Age-Related Macular Degeneration *(AMD)* and Polypoidal Choroidal Vasculopathy *(PCV)*

Guidelines

Developed by the AMD & PCV
Steering Committee from:

MINISTRY OF HEALTH
MALAYSIA

Rationale for the **guidance**

Age-related macular degeneration (AMD) is the commonest cause of severe visual impairment in older adults in the developed world. The two main late AMD phenotypes geographic atrophy and exudative AMD are responsible for visual impairment or blindness. AMD in Asian populations reveals many differences from the western populations, especially in phenotypic manifestations and prevalence of clinical subtypes. In the past, AMD was perceived to be more prevalent in Caucasians than Asians. However, recent studies have shown that although that is true of late AMD, the prevalence of early AMD among both Caucasians and Asians are comparable. The pooled prevalence estimates of early and late AMD in Asian populations aged 40 to 79 years were 6.8% and 0.56%, respectively.[1]

In Asian populations, polypoidal choroidal vasculopathy (PCV) constitutes a high percentage of patients presenting with exudative AMD. Prevalence rates of 4%,[2] 7.8%,[3] 8.2%,[4] and 9.8%[5] have been reported in Caucasian patients with presumed AMD, compared with substantially higher prevalence rates of between 23.9% and 54.7% in Asian patients with presumed AMD.[6-10]

The past decade has witnessed an increase in therapeutic options with novel strategies to target neovascularisation without damaging the neural retina or other equally important tissues. The management of AMD is a fast changing field and there are strong epidemiological and clinical reasons to issue new guidelines to keep pace with these developments. The guidelines are intended to set the standards for best practice in Kementerian Kesihatan Malaysia Hospitals with ophthalmic service. The guidelines will act as a benchmark for service planning by providers and set national standards for audit.

Diagnosis: **Clinical**

Geographic atrophy (GA)
The presentation of GA is usually insidious and often detected during routine fundus examination. When GA is bilateral and involves the fovea of both eyes, patients may complain of deterioration of central vision. A common mode of presentation is difficulty with reading initially with the smallest sizes of print and then later with larger print and or words. The confirmation of the diagnosis of GA is by clinical examination using a high definition fundus lens for stereo biomicroscopy. This will reveal the characteristic area or areas of pallor with sharply defined and scalloped edges. When the area of GA is larger than 500 microns, large choroidal vessels are clearly visible within the area of pallor. Usually areas of drusen and focal hyperpigmentation are visible in the retina adjacent to the patch of GA.

AMD-CNV
The onset of AMD-CNV is heralded by the appearance of central visual blurring and distortion.

Examination of the macula usually reveals an exudative macular lesion with or without hemorrhage along with other features of early AMD such as drusen and pigmentary irregularities. Sometimes these latter features are not observed once AMD-CNV has supervened. However the fellow eye if free of advanced disease will usually exhibit some or all of these early clinical signs and their presence is helpful in confirming that the neovascular lesion is due to AMD.

Polypoidal Choroidal Vasculopathy (PCV)
This is a variant of neovascular AMD in which highly exudative lesions with haemorrhagic pigment epithelial detachments can occur anywhere within the macula and even outside the macula. High speed fluorescein or indocyanine green angiography typically reveals hyperfluorescent dilated complexes of choroidal vessels that leak in the later phases of the angiograms. These dilated complexes look like polyps or grapes and hence the name of PCV. PCV is considered part of the spectrum of AMD and a strong association with hypertension, ischaemic heart disease and smoking has been noted. The use of confocal high speed imaging devices allows PCV to be diagnosed more frequently and it accounts for more than a third of serosanguinous maculopathy in older adults in Asian populations and for some 8-13% of that seen in Caucasians.[13]

Conditions mimicking **AMD**

A number of disorders can result in macular lesions which have to be distinguished from AMD.

