Common eye disorders in the elderly – a short review

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Abstract

As the eye ages, certain changes occur that may affect vision. *Presbyopia* is corrected by the use of reading glasses. *Cataracts* are common and vision can be restored following a reasonably simple operation. Visual loss due to *glaucoma* can be minimised by early detection and treatment, but once vision has been lost it cannot be recovered. Much research is being done on the treatment of *age-related macular degeneration,* but this currently remains the main cause of irreversible loss of vision in the elderly. Results of *macular hole* surgery are improving due to improved surgical techniques and better diagnostic equipment (Optical Coherence Tomography). Underlying vascular disease (systemic hypertension, diabetes, atherosclerosis, vasculitis) is usually present in patients with *retinal artery occlusion, retinal vein occlusion* or *anterior ischaemic optic neuropathy*.

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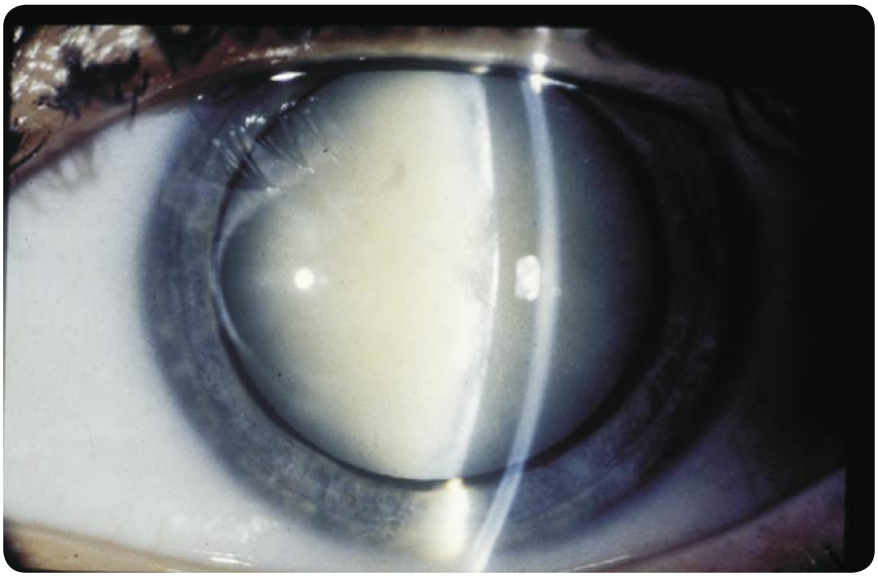
Introduction

Loss of vision in the elderly can have a multitude of causes. Often the remedy is simple, for example prescribing spectacles (as in the case of presbyopia) or a reasonably small operation (as in the case of cataract) and all that is needed from the general practitioner, is to make the diagnosis, reassure the patient and refer him /her to the eye care practitioner for definitive management. In cases of profound and sudden loss of vision, where vascular occlusion (retinal artery or vein occlusions and anterior ischaemic optic neuropathy) is suspected, it is however important for the general practitioner to be able to recognise the entity and institute initial emergency management prior to referral. These patients also usually have some underlying vascular disease (systemic hypertension, diabetes, atherosclerosis or vasculitis), which may need investigating. Age-related macular degeneration (AMD) remains the main cause of irreversible loss of vision in the elderly, and unfortunately, though there are numerous treatment options available, until recently, results of most of these have been disappointing. Success of treatment has been measured in terms of less loss of vision rather than a gain in vision. Recent advances in the treatment of AMD with intraocular injections are however showing promise with many patients regaining some of the vision initially lost.

Presbyopia

Presbyopia ("farsightedness of aging") is the gradual loss of accommodative response of the eye, resulting from the loss of lens elasticity. It is the most common and one of the earlier age-related disorders of the eyes. Symptoms of presbyopia usually begin between the ages of 40 and 45 years, although they may begin earlier in hyperopes (farsighted people) and later in myopes (nearsighted people).[1] Patients notice that they need to hold reading material further away to read clearly. The problem is easily remedied by the use of reading glasses (convex or "plus" lenses). The rule of thumb for

Figure 1: Mature cataract



choosing these glasses is age less 40 divided by 10. Thus, a fifty-year-old patient would probably need +1 dioptre reading glasses.

