Common Eye Diseases of Elderly People: Identifying and Treating Causes of Vision Loss

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Key Words
Age-related macular degeneration · Cataract · Diabetic retinopathy · Glaucoma · Verteporfin therapy

Abstract
Of the 38 million people who are blind, the majority, 22 million, are over the age of 60 years (World Health Organization 1994 estimate). Even more people (110 million) suffer from low vision. These factors, combined with an increase in life expectancy [2], highlight the need for effective eye care for elderly people to minimize vision loss from age-related conditions such as age-related macular degeneration (AMD), cataract, glaucoma, and diabetic retinopathy. Of these, AMD is the leading cause of registered blindness in people over the age of 50 years in the western world. However, until recently, the treatment options for people with AMD have been severely limited. Verteporfin therapy is a new treatment that is efficacious and safe in selected patients with AMD who are at high risk of central vision loss. Physicians who are in regular contact with elderly people can help to minimize vision loss in this group of patients by being alert to the symptoms and signs of age-related eye diseases. This paper reviews each of the common eye diseases, with an emphasis on AMD because of the recent advances in treatment.

Introduction

Of the 38 million people who are blind worldwide [1], 22 million are over the age of 60 years (World Health Organization 1994 estimate). Even more people (110 million) suffer from low vision. These factors, combined with an increase in life expectancy [2], highlight the need for effective eye care for elderly people to minimize vision loss from age-related conditions such as age-related macular degeneration (AMD), cataract, glaucoma, and diabetic retinopathy. Vision loss is only one aspect of the impact that these eye diseases have on the person and is frequently accompanied by a corresponding decrease in their ability to carry out activities of daily living and an increased risk of depression [3]. These aspects, which are often undervalued [4], may compromise an elderly person's ability to live independently.

Early diagnosis and prompt referral of patients with eye disease are important to try to maximize treatment benefits. When primary care physicians or primary health care professionals are aware of both the clinical signs and symptoms and the treatment options that are available for the common age-related eye diseases, they can facilitate the improvement of these conditions in their patients. In this article, the diagnosis and treatment options for the
common age-related eye diseases are reviewed, with an emphasis on AMD because of recent advances in treatment.

**Age-Related Macular Degeneration**

AMD is the most common cause of severe vision loss in people over 50 years of age in the western world [5]. It is a degenerative disorder of the retinal macula (fig. 1) that results in loss of central vision. AMD is classified into two types: non-neovascular (also known as non-exudative or atrophic AMD) and neovascular (also known as exudative or serous AMD).

Non-neovascular AMD accounts for approximately 80% of all AMD and is associated with the formation of drusen [6]. These are localized deposits of extracellular material that often can be seen with direct dilated ophthalmoscopy as yellow deposits in the center of the retina (fig. 2). More than 50% of people over 70 years of age have drusen [5]; however, drusen cannot be taken as an absolute indication of AMD. Almost all cases of severe vision loss in non-neovascular AMD result from geographic atrophy. Atrophy of the retinal pigment epithelium (RPE) is thought to be followed by loss of macular photoreceptors and of the blood supply to the RPE, creating blind spots. Neovascular AMD accounts for only about 20% of all AMD, but is responsible for 90% of cases of severe, irreversible central vision loss [6].

Progression from non-neovascular AMD to neovascular AMD occurs in approximately 10–20% of people with AMD [6]. The time period for the transition from non-neovascular to neovascular AMD is variable, ranging from a few months to several years. Neovascular AMD is characterized by choroidal neovascularization (CNV), the formation of new, fragile blood vessels growing from the choroid into the subretinal and pigment epithelial space (fig. 1). These vessels leak blood and fluid, resulting in scarring, which can reduce visual acuity.

Fluorescein angiography is the most frequently used method to visualize choroidal neovascular lesions. This technique can be used to classify the lesion according to its anatomical location in the macula: subfoveal (directly under the fovea), juxtafoveal (adjacent to the fovea), or parafoveal (outside the fovea).

**Fig. 1.** Anatomy of the eye (a) and the macula (b), with a cross-section showing damage resulting from AMD (c). Reproduced with permission from Novartis Ophthalmics AG.