Exudative Macular lesions mimicking AMD
Diabetic maculopathy: This is the most common exudative central macular disorder in older adults. Patients with diabetes frequently exhibit retinal microaneurysms, haemorrhages and exudates often set in a background of macular oedema. The presence of more extensive vascular signs outside the macular arcade along with venous engorgement or beading should alert the clinician to a diagnosis of diabetic maculopathy. Fluorescein angiography is needed to confirm the absence of choroidal neovascularisation and sub RPE pathology. Sometimes exudative AMD and diabetic maculopathy can coexist as both are common conditions.

High myopia can be associated with choroidal neovascularisation. These neovascular complexes are believed to occur as a consequence of the development of minute cracks in thinned Bruchs membrane allowing choroidal vessels to access the subretinal space.

Inflammatory CNV: A number of the choroidal inflammatory diseases such as Vogt Koyanagi Harada disease, toxoplasmosis and white dot syndromes (eg. presumed ocular histoplasmosis, punctate inner choroidopathy, multifocal choroiditis) can be associated with inflammatory neovascular membranes.

Central Serous Chorioretinopathy (CSCR): A collection of serous fluid in the sub-neurosensory retina without any evidence of neovascularisation. Chronic CSCR can sometimes be confused with AMD, again the history, symptoms and a combination of retinal imaging usually helps distinguish between the two.

Macular telangiectasia: Idiopathic macular telangiectasia (MACTEL) also sometimes termed perifoveal or juxtafoveal telangiectasia can be easily confused particularly with the RAP form of neovascular AMD. In MACTEL abnormal retinal vessels showing telangiectactic changes are detectable in the macular region. Two types are recognised. Type 1 MACTEL occurs in middle age persons and the condition is usually unilateral and exhibits exudative features as the vessels are leaky and intraretinal fluid accumulation occurs with a cystic maculopathy evident using OCT imaging. Type 2 MACTEL occurs in older people and is usually bilateral with evidence of crystalline deposits, pigmentary charges, and right angled venules evident temporal to the fovea and extending to the entire perifoveal region. While leakage is detectable on fluorescein angiography, there is no evidence of increased retinal thickening. Cystic spaces are evident within the retina using OCT and these spaces are thought to reflect the loss of retinal tissue. Occasionally, sub-retinal neovascularization develops and arises from the retinal circulation.[14]

Retinal **imaging**

Retinal imaging is an integral part of patient management and is required for diagnosis and monitoring response to therapy.

Fundus photography
Colour fundus photography provides a record of the appearance of the macular retina.

Fundus Fluorescein angiography (FFA)
FFA is currently the gold standard for diagnosing CNV in AMD.
Colour photographs must accompany the FFA as they yield important additional information on the composition of the macular lesion allowing interpretation of the FFA. Haemorrhage, pigment and exudate all of which are seen as dark areas on FA are easily distinguished from each other on colour images. Early phases of the angiogram must be captured as they are important for the visualization of the choroidal phase and the early arterial phases when pathology is better seen before obscuration of details by leakage and pooling of fluorescein dye.

Late images (10 minutes) are also important for distinguishing late leakage from drusen (which can take up fluorescein but which fade towards the end of the fluorescein run) and RPE window defects, and inactive scars. This is necessary to distinguish active from inactive pathology, which may be important for initiating or continuing treatment.

ICG angiography (ICGA)
Indocyanine green (ICG) is an adjunctive dye to fluorescein which is used to visualize the choroidal circulation. This dye has higher binding to plasma protein and hence does not egress through the fenestrate of the choroidal vessels, instead remaining within the vascular compartment. Choroidal vessel morphology is therefore better delineated. ICGA is imaged using infrared wavelengths which can pass through the RPE and blood therefore permitting visualization of pathology which can block the transmission of wavelengths that excite fluorescein. ICGA also has some limitations and very thick blood or pigment can reduce or block transmission of the infrared wavelengths and the emitted light is of lower intensity compared with fluorescein. The use of the scanning laser ophthalmoscope (SLO) with video capture can however yield images of high resolution. Video ICGA also allows better imaging of RAP. As ICG dye does not leak into the subretinal and subpigment epithelial spaces to the same extent as fluorescein the enhanced definition of the vascularised tissue as a hotspot is possible and a combination of FFA and ICGA can produce complementary information. A dose of 25mg of ICG in aqueous solution is usually injected intravenously and images acquired for up to 30 minutes.