Cataract

Age-related cataract is a very common cause of visual impairment in older adults. The prevalence of cataracts in people aged between 65 and 74 years is 50% and it increases to 70% in those over the age of 75 years.[2] There are three main types of age-related cataracts: nuclear, cortical and posterior subcapsular. Components of more than one type usually are present.

Nuclear cataract is caused by an excessive amount of hardening and yellowing of the lens nucleus and can be seen as a central black ring against the red reflex when examining the eye with an ophthalmoscope with the pupil dilated. It causes greater impairment of distance rather than near vision. Early in the condition, the hardening of the lens nucleus leads to a change in the refractive index of the lens, which in turn leads to a myopic shift in refraction. This temporarily enables the presbyopic patient to read without reading glasses and is sometimes referred to as "second sight". In advanced cases of nuclear cataract, the lens nucleus becomes opaque and brown and is called a brunescent nuclear cataract.

Cortical cataracts start as wedge-shaped peripheral lens opacities, often called "cortical spokes", which are seen as dark shadows against the red reflex. The early symptoms are glare from intense focal light sources such as the headlights of oncoming cars and, sometimes, monocular diplopia. They enlarge and coalesce with time. When the entire cortex becomes opaque and white, the cataract is said to be mature (see Figure 1).

Posterior subcapsular cataracts are seen more often in younger patients. The central posterior pole of the lens is affected and near visual acuity is more affected than distance visual acuity. Patients also complain of glare and poor vision under bright lighting conditions. A dense central opacity is seen against the red reflex.

The treatment of cataract entails its surgical removal with the implantation of an intraocular lens (IOL) (see Figure 2). The modern technique of small incision, sutureless phacoemulsification with foldable lens implantation is a safe procedure, performed under local or topical anaesthesia as ambulatory/day case surgery and results in immediate visual recovery.

A more recent advance in cataract surgery is the implantation of multifocal IOLs, which simulate accommodation by allowing pseudophakic patients to visualise images at different focal distances (near and far).[3]

Discovering co-existing pathology does not preclude the patient from having cataract surgery, but may necessitate advising the patient on a guarded visual prognosis. There is some controversy whether cataract surgery can hasten the progression of some disease processes, such as age-related macular degeneration, glaucoma and diabetic retinopathy. Furthermore, the choice of IOL and postoperative management may differ from routine cases.[4]

The decision regarding whether or not cataract surgery is warranted is based not on a specific visual acuity, but rather on whether reduced visual function substantially interferes with a patient's desired activities.

Figure 2: IOL in the capsular bag following cataract surgery

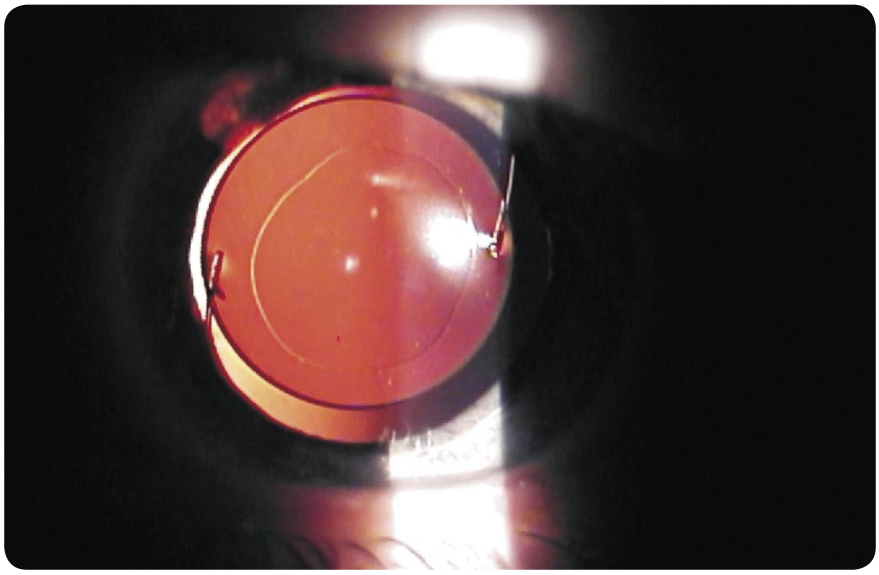
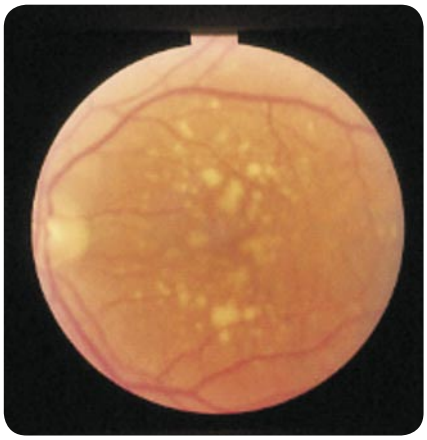


