupboard” in main ED
- Limbal paracentesis of 0.1-0.2ml of aqueous (remove the plunger on an insulin syringe to allow passive filling)
- IV Diamox 500mg

If autologous fat has been used all above measures should be attempted except for hyaluronidase

Ref 1: **Treatment Options for Central Retinal Artery Occlusion** *Curr Treat Options Neurol*. 2013 Feb; 15(1): 63–77. Published online 2012 Oct 16. https://doi.org/10.1007/s11940-012-0202-9 PMCID: PMC3553407

Ref 2**: Central retinal artery occlusion – rethinking retinal survival time** *BMC Ophthalmology* 2018 **18**:101 https://doi.org/10.1186/s12886-018-0768-4

Ref 3: **The treatment of hyaluronic acid aesthetic interventional induced visual loss (AIIVL): A consensus on practical guidance.** *Journal of cosmetic dermatology.* 8 June 2018 https://doi.org/1111/jocd.12672

Guidelines for the Management of Post-Operative Endophthalmitis (POE)

1. Introduction

Endophthalmitis is an extremely serious and rapidly destructive intra-ocular infection, which may originate from an exogenous source (e.g. recent intra-vitreal injection or intra-ocular surgery, suture abscess, blebitis or penetrating eye injury) or an endogenous source (e.g. via the bloodstream). The focus of this guidance is to aid in the management of acute post-injection or post-operative endophthalmitis (POE), although reference to other types of intra-ocular infection will be made.

No matter what the source, the primary aim of treatment is to **deliver a therapeutic dose of suitable antimicrobial agents into the vitreous cavity WITHOUT DELAY (<2 hours).** The secondary aims are **intra-ocular fluid sampling** for microbiological analysis, and **clearance of intra-vitreal toxins/organisms** by pars plana vitrectomy in selected cases – see below.

2. Common Microbes Implicated in POE

Gram +ve bacteria (most common)

- Coagulase -ve staphylococcus spp (e.g. S. epidermidis) (skin flora, less virulent)
- Staphylococcus aureus (skin flora, highly virulent, presents rapidly)
- Streptococcus spp (oral flora, highly virulent, presents rapidly)
- Bacillus spp (environmental flora, highly virulent, presently rapidly)
- Propionibacterium acnes (skin flora, low virulence, chronic presentation)

Gram –ve bacteria (less common)

Fungi (rare)

3. Selection of Antimicrobial Agents

Antibiotic selection is made before microbiological culture/sensitivities are available, and therefore a **combination of TWO broad-spectrum antibiotics** is used. To achieve the concentration of antibiotics required, **intra-vitreal injection** is administered.

FIRST LINE

Drug	Dose	Volume
VANCOMYCIN (gram +ve coverage)	1 mg	0.1ml
CEFTAZIDIME (gram –ve coverage)	2 mg	0.1ml

SECOND LINE (may substitute for ceftazidime in case of serious penicillin allergy)

AMIKACIN (gram –ve coverage)	400 mcg	0.1ml

IF FUNGAL AGENT SUSPECTED

Rare cause of POE, but more common in poorly controlled diabetic/ immunocompromised patients. Where strongly suspected or culture-proven, **one of the following** intra-vitreal anti-fungal agents may be administered:

AMPHOTERACIN B	5 mcg	0.1ml
VORICONAZOLE	50-100 mcg	0.1ml

NOTE: Cases of suspected fungal endophthalmitis should always be discussed with Microbiology upon admission, who will advise regarding systemic anti-fungal treatment.

4. Preparation Of Antimicrobial Agents

All necessary equipment for preparation of intra-vitreal antibiotic injections is provided in kit form, and stored in Eye Theatres. It is the responsibility of theatre staff to ensure check adequacy and expiry dates of stock at all times.

The dose and volumes of antibiotics required are critical and are outlined above. **Mistakes may result in irreversible retinal damage.** It is the responsibility of at least TWO members of staff to assist and supervise in the preparation of each intra-vitreal injection.

5. Sampling and Administration

Sampling of intraocular fluids and intra-vitreal antibiotic injection are performed together, usually under sub-tenons anaesthetic. It is advisable to complete the steps in the following order:

AQUEOUS TAP

- Use 30G needle on a 1ml syringe to gently aspirate 0.1ml of aqueous by withdrawing the plunger (or assistant can do this).

- Go through clear cornea, directing the needle on the horizontal plane and anterior to the iris at all times, to avoid contact with the lens.