Optical coherence tomography (OCT)
OCT may be used for screening the macula prior to performing more invasive imaging such as FFA. OCT alone may be able to provide sufficient information to permit decisions on clinical management and follow up, however it does not replace FFA in diagnosing CNV in AMD. To obtain relevant information appropriate high quality scans with the more recent generation of OCT's are required. As high speed spectral domain OCT machines perform many thousands of scans across the macula, pathology is less likely to be missed and the technology will contribute in helping distinguish different diagnoses.

Practical Points
- Fluorescein angiography is indicated to determine the extent, type, size and location of CNV.
- ICGA is indicated when assessing patients with macular haemorrhage or suspected of having, polypoidal choroidal vasculopathy, retinal angiomatous proliferative lesions or non-vascularised vs. vascularised PEDs.
- Good resolution OCT is mandatory for monitoring response to therapy.

Polypoidal choroidal vasculopathy (PCV) is another component of the spectrum of exudative AMD. PCV are seen as focal areas of abnormal dilated choroidal vessels

and result in a highly exudative picture with considerable accumulation of lipid and or haemorrhage in the subretinal space. These are best visualised by ICG angiography.

Therapies for management of **AMD-CNV**

Laser photocoagulation
The objective of this treatment is to destroy the neovascular complex with heavy confluent laser, and thereby try to reduce further loss of vision resulting from its further enlargement and/or ongoing leakage.

Practical Points
- Patients undergoing thermal laser treatment should have it performed as soon as possible and within a week following fluorescein angiography. Laser treatment should be avoided for subfoveal or juxtafoveal lesions.
- Patients should be warned that treatment will produce a permanent scotoma and that they will need monitoring. Recurrences are usually subfoveal and if this happens alternative treatments may be required.
- Patients should be examined 2 weeks following laser to confirm obliteration of the CNV and fluorescein angiography performed. Patients should be reviewed 4- 6 weeks later and thereafter depending on clinical findings.
- The majority of CNV recurrences after photocoagulation occur in the first year.

Photodynamic therapy with Verteporfin (vPDT)
This is a procedure whereby the photosensitising dye Verteporfin (Visudyne, Novartis) is given intravenously. Verteporfin is taken up selectively by rapidly proliferating endothelial cells which have increased expression of low-density lipoprotein (LDL) receptor. This is followed by the delivery of laser light of a wavelength of 689nm to the CNV lesion as a single spot with a diameter 1000μm larger than the greatest linear diameter of the lesion. The energy from the laser is taken up by the Verteporfin, and this leads to damage to vascular endothelial cells and thrombotic occlusion of the blood vessels within the CNV lesion. The major advantage of this approach over laser photocoagulation is that there is minimal damage to the overlying retina and choroid.[13,16]

Practical Points
- vPDT is no longer justified as monotherapy for nAMD.
- PDT is recommended only in patients with polypoidal choroidal vasculopathy (PCV). It is recommended that it is performed within 1-2 weeks of fluorescein angiogram / ICGA and then as required 3 monthly.
- It should be clearly explained to patients that treatment with PDT will reduce the risk of moderate and severe visual loss but that most patients will still lose vision

and visual improvements are unusual.
- Severe vision loss can occur immediately after treatment in 1-4% of patients and this may be permanent in a small proportion of cases.
- Idiosyncratic back pain occurs in 1-2% of patients which resolves when the infusion is stopped.
- Patients should be advised to avoid direct sunlight exposure for 2 days following treatment.