Figure 3: Drusen at the posterior pole



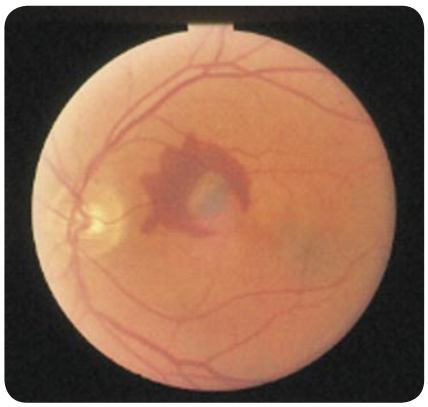
Glaucoma

From the literature from the USA, primary open angle glaucoma (POAG) occurs in 1.3% to 2.1% of the population over the age of 40 years and the incidence increases to nearly 15% in the population over 80 years.[5] The figures for South Africa are not readily available, but it is known that POAG occurs more frequently, has an earlier onset and is associated with higher intraocular pressures in black people than in white people. [See article in this issue on glaucoma – p46]

Age-related macular degeneration (AMD)

AMD is the leading cause of blindness in patients over the age of 50 years in the Western world. At least

Figure 4: Choroidal neovascular membrane with surrounding subretinal haemorrhage



10% of people aged between 65 and 75 will have lost some central vision due to AMD. In those older than 75 years, 30% will be affected to some degree.[6] It is more common in Caucasians. The earliest clinical manifestation of AMD is the appearance of small, discrete, slightly elevated, yellow-white spots at the posterior poles of both fundi, called drusen (see Figure 3). They are rarely seen before the age of 45, are not uncommon between the ages of 45 and 60 and are frequently seen thereafter. Although many eyes with drusen maintain good vision throughout life, a significant number of elderly patients develop decreased central vision due to advanced AMD. Two main types of advanced AMD are recognised – "dry" or non-exudative and "wet" or exudative.

Dry AMD accounts for approximately 90% of cases and typically causes gradual mild to moderate visual impairment over months to years.[6] Clinically it is characterised by sharply circumscribed circular areas of retinal pigment epithelial (RPE) atrophy with varying degrees of choriocapillaris loss at the macula. Later on, larger choroidal vessels become prominent within the atrophic areas and the pre-existing drusen disappear.

Wet AMD is sometimes referred to as "neovascular" AMD. Even though it is less common than dry AMD, only accounting for approximately 10% of cases, it accounts for 90% of cases of more severe visual loss (visual acuity < 6/60).[7] New vessels grow from the choriocapillaris into the sub-RPE space and later into the subretinal space. Leakage or haemorrhage from these vessels leads to serous or haemorrhagic detachment of the RPE and/or sensory retina. Initial symptoms include metamorphopsia (distortion of vision) and blurring of central vision. Examination of the fundus reveals a grey-green, slightly elevated lesion, which is often accompanied by intraretinal or subretinal haemorrhage and/or lipid exudation (see Figure 4).