**Fig. 2.** Color fundus photographs showing the appearance of (a) a normal healthy retina (Image obtained from National Eye Institute, National Institutes of Health), (b) AMD with drusen (arrows) (Reproduced with permission from Wilmer Photograph Reading Center), (c) diabetic retinopathy with macular edema (arrows) (Image obtained from National Eye Institute, National Institutes of Health).
Table 1. Symptoms and signs of age-related eye diseases

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Visual Acuity Decrease</th>
<th>Ophthalmoscopy</th>
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<tbody>
<tr>
<td>AMD Non-neovascular: blurred or distorted vision, central scotoma Neovascular: central scotoma, image distortion, increased glare sensitivity, decreased color perception, photopsias, formed hallucinations, blindness</td>
<td>Non-neovascular: slow decline or no change Neovascular: rapid, steady decrease in visual acuity</td>
<td>Non-neovascular: drusen, atrophy of the RPE (visible as alternating area of depigmentation and hyperpigmentation) Neovascular: drusen (especially if there are numerous (&gt;20), soft, large (&gt;6 mm in diameter) or confluent)</td>
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<td>Cataract</td>
<td>Decreased visual acuity, clouding of the visual field, double vision, near-sightedness, decreased color perception, reduced contrast sensitivity, increased glare sensitivity, blindness</td>
<td>Variable, depending on the extent and progression of glaucoma; visual acuity can stabilize without treatment</td>
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<tr>
<td>Glaucoma Early stages are asymptomatic. In the later stages – headaches or blurred vision after vigorous exercise, headaches accompanied by misty vision or haloes when reading in low illumination, reduced visual field, blurred vision, blindness</td>
<td>Slow, progressive decrease in open-angle glaucoma; can be very rapid in angle-closure glaucoma</td>
<td>Open-angle glaucoma: optic nerve cupping</td>
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<tr>
<td>Diabetic retinopathy Nonproliferative: asymptomatic Proliferative: blurred vision, floaters, loss of central vision, decreased color perception, poor night vision, blindness</td>
<td>No decrease until disease progresses to the proliferative stage when visual acuity loss can be rapid</td>
<td>Nonproliferative: microaneurysms, intraretinal hemorrhages, soft exudates, venous looping, venous beading, macular edema, dilated retinal veins Proliferative: proliferating endothelial cell tubules, vascular fibrosis, intraretinal and peri-retinal hemorrhages, vitreous hemorrhage</td>
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beneath the foveal avascular zone (FAZ), juxtafoveal (1–199 \( \mu \text{m} \) from the center of the FAZ), or extrafoveal (\( \geq 200 \mu \text{m} \) from the center of the FAZ). The visual prognosis is worst for subfoveal lesions; approximately 60% of CNV associated with AMD is subfoveal [7]. Fluorescein angiography can also be used to classify the pattern of fluorescence from CNV as classic, occult, or a mixture of both depending on its appearance. The development of classic CNV is associated with a trend toward an increased risk of vision loss within a year [8]. If all these factors are combined, it seems that the worst visual prognosis in AMD occurs in patients who have neovascular lesions that are subfoveal with a large classic component.

### Signs and Symptoms

Patients with non-neovascular AMD may experience slow, progressive loss of central visual function, which is most noticeable during tasks that require near vision, especially in the early stages of the disease [9]. Also, geographic atrophy may cause blurred or distorted vision. Neovascular AMD can cause a rapid deterioration of central vision: approximately 70% of eyes subfoveal CNV have a visual acuity of 20/200 with years of diagnosis [10]. Furthermore, once CNV developed in one eye, there is a significant risk that the second eye will develop CNV [11].

A decrease in central vision and a blind spot (scotoma in the visual field (fig. 3) are consequences of bleeding scarring from CNV (table 1). In addition, a patient’s neovascular AMD may experience image distortion (tromatopsia). For example, they may report that normally straight lines, such as pillars or fence posts, appear ‘wavy’ or ‘bent’. The patient may also report increased glare sensitivity, decreased color vision, sensation of flashing or sparkling lights (photopsias), and formed hallucinations [12]. Contrast sensitivity (the ability to distinguish between an object and its background) may also be reduced in patients with neovascular AMD [13].
atrophy of macula; areas of depigmentation, especially if they are large (>63 μm).

if lens can mottled vision. Vision loss resulting from AMD has a considerable impact on the patient's ability to undertake daily activities, especially if both eyes are affected [11]. The ability to read, recognize common household objects, and distinguish CNV harbor among facial expressions are all affected [14]. Furthermore, the vision loss associated with AMD means that patients have a greater risk of falls and hip fracture (scotomata) [15, 16]. These factors contribute to the decrease in a person's quality of life. However, it appears that clinicians may underestimate the effect that AMD has on the individual [4].