VITREOUS TAP

- Make a limited peritomy using Wescott scissors at the most convenient quadrant (usually superotemporal)
- Using a 23G MVR blade (or a 23G trocar), make a sclerotomy (4mm/3.5mm from limbus in phakic/pseudophakic eye respectively)
- Connect the vitrector hand-piece to a 2ml syringe via the cutter aspiration line, and ask assistant/scrub nurse to hold syringe
- This is a 'undiluted' tap, so no infusion is required
- Using the 23G cutter, go through the sclerotomy (or trocar) with the cutter directed towards the center of the globe
- Use 'Hill 23G VIT PN' Oertli OS3 settings at 3000 cut rate - move the pedal all the way to the right, without depressing it, to cut. No cassette is required, nor will the machine aspirate. While cutting, ask assistant/scrub nurse to gently aspirate liquid vitreous through the syringe to fully fill the tubing *without* filling the syringe. The complete tubing fill is all that is required - further aspiration will soften the eye excessively

ANTI-MICROBIAL INJECTION

- Through the sclerotomy (or trocar), inject the prepared anti-microbial agents +/- 0.1ml of dexamethasone.
- Digitally check IOP and reform with BSS if globe grossly soft
- Remove trocar if used, and suture with 8.0 vicryl to sclera and conjunctiva
- Site pad/Cartella

NOTE: An alternative vitreous tap technique would be to insert a 23-25G needle on a 2ml syringe trans-conjunctivally, using the landmarks above, to withdraw ~0.2ml of liquid vitreous. Although acceptable, a higher rate of 'dry taps' (no sample obtained, due to formed vitreous) is associated with this simpler technique.

The samples should be sent **immediately** to microbiology. Gram film and culture/sensitivity are mandatory; call lab **in advance** so that they are expecting and will process them straight away. If you suspect fungal endophthalmitis, inform the lab to enable the necessary stains/cultures to be prepared.

6. Role of Intra-Vitreal Steroids

- Dexamethasone 400mcg/0.1ml

The role of intra-vitreal steroids given at the time of initial intra-vitreal antibiotic administration is *controversial.* These are contra-indicated in suspected fungal/viral endophthalmitis, but may be beneficial in dampening an early and aggressive inflammatory reaction, often seen in bacterial endophthalmitis. Consider administering intra-vitreal steroids in the following situations:

- Where a highly virulent organism is suspected (rapid onset of symptoms and worsening of clinical signs – e.g. streptococcus spp. following intra-vitreal injection)

- Where use of supplementary systemic steroids is undesirable (e.g. diabetics)

7. Role of Pars Plana Vitrectomy (i.e. when to speak to VR!)

The pivotal Endophthalmitis Vitrectomy Study (EVS) concluded that patients with presenting visual acuity (VA) worse than 'hand movements' benefitted from immediate pars plana vitrectomy (PPV). However, these findings are somewhat out-dated, and *there is currently a trend towards early PPV for numerous additional non-EVS indications.*

Consider early involvement with the VR team in the following situations:

- VA <HM at presentation
- Suspicion of a highly virulent organism, regardless of VA (rapid onset of symptoms and worsening of clinical signs – may occur over hours c.f. days)
- Exogenous endophthalmitis occurring post intra-vitreal injection, related to suture abscess/blebitis/trauma OR endogenous endophthalmitis (higher likelihood of virulent organism)
- No clinical improvement at 24-48 hours post administration of intra-vitreal antibiotics
- Onset/persistence of post-endophthalmitis complications (e.g. retinal detachment, dense vitreous opacities)

NOTE: Do **NOT** delay administration of intra-vitreal antibiotics whilst awaiting a VR opinion

8. Further Management Of Bacterial POE

The following should be prescribed **upon admission**:

SYSTEMIC ANTIBIOTICS

- Ciprofloxacin 750mg PO BD

Note: Consider involvement with Microbiology early on, especially in complex cases, so that they may advise regarding optimal systemic anti-microbial treatment

TOPICAL ANTIBIOTICS

These play a *secondary* role in the management of POE

- POE: g. chloramphenicol QDS

The exception is in the case of a corneal suture or bleb related endophthalmitis, where high frequency topical antibiotic may play an important role:

- Suture abscess/blebitis related endophthalmitis: g. ofloxacin every 1-2 hours (06.00-24.00 hours) (suggest liaise with corneal/glaucoma team re topical antibiotic selection)

TOPICAL STEROIDS

- g. Prednisolone 1% every 1-2 hours (06.00-24.00 hours)
  (suggest liaise with corneal/glaucoma team if suture abscess/bleb related)

MYDRIATICS

- g. Atropine 1% BD

At 24-48 hours: If clinical course remains stable or improving, and consistent with bacterial infection, consider adding supplementary systemic, steroids. Exert caution in diabetic/frail patients, who will require careful systemic monitoring whilst on oral steroids. In such patients, peri-ocular/intra-ocular steroid administration may be the safest option. If fungal or viral endophthalmitis is suspected, *do not* routinely administer supplementary steroids at this stage (may potentiate infection). In immunocompromised patients, supplementary steroids may not be safe or necessary (tendency to mount a poor inflammatory response).

SYSTEMIC STEROIDS (plus gastro-protection)

- Prednisolone 40-60mg PO OD, up to 1mg/Kg, tapering swiftly according to clinical response