The haemorrhagic episode is followed by a gradual organisation of the blood and eventually the formation of a fibrous disciform scar at the fovea (see Figures 5a and 5b).

Figure 5a: Right and left fundi with exudative AMD

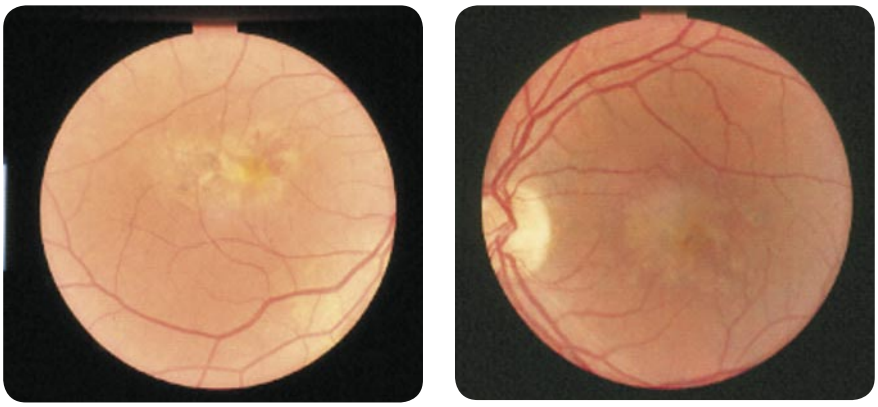
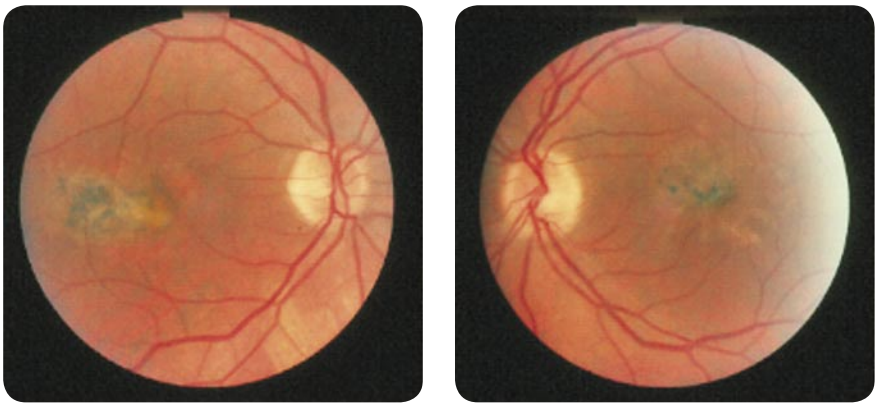


Figure 5b: Same patient with bilateral disciform scars a year later



Management

Studies have shown that smokers are twice as likely to have AMD than are non-smokers.[8] It is therefore very important to encourage smokers to stop smoking. The Age-Related Eye Disease Study (AREDS) Research Group[9] has found that, in patients with a high risk of AMD progression (those with extensive intermediate drusen, or at least one large druse, or noncentral geographic atrophy, or advanced AMD in one eye), there was a statistically significant reduction in the development of advanced AMD in patients using antioxidants and zinc (see Table I).

Table I: AREDS formulation

Vitamin C	500 mg
Vitamin E	400 IU
Beta Carotene*	15 mg
Zinc	80 mg
Cupric oxide	2 mg

*Not to be given to smokers
(Very high dosages not without risk)

Historically, laser photocoagulation was the mainstay of treatment.[10] The intense thermal treatment attacked the choroidal neovascular membranes (CNVMs) and resulted

in retinal death in the treated area. Unfortunately, this can only help to maintain central vision if the lesion is outside the foveal centre and most cases of wet AMD present with foveal involvement

