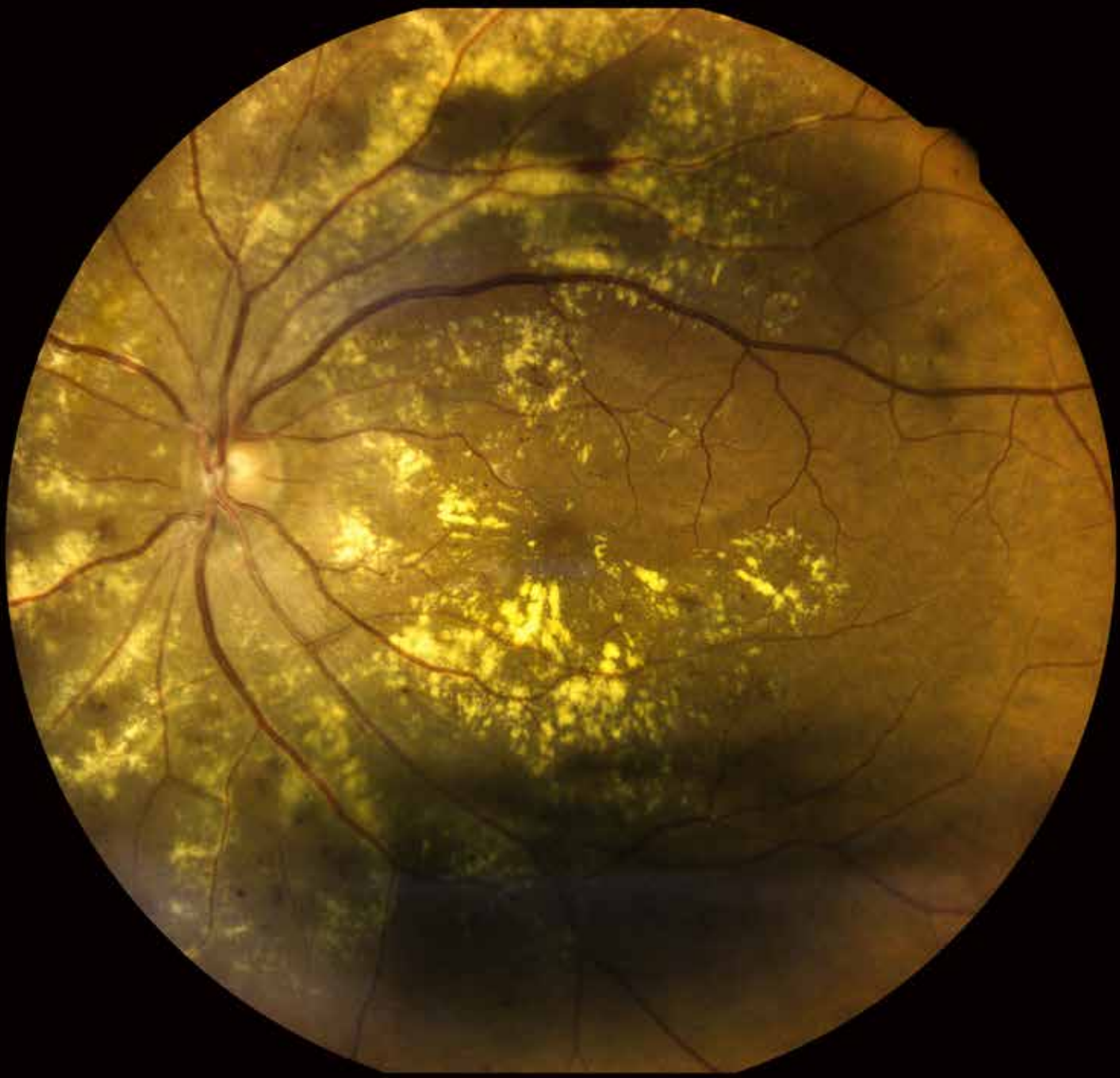

Age-Related Macular Degeneration

Kimberly Batchelor, CST

The eyes of the human body serve to send a photographic memory to our brain. Essentially the connection between the eyes and brain is one of a high efficiency recording of everything we see and experience beginning the day of birth. The cornea and lens bend light to direct the image with the focal point being directly on the retina.[1] This allows the person to focus on the object of their choice and "blurs" out the surrounding images. However, imagine if the primary image was blurred, out of focus, or even unrecognizable while the surroundings were clearly seen. This is the type of image that plagues individuals who suffer from age-related macular degeneration (AMD).

OCULAR ANATOMY

The eye consists of three layers – outer fibrous tunic, middle vascular tunic, and inner nervous tunic (see Figure 1). The cornea, referred to as the window of the eye, is part of the outer tunic. The function of the cornea is to focus the entering light rays. It is composed of connective tissue whose surface is covered by a thin layer of epithelium. Because the cornea contains no blood vessels it is transparent. However, the cornea has a good supply of sensory nerve fibers that enter at its margins and radiate to the center. The fibers include cold and pain receptors, but no heat or touch receptors.