Several factors may delay a diagnosis of AMD. Despite a recent increase in public awareness, the prevalence of AMD and its potential impact on patients, awareness of neovascular AMD is not as widespread as it is for other eye diseases such as cataract or glaucoma – in a survey conducted in 1999, 70% of adults had not heard of AMD [17]. Also, elderly people often do not report the symptoms of vision loss [18]; they may ignore them, thinking their symptoms are a normal part of aging, or they may believe nothing can be done to help [19]. Alternatively, older people may assume that symptoms of AMD are due to the progression of cataracts. To help overcome these obstacles to early diagnosis, elderly people should be encouraged to have eye examinations every 1–2 years, even in the absence of symptoms (American Academy of Ophthalmology Policy Statement, 1990).

Who Is Most at Risk of AMD?
The single greatest risk factor for AMD is increasing age, although a number of other risk factors have been suggested. People who have neovascular AMD in one eye should be monitored very carefully because, over a 5-year period, approximately 42% of patients will develop the disease bilaterally [11]. Studies have found that the prevalence of AMD is associated with gender (women are more likely to have AMD than men), race (lower prevalence in Afro-Caribbean American and Hispanic people than Caucasian), and smoking. Other risk factors include a family history of AMD, high blood pressure, diabetes, and cardiovascular disease. People with these risk factors should be aware of the signs and symptoms of AMD and get regular eye examinations.
Fig. 4. When fixating on the center of an Amsler grid, a patient with neovascular AMD may perceive distortions or discoloration of the grid.

Asian people), and cigarette smoking [20, 21]. Family history also suggests genetic factors are involved [22]. Other possible risk factors that have been identified include cardiovascular disease, elevated serum cholesterol levels, and hypertension [11].

Studies suggest that vitamin [23] and mineral [24] supplements may contribute to a reduction in the risk of developing a degenerative eye disease such as AMD. Antioxidant supplements may prevent cellular damage in the retina by reacting with, and thereby removing, free radicals produced in the process of light absorption [25]. However, the use of antioxidant vitamin and mineral supplements in AMD is controversial and further investigation is necessary [25].

**Identifying Neovascular AMD**

Direct ophthalmoscopy after pupillary dilation can reveal features that suggest CNV secondary to AMD. In particular, the presence of drusen, especially if they are numerous (>20), soft, large (>63 μm), or confluent, carries a significant risk for the development of neovascular AMD [11]. Patients at risk of developing neovascular AMD should understand the nature of the disease and self-test regularly, perhaps with an Amsler grid. The Amsler grid (fig. 4), a printed grid of straight lines, is probably a useful tool for monitoring the central vision and can be used as a means to detect early, subtle changes, as well as monitoring changes once they have been detected. The grid should be held at a normal distance and the patient should focus on the dot, one eye at a time (covering the other with their hand while wearing any corrective vision aids that they normally wear). Testing each eye separately helps the patient to recognize visual symptoms that are in the other eye. They should be asked to report any wavy, bent distorted lines, blurred vision, discolored lines, or spots [26]. If distortions are detected with the Amsler grid, the patient should be assumed to have CNV unless there is evidence to the contrary. These patients should be promptly referred for treatment by an ophthalmologist to help to minimize the risk of vision loss [27].

For patients with neovascular AMD in one eye, the presence of five or more small drusen or one or more drusen in the fellow eye, focal hyperpigmentation, tinal or intraretinal blood or hemorrhage, or systolic hypertension may increase the risk of CNV in the eye [11, 28]. Referral is advisable for these patients to ensure CNV is detected at an early stage.

**Treatment Options for Neovascular AMD**

During the 1990s, the Macular Photocoagulation Study (MPS) Group published several papers showing that laser photocoagulation could be effective in patients with neovascular AMD. Recently, photodynamic therapy with verteporfin (Visudyne®️, Novartis AG) has been approved for use in treatment of selected cases of neovascular AMD at this time, though there is no cure or preventative treatment for neovascular AMD at this time, these treatments reduce the risk of vision loss.