Anti-angiogenic therapy

Ranibizumab (Lucentis, Genentech Inc/Novartis)
Ranibizumab is a recombinant, humanised Fab fragment of a monoclonal antibody with a high affinity for VEGF A. Ranibizumab binds and inactivates all isoforms of VEGF, including the soluble VEGF isoforms 110, 121 and 165 and the tissue-bound isoforms 189 and 206.17,18 While bevacizumab was developed for long systemic retention in the treatment of metastatic cancer, ranibizumab was designed for rapid systemic clearance by removing the Fc fragment from the parent molecule.19 Additionally, the affinity of the compound for VEGF was enhanced by modification of five amino acids. Ranibizumab, with its molecular weight of 76 kDa, was found to penetrate the retina well after intravitreal injection.20 With a short systemic half-life and a rapid systemic clearance, the systemic safety of ranibizumab is extremely high.[21] Ranibizumab monotherapy has, therefore, become the reference standard for treatment of CNV.

Bevacizumab (Avastin, Genentech Inc/Roche)
Bevacizumab is a full-length recombinant monoclonal antibody that binds all VEGF isoforms. Bevacizumab is derived from the same monoclonal antibody as ranibizumab, therefore it is likely to recognize the same epitope on all isoforms of VEGF as ranibizumab, but has a lower binding affinity for VEGF than ranibizumab. The serum and vitreous half lives of bevacizumab are longer than those of ranibizumab. Bevacizumab was designed for use as a cancer therapeutic and is not licensed for ocular usage in Malaysia.

Aflibercept (Eylea, Regeneron/Bayer)
Aflibercept is a fusion protein which inhibits all isoforms of VEGF-A, VEGF-B, as well as placental growth factor. Aflibercept promises to decrease injection frequency and appears to serve as an alternative drug for patients who are less responsive to previously approved anti-VEGF drugs.

Combination **Treatments**

PDT and anti-VEGF therapy
A major limitation of VEGF inhibition therapy is the need for repeated intravitreal

injection with its attendant risks of endophthalmitis, retinal detachment and traumatic cataract. vPDT's role as an adjunctive agent is currently being explored. Combination treatments of ranibizumab and PDT have been tried and appeared to reduce the need for retreatment, but visual results have not been as good as ranibizumab alone for wet AMD. An exception is in the treatment of polypoidal choroidal vasculopathy (PCV), for which vPDT has been shown, in the 'EVEREST' study, to produce superior anatomical outcomes, either alone or in combination with ranibizumab, than ranibizumab alone.23

Treatment with anti-VEGF agent is indicated when;

- There is active subfoveal neovascularisation of any lesion type
- In patients with occult CNV with minimal symptoms or without documented evidence of disease progression a period of observation can be undertaken.

Progression is defined by the presence of at least one of the following criteria:

- The appearance of sight threatening CNV which was not previously suspected or thought to be present.
- Evidence of new haemorrhage and/or subretinal fluid.
- A documented recent visual decline in the presence of CNV.
- An increase in CNV size between visits.

- There should be no significant permanent structural damage to the fovea before treatment is commenced. Significant structural damage is defined as:
  - longstanding fibrosis or atrophy in the fovea,
  - or a large disciform scar with sub-retinal fibrosis and no evidence of disease activity surrounding or adjacent to it
  - for these cases, low vision aids may be helpful

Associated clinical features affecting treatment;

- Predominantly haemorrhagic lesions: Foveal haemorrhage or haemorrhage of greater than 50% of the total CNV lesion, are not considered reasons to withhold treatment with anti-VEGF
- Raised intraocular pressure: Elevated intraocular pressure (IOP), even of >30mm Hg, should not preclude treatment provided the IOP is treated simultaneously.
- Intraocular surgery: It is advised that in the presence of exudative AMD and co-existing cataract, the former should be treated and the CNV activity controlled prior to cataract surgery, wherever possible. If CNV is diagnosed after intraocular surgery or there is reactivation of an existing CNV, treatment with anti-VEGF can be commenced immediately. However, attention should be paid to the cataract wound.