The lens are a part of the middle tunic. Delicate fibers called the zonules or suspensory ligaments project inward from the ciliary processes to hold the lens in place. The lens is positioned directly posterior to the iris and pupil. It consists of columnar epithelial cells,

LEARNING OBJECTIVES

- Read about the studies involving age-related eye disease
- Review the anatomy and epidemiology of AMD
- Explore the stages of age-related macular degeneration
- Learn about the symptoms and detection methods of AMD
- Evaluate the treatment methods for this type of disease

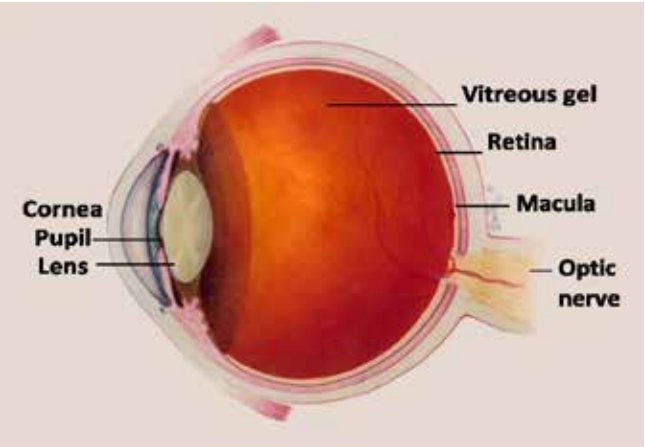

Figure 1: Eye anatomy

but also contains no blood vessels. The lens capsule is a clear, membranous layer that is elastic which contributes to it being under constant tension to assist the lens in assuming a globular shape while the zonules that are also under tension can pull laterally to flatten the capsule and lens.

The retina is the inner tunic layer. The retina is a transparent layer of tissue that is continuous with the optic nerve in the posterior of the eye and continues anteriorly as the inner lining of the eyeball ending posteriorly to the margin of the ciliary body. In the center of the retina is a very small yellowish-colored area called the macula lutea and in its center is the fovea centralis that represents the area of sharpest vision, providing focus and fine detail.

The retina contains the photoreceptors called cones and rods. The photoreceptors are a type of neuron, but each have distinct shapes. The shape of cones are short and blunt, and rods are long, thin projections. The retina contains approximately 3 million cones and 100 million rods.[2] There are key differences between cones and rods. Cones provide the ability to see colors and rods produce black and white vision. Rods are much more sensitive to light, and therefore, provide vision in low light. This explains why dogs can see so much better in the dark as compared to humans; they only have rods to produce black and white images, thus superior vision in low light. However, humans have the advantage of seeing color images.

The fovea centralis does not have rods but has thousands of cones. The retinal layers including its blood vessels are located to each side of the fovea centralis that increases the exposure of the cones to incoming light. Therefore, to view an image in detail, a person will adjust the eyes to allow the details of the image to fall upon the fovea centralis.

EPIDEMIOLOGY

AMD does not cause blindness, but is the leading cause of vision loss in people over the age of 65.[3,6] The disease causes a loss of central vision by deterioration of the macula lutea. Therefore, it interferes with everyday activities, such as seeing faces, reading, writing, or doing repairs around the house.

Estimates of the direct healthcare costs because of AMD in the U.S., Canada, and Cuba combined is approximately $98 billion.[12] The estimate for the global cost is $255 billion in direct healthcare costs.[11] In 2010, 2,069,403 people in the U.S. had AMD.[4,5] It is projected this will increase to 3,664,044 in 2030 and 5,442,265 in 2050.[4,5]

Age is the primary risk factor for AMD.[3] Other major risk factors include:

- Family history: People who have a family history of AMD are at an increased risk for AMD.[3]
- Gender: In 2010, 65% of AMD cases were women compared to 35% in men.[6-8] Two explanations are the longer life expectancy of women compared to men and the use of hormone therapies have a protective effect against the development of AMD.[6-8]
- Genetics: Researchers have identified over 20 genes that can affect the risk for developing AMD. However, there are currently no genetic tests that can contribute to a diagnosis of AMD as well as cannot definitively predict who will develop it. The American Academy of Ophthalmology currently does not support genetic testing for AMD.
- Race: AMD occurs more often in Caucasians than African Americans, Hispanics, or Latinos. AMD affects more than 14% of Caucasian Americans 80 years of age and older.[6] In 2010, 89% of Americans with AMD were Caucasian and by comparison, the African American and Hispanic American populations accounted for 4% of AMD cases.[6]
- Smoking: Research indicates that smoking doubles the risk for developing AMD.