**Laser Photocoagulation**

Although laser photocoagulation may limit the extent of damage caused by neovascular AMD, it is applicable only to a small subset of patients [29]. Strict eligibility criteria for treatment with laser photocoagulation rely on the composition, size, and location of the CNV. Accordingly, this treatment has been recommended for use in patients with juxtafoveal or extrafoveal lesions, whom it confines lesion growth to a smaller area without treatment. The overall benefit of laser photocoagulation decreases as choroidal neovascular lesions tend towards the FAS. When the CNV is subfoveal, photocoagulation can cause an immediate and permanent decrease in visual acuity due to destruction of the outer retinal layers.
ing healthy retinal photoreceptors, leaving an absolute scotoma [30]. Furthermore, a study showed that after 36 months, 82% of eyes with subfoveal CNV treated with laser photocoagulation were recorded as having a visual acuity of 20/200 or worse [30].

Verteporfin Therapy

Verteporfin therapy is a minimally invasive procedure that can be carried out on an outpatient basis by trained ophthalmologists. It is a two-step process in which the drug (verteporfin) is administered by intravenous infusion, then activated by a laser. After intravenous infusion, verteporfin accumulates in the choroidal neovasculature. A low-intensity, non-thermal laser that uses a specific wavelength of light activates the drug, which can cause highly localized damage to the choroidal neovasculature. The whole procedure takes approximately 20 minutes. Verteporfin has a short plasma half-life, which results in rapid clearance from the body [31]. Because the laser used to activate verteporfin is non-thermal, retinal function may be maintained in areas overlying the verteporfin-treated choroidal neovascular lesions [32]. Verteporfin therapy is therefore suitable for the treatment of substantially more patients with subfoveal lesions than laser photocoagulation, and without immediate loss of vision [13, 33].

Verteporfin therapy is approved for the treatment of neovascular AMD in patients with predominantly classic lesions (lesions in which the area of classic CNV is ≥ 50% of the area of the entire lesion on fluorescein angiography) that are subfoveal [13, 34]. The 2-year results of the treatment of AMD with photodynamic therapy (TAP) investigation showed that verteporfin therapy significantly reduced the risk of moderate and severe vision loss in eyes with predominantly classic subfoveal lesions at baseline [13]. In this subgroup, 65 (41%) of 159 verteporfin-treated eyes had lost at least 15 letters of visual acuity (the primary endpoint) compared with 57 (69%) of 83 eyes receiving placebo (p < 0.001) (fig. 5).

Recently, the verteporfin in photodynamic therapy (VIP) trial evaluated verteporfin therapy in patients with occult CNV secondary to AMD [35]. Data indicate that verteporfin therapy is effective at reducing vision loss in patients with occult CNV with no classic CNV with presumed recent disease progression. Verteporfin therapy appears to be safe and well tolerated; most adverse events were mild to moderate and transient in nature. Acute severe vision decrease (loss of ≥ 4 lines of visual acuity within 7 days of treatment) occurred in 1% of patients treated with verteporfin in the TAP investigation [13], and 4% of patients with AMD in the VIP trial [35]. Considering the large sample size of the TAP investigation, there were relatively few treatment-related ocular adverse events. Patients should be aware that following verteporfin therapy, they may experience a period of photosensitivity (3.5% of patients had photosensitivity reactions in TAP investigation) and should avoid exposure of unprotected skin or eyes to direct sunlight or bright indoor lighting (especially halogen lighting) for 48 hours.

One estimate suggests that verteporfin therapy will allow two to three times as many patients with CNV secondary to AMD to be effectively treated than were previously treatable [36]. This is likely to be an underestimate given the recent finding that verteporfin therapy has been shown to be effective at reducing vision loss in patients with occult CNV with no classic CNV with presumed recent disease progression [35].

Cataract

Cataract, which is a lens opacity that interferes with visual function, accounts for approximately 16 million cases of blindness (visual acuity <20/400, Snellen equivalent) worldwide (World Health Organization 1997 estimate). The prevalence of vision loss caused by cataract increases with age. In one study, the prevalence rose from
4.5% in people aged 52–64 years to 45.9% in people aged 75–85 years [37]. Also, the prevalence of cataract appears to be highest in African Americans [38].