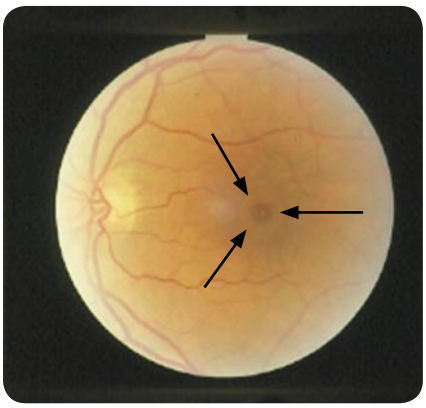
Beginning in 2001, photodynamic therapy (PDT) became the standard of care for treatment of most patients with wet AMD.[11] The treatment uses a photosensitive intravenous dye that is activated by a low-dose laser. This leads to closure of the growing blood vessels while selectively sparing the overlying retina. However, it is only effective in a small subset of CNVMs and, although statistically better than natural history, most patients still lose visual acuity and only a very small percentage regain lost sight.

Most recent advances have been in the use of antiangiogenic substances to attack the underlying mechanisms driving the process. These include Macugen, Lucentis, Avastin (all given as repeated intravitreal injections), Retaane (administered as a sub-Tenon's injection) and Squalamine (given intravenously). Thus far, only Macugen has obtained FDA approval for use in AMD and the visual results compare favourably to those of PDT.[12,13]

Macular hole

Idiopathic macular holes occur primarily in women in their sixth to eighth decades.[14] A full-thickness macular hole is visible as a sharply demarcated round or ovoid defect at the fovea, often associated with yellow precipitates on the underlying RPE and a narrow rim of subretinal fluid (see Figure 6). The incidence of bilaterality is estimated to be 25% to 30%.

Figure 6: Full-thickness macular hole with rim of subretinal fluid



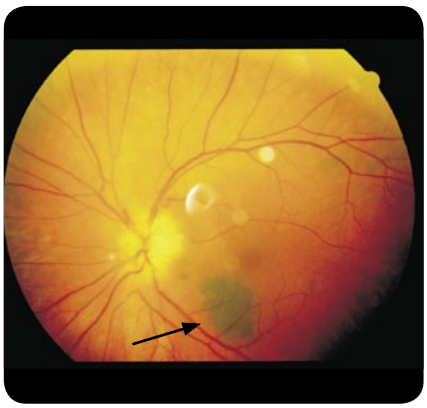
Surgery is considered when the vision drops below 6/18. The success of the surgery is dependent on the size and duration of the hole.

Anterior ischaemic optic neuropathy (AION)

AION is a relatively common cause of severe visual loss in the middle-aged and elderly. It is the segmental or generalised infarction of the anterior part of the optic nerve caused by occlusion of the short posterior ciliary arteries.[15] It can be classified as being arteritic or non-arteritic. Arteritic AION is associated with giant cell arteritis (GCA). It is a medical emergency, and prevention of blindness depends on its prompt recognition and treatment (see Tables II and III).

Non-arteritic AION typically occurs as an isolated event in patients between the ages of 45 and 65 years, who are either healthy or have hypertension as the only sign of vascular disease. Visual loss is usually less profound than with arteritic AION. Fundus examination of both arteritic and non-arteritic AION shows a pale swollen disc, often associated with a few splinter-shaped haemorrhages (see Figure 7).

Figure 7: Pale swollen disc seen in a patient with AION (incidental finding of a choroidal naevus indicated by arrow)



Investigations to exclude GCA include: ESR, C-reactive protein (CRP) (both high in GCA) and a temporal artery biopsy to histologically confirm the diagnosis.