Criteria for not **commencing treatment**

It is recommended that treatment with anti-VEGF therapy should not be commenced in the presence of

- Permanent structural damage in the fovea.
- Evidence or suspicion of hypersensitivity to anti-VEGF agent. Such evidence should lead to avoidance of therapy, and alternate treatments sought.
- Recent thrombo-embolic phenomena, including MI or CVA in the preceding 3 months, or recurrent thrombo-embolic phenomena.

Drug Holding and **Cessation of Therapy**

Consider discontinuing treatment if:

i. There is no disease activity
   The disease should be considered to have become inactive when there is:
   a. Absence of FFA leakage or other evidence of disease activity in the form of increasing lesion size, or new haemorrhage or exudates (i.e. no increase in lesion size, new haemorrhage or exudates) even if there is persistent fluid on OCT.
   b. No re-appearance or further worsening of OCT indicators of CNV disease activity on subsequent follow up following recent discontinuation of treatment.
   c. No additional lesion growth or other new signs of disease activity on subsequent follow up following recent discontinuation of treatment.
   d. No deterioration in vision that can be attributed to CNV activity.

ii. There has been one or more adverse events related to drug or injection procedure including:
   a. endophthalmitis
   b. retinal detachment
   c. severe uncontrolled uveitis
   d. other serious ocular complications attributable to ranibizumab (drug) or injection procedure
   e. thrombo-embolic phenomena, including MI or CVA in the preceding 3 months, or recurrent thrombo-embolic phenomena which are thought to be related to treatment with anti-VEGF therapy
   f. other serious adverse events (SAE) e.g. hospitalisation

iii. Reduction of BCVA in the treated eye to less than counting fingers attributed to AMD. Patients should be then referred to the medical retina unit for reassessment.

iv. There is evidence of deterioration of the lesion morphology despite optimum treatment. Such evidence includes progressive increase in lesion size confirmed with FFA, worsening of OCT indicators of CNV disease activity or other evidence of disease activity in the form of significant new haemorrhage or exudates despite optimum therapy over a 3 consecutive visits. These cases also should be referred to the medical retina unit for assessment.

Practical Points

- Patients should be advised of the need for frequent monitoring when commencing a course of intravitreal drug treatment for AMD. They should be prepared to attend the eye clinic every 4 weeks for the first 3 months for monitoring and treatment. Treatment and follow-up may need to be continued for up to and beyond 2 years.

  Anti-VEGF treatment will only result in significant vision improvement in a third of patients. The majority will maintain vision and some 10% will not respond to therapy.

- Patients should understand the risk associated with intravitreal injections and be instructed to report symptoms suggestive of endophthalmitis without delay.

Indications for referral **to the medical retina unit**

1. Doubt of diagnosis
2. Need for ICGA in predominatly haemorrhagic lesions
3. Reduction in vision due to increasing disease activity after the 1st or 2nd injection. These cases should be referred within 2 months.
4. Recurrence of disease activity after the 1st three injections of ranibizumab. During the subsequent reviews if there is a recurrence, these cases should be referred within 2 months.

Ideals to aim for:

As set out above, immediate rapid access to retinal specialists with expertise in the management of exudative AMD for all patients should be available, irrespective of geographic location. It is recommended that if in doubt, the digital images of the color photos and angiograms be sent to a medical retina unit in order for them to advise a plan of management. The consultation and plan should be formulated by

a specialist with medical retinal expertise within two weeks of diagnosis, and, there should be no more than four weeks between evaluation and treatment.

All patients suspected to have treatable exudative AMD by the general ophthalmologist should be assessed by the measures stated and if in doubt referred directly to the nearest Medical retina service clinic. The anti-VEGF injection may be given by the general ophthalmologist at the referring centre. It is suggested that those general ophthalmologists treating patients with exudative AMD undergo a training period for credentialing at a medical retina unit for a period of 3- 6 months.

Practical Points

Patients with exudative macular degeneration must be assessed and treated promptly to gain maximum benefit from treatment. The Medical Retina Specialist has a leading role in ensuring clear referral pathways exist within Ophthalmology departments.

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