Signs and Symptoms
A general blurring of vision (fig. 3) and sensitivity to bright lights (table 1) are the characteristic symptoms reported for cataract [39]. Opacity of the lens can vary from a small local opacity to a diffuse general loss of transparency. Typically, the first changes a patient becomes aware of are clouding of the visual field, double vision, or both [39]. As patients with cataract may experience progressively worsening nearsightedness, frequent prescription changes for vision aids may be required. Unfortunately, as cataract progresses, stronger glasses can no longer correct the worsening vision. Cataract can cause other visual disturbances; for example, as color vibrancy diminishes, the patient’s vision may take on a yellow tint. Reading may become difficult because of reduced contrast between letters and their background [39]. Patients may also report that they cannot drive at night due to glare from the headlights of oncoming cars.

Although cataract is most commonly a result of aging, it may occur secondary to inflammation, trauma, diabetes mellitus, ultraviolet radiation, or metabolic or nutritional disorders [39–41]. Limited evidence suggests that vitamin supplements, such as vitamins C, E, or B2 (riboflavin), may lower the risk of cataract—especially nuclear cataract [42, 43]. Benefits may be greatest if these nutrients are used for more than 10 years; however, the specific nutrients that may be responsible for lowering the risk of cataract have not been identified [43].

Treatment Options
Early detection is important because cataract is a progressive disease that is associated with a corresponding progressive decrease in vision. Vision aids can decrease glare, increase image contrast, correct for refractive errors, and correct for differences in vision between the affected and unaffected eye in patients with cataract. The extent of visual disability will determine the need for surgical intervention to remove cataract. Cataract surgery is associated with a small risk of vision decrease or permanent vision loss; however, standard cataract extraction with intraocular lens implantation results in improved vision in most patients [44].

Glaucoma
Approximately 6.4 million people worldwide affected by blindness due to glaucoma (World Health Organization 1997 estimate). Of those people who are blind (visual acuity of 20/200 or worse in the better eye) because of glaucoma, 75% are older than 65 age [45].

Glaucoma can be classified into two broad types: angle and angle-closure, each of which can be categorized primary or secondary. When the cause of glaucoma is known, it is termed primary glaucoma; it is classified secondary when the condition is due to another cause. In open-angle glaucoma, the peripheral part of the iris bunches up against trabecular meshwork (spongy tissue located near the cornea) and prevents aqueous from draining from the chamber, causing high intraocular pressure [46]. Angle glaucoma accounts for 80% of all glaucoma cases and becomes more common as age increases [47].

Signs and Symptoms
Elevated intraocular pressure, reduced visual field (fig. 3), and optic nerve cupping indicate that a patient may have glaucoma (table 1). The rate of progression of glaucoma depends on the level of intraocular pressure, susceptibility of the optic nerve to damage, and the type of the disease. The mild and moderate stages of glaucoma are normally asymptomatic. As the disease progresses, visual field defects develop in the area of peripheral vision and spread to create visual field defects (fig. 4). Central vision may be affected late in the process [46]. The patient may report that they are aware that parts of objects appear to be missing (e.g., letters of words). They may also complain of blurred or dim vision or eye pain, particularly after vigorous exercise. Headaches while reading or during low levels of illumination are suggestive of glaucoma, especially if accompanied by misty vision or haloes. Poor color perception and reduced night vision may also suggest glaucoma.

The risk of glaucoma is greater in certain groups of people. Age is the greatest risk factor for the development of glaucoma; prevalence is four to ten times higher in people over 40 years of age [48]. Also, patients with intraocular pressure (>21 mm Hg) are much more likely to develop glaucoma [48]. Other factors that are associated with glaucoma include: race, family history, high blood pressure, diabetes mellitus, high myopia, and ocular hypertension [49].
Treatment Options

Current treatment for open-angle glaucoma usually begins with pharmacological intervention, leading to laser trabeculoplasty or surgery, when necessary. The purpose of each treatment is to lower the intraocular pressure. The most commonly used classes of drugs for open-angle glaucoma are beta-adrenergic antagonists, alpha-adrenergic agonists, parasympathomimetic agents, carbonic anhydrase inhibitors, and prostaglandin-receptor agonists. However, these medications may cause systemic side effects. For angle-closure glaucoma, the treatment sequence is as follows: pharmacological intervention, peripheral iridotomy, and, if necessary, filtration surgery. For both open-angle and angle-closure glaucoma, the sequence of treatment options is designed to maximize the benefit of the treatment, while minimizing the risk of harm to the patient. However, as glaucoma is a chronic, progressive disease with no known cure, many patients may require all three treatment options. Patients must be aware that drug treatment will be lifelong and requires their full compliance, even in the absence of symptoms.