Table II: Symptoms and signs of GCA

Scalp tenderness
Jaw claudication
Headache
Polymyalgia rheumatica
Sudden profound loss of vision
Afferent papillary defect
Tender, inflamed and nodular temporal arteries

Retinal vascular occlusions

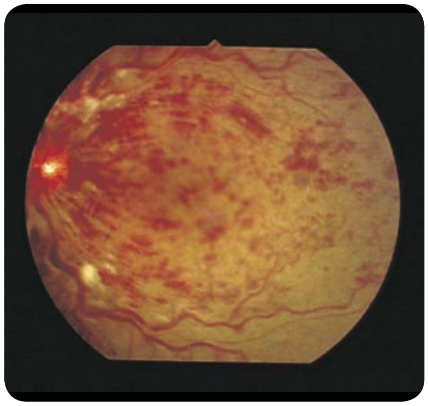
Retinal vein occlusions mostly affect patients in the sixth or seventh decades of life.[16] Other predisposing diseases include systemic hypertension, diabetes, blood dycrasias, raised intraocular pressure, hypermetropia and periphlebitis. Patients present with a sudden loss of vision, often noticed upon waking in the morning.

Table III: Treatment of arteritic AION

Drug & dosage	Duration
Methylprednisolone 1 g daily IV plus Prednisone 80 mg daily orally	3 days
Reduce Prednisone to 60 mg daily orally	4 days
Reduce Prednisone by 5 mg per week to 10 mg daily orally	10 weeks
Prednisone 10 mg daily orally (maintenance)*	1 – 2 years

* Serial ESR and CRP measurements aid decision on when to discontinue maintenance therapy

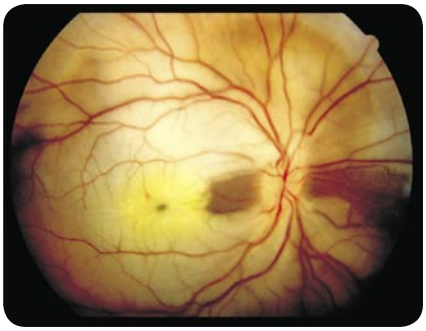
Figure 8: Retinal haemorrhages and cotton-wool spots in a central retinal vein occlusion



On funduscopy one can see dilated and tortuous veins, flame-shaped as well as dot-blot haemorrhages, cotton-wool spots and retinal oedema (see Figure 8). Ischaemic vein occlusions may be complicated by the development of new vessels – either on the iris (central retinal vein occlusions) or on the retina (branch retinal vein occlusions). These patients need monthly reviews for six months and, should new vessels develop, they need treatment with panretinal photocoagulation (laser).

Retinal artery occlusions are usually caused by emboli from either the heart or the carotid arteries. A patient with a central retinal artery occlusion will present with an acute and profound loss of vision and will have an afferent pupillary defect. The retina appears white as a result of cloudy swelling caused by intracellular oedema (see Figure 9). The thinner central fovea, devoid of inner retinal layers, contrasts with the surrounding opaque retina, giving rise to the "cherry-red-spot" appearance. In about 20% of cases, a portion of the papillomacular bundle is supplied by one or more cilioretinal arterioles from the ciliary circulation – in these patients some central vision may be spared.

Figure 9: Central retinal artery occlusion with sparing of the cilioretinal artery



The treatment of an arterial occlusion (Table IV) is aimed at restoring retinal circulation as quickly as possible. Retinal tissue cannot survive ischaemia for more than a few hours, but because most occlusions are not complete it is reasonable to treat all cases seen within 48 hours.

In conclusion

- Cataracts are the most common cause of reversible visual loss. Cataract surgery is the most cost-effective surgical procedure, and the second most cost-effective medical intervention (after immunisation)
- Elderly patients must be screened for glaucoma to prevent irreversible painless loss of vision
- Smokers are twice as likely to have advanced age-related macular degeneration as non-smokers
- Sudden loss of vision in the elderly may be caused by giant cell arteritis: prompt diagnosis and treatment is imperative to prevent second eye involvement
- Most retinal vein occlusions have hypertension or diabetes as the underlying cause

Acknowledgement

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See CPD Questionnaire, page 50

This article has been peer reviewed

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Table IV: Emergency treatment of central retinal artery occlusion

Treatment	Effect
Intermittent ocular massage for at least 15 minutes	Lower IOP, increase blood flow, dislodge embolus
Inhalation of 95%/5% oxygen-carbon dioxide mixture	Vasodilatation
Oral acetazolamide	Lower IOP
Anterior chamber paracentesis	Lower IOP