Diabetic Retinopathy

Diabetic retinopathy is the most serious sight-threatening complication of diabetes mellitus and accounts for about 2.4 million cases of blindness globally (World Health Organization 1997 estimate). It can be classified, based on the extent of proliferation of new blood vessels, into two broad categories - nonproliferative diabetic retinopathy and proliferative diabetic retinopathy. In patients with nonproliferative diabetic retinopathy, progression to the more severe proliferative form may occur. Microaneurysms, intraretinal hemorrhages, soft exudates, hard exudates, intraretinal microvascular abnormalities, venous looping, or venous beading characterize the nonproliferative form. The most common cause of vision loss in nonproliferative diabetic retinopathy is macular edema. Proliferative retinopathy is characterized by proliferating endothelial cell tubules, fibrous proliferation, intra- or peri-retinal hemorrhage, or vitreous hemorrhage. There is significant risk of vision loss in patients with proliferative retinopathy. One study found that the prevalence of blindness (visual acuity of 20/200 or worse) and visual impairment (visual acuity of 20/40 or worse) due to diabetic retinopathy was higher in older patients than younger patients. The 10-year incidence of blindness for people whose diabetes was classed as younger-onset (<30 years of age when diagnosed), older-onset (≥30 years of age when diagnosed) insulin-dependent, and older-onset non-insulin-dependent was 1.8%, 4.0%, and 4.8%, respectively; 10-year incidences of visual impairment were 9.4%, 37.2%, and 23.9%, respectively.

Signs and Symptoms

Some patients will notice a change in central (fig. 3) or color vision. Blurred or distorted vision, and reduced central vision may be reported by patients with macular edema (fig. 2) [51]. Patients may notice very small gray patches or spots in the visual field and their vision may be worse in the morning than in the afternoon. However, for most patients with diabetic retinopathy, there are no early symptoms. Many patients do not develop any visual impairment until the disease has advanced well into the proliferative stage. At this stage of the disease, vision that has been lost cannot be restored.

Early detection of diabetic retinopathy is essential as early treatment can reduce the risk of severe vision loss. Patients at high risk of diabetic retinopathy should be encouraged to undergo annual screening for the disease. All patients with diabetes mellitus should have annual fundus examinations (after dilation of the pupil), and yearly examinations are recommended for patients with diabetic retinopathy [50]. Nearly all patients with diabetes mellitus will develop diabetic retinopathy, so it is important for them to learn about the disease process and the risks of developing ocular signs and symptoms that may accompany vision loss.

Treatment Options

Dietary and pharmacological interventions aimed at controlling blood sugar levels should be combined as the mainstay of treatment to prevent retinopathy [52]. For patients with insulin-dependent diabetes, intensive management to normalize blood sugar levels can delay the onset and slow the progression of clinically important retinopathy, including vision-threatening lesions, by more than 70% [53]. Macular edema is a frequent complication in patients who have diabetes and may be associated with loss of vision. Focal laser photoagulation of clinically significant macular edema may reduce the risk of vision loss in these patients [50]. Furthermore, controlling blood pressure and cholesterol levels may reduce the risk of clinically significant edema [51]. Scatter laser photoagulation surgery can reduce the risk of severe vision loss by 50-60% in patients with proliferative retinopathy and can also be considered in pa-
patients with older-onset diabetes with severe nonproliferative retinopathy that appears to be approaching proliferative retinopathy [54]. The laser beam is used to make hundreds of small burns over the retinal surface that destroy the new, growing blood vessels. Cloudy vision can be treated by vitrectomy, a surgical procedure that clears hemorrhaged blood.

Conclusions

AMD, cataract, glaucoma, and diabetic retinopathy are common causes of reduced vision in people over the age of 50 years. Early detection and treatment of these conditions are important because they can improve vision and quality of life and reduce the risk of further vision loss. By being alert to the early signs and symptoms of these eye diseases, geriatricians have an important role to play in their management. Geriatricians are in a unique position to raise awareness of the symptoms of these conditions among elderly patients so that vision loss is an inevitable, and untreatable, aging process.

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