STAGES OF AMD

There are three stages of AMD – early, intermediate, and late. The stage is determined according to the size and number of drusen present.[3] Patients can have AMD in one eye only or both. Additionally, one eye could have a later stage as compared to the contralateral eye.[3]

Drusen are yellow-colored deposits that accumulate between the retinal pigment epithelium and Bruch's membrane, the innermost layer of the choroid, that consist of lipids and proteins.[9] Drusen occur normally with age and are not known to cause AMD, but they are considered the primary sign of AMD.[7,9] Usually they are incidentally discovered during a normal eye exam because they don't produce symptoms.[9] A significant number of large drusen is usually an early sign of geographic AMD.

- Early AMD is indicated by the presence of medium-sized drusen that have an approximate diameter comparable to a human hair.[2] Patients with early AMD usually do not have vision loss. Not all patients diagnosed with early AMD will deteriorate and develop late AMD. For patients who have early AMD in one eye, but no signs of AMD in the contralateral eye, approximately 5% develop advanced AMD after 10 years.[3] For patients who have early AMD in both eyes, approximately 14% develop late AMD in at least one eye after 10 years.[3]
- Intermediate AMD is diagnosed by the presence of large drusen with or without pigment changes in the retina.[3] These changes, just like those associated with the early stage, are only detected during an eye examination. Intermediate AMD can cause some vision loss, but most patients do not exhibit symptoms.
- Late AMD is characterized by drusen and vision loss because of damage to the macula. There are two types of late AMD – geographic and neovascular. Patients can develop both conditions in the same eye and either can appear first.[3]
  - Geographic AMD, also called dry AMD, is characterized by the loss of retinal pigment epithelium (RPE) and choroid in the macula lutea causing the gradual loss of cones and vision.[3,7] Geographic AMD occurs more slowly as compared to neovascular AMD. It is the most common type accounting for 90% of diagnosed cases.[4]
  - Neovascular AMD, also called wet AMD, is caused by the abnormal growth of blood vessels from the choroid into the normally avascular RPE.[7] The vessels often leak blood that causes the macula lutea to swell and become damaged. The damage can be rapid and severe. Wet AMD accounts for approximately 10% of cases but results in 90% of patients being legally blind.[5]

DETECTION OF AMD AND DRUSIN

Symptoms are easy to misinterpret or overlook if only one eye is affected. Often patients who have a disease that is slowly affecting one eye do not seek care until their activities of daily living are affected. The healthy eye will adjust and make up for what the affected eye is lacking causing the diagnosis to occur once the disease has progressed enough to result in irreversible vision loss. An earlier indication of macular degeneration would be the need for brighter lighting and having difficulty adjusting to lower light settings. However, those symptoms are commonly overlooked as having vision trouble. Most symptoms that lead to a diagnosis are blurriness, distortion of straight lines, seeing blurry spots, and worst of all unable to recognize faces. The only exam that will detect AMD is a dilated eye exam that may include the following.

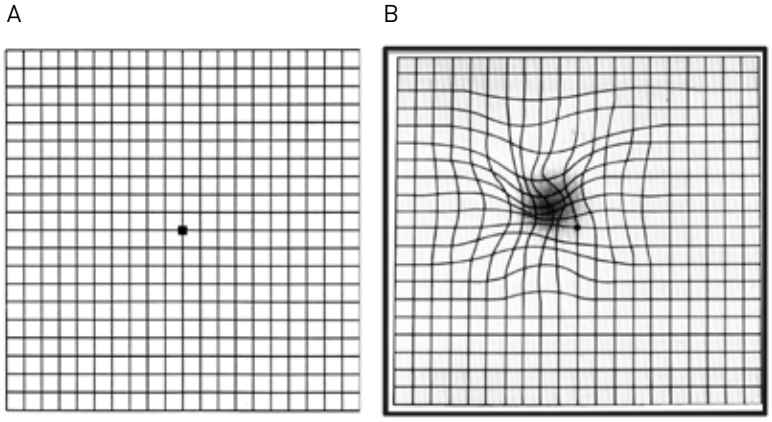

Figure 2: (A) Normal view of Amsler grid, (B) View of grid by patient with AMD.

- Amsler grid: The changes in the patient's central vision will cause the grid lines to appear wavy or disappear (see Figure 2).[3]
- Dilated eye exam: Allows the physician to view the retina and optic nerve for signs of AMD.
- Fluorescein angiogram: Fluorescein dye is injected into a vein usually on the medial side of the elbow and the images are taken. This allows the physician to identify if there are vessels leaking blood that is often a sign of late AMD.

- Optical coherence tomography (OCT): OCT is analogous to ultrasound imaging except the imaging test uses light to take high resolution cross-section images of the retina allowing the physician to see each of the retina's distinctive layers.[10] Because the imaging test relies on light waves it cannot be used in the presence of conditions that interfere with light passing through the tissues of the eye such as cataracts.[10] In the case of AMD, the physician uses OCT to detect the presence of drusen.[10]

AGE-RELATED EYE DISEASE STUDY (AREDS)

There is no cure for AMD; all treatments are aimed at stopping further vision loss, but even then, the disease may continue to progress with treatment. The most promising advances have been made by the researchers at the National Eye Institute (NEI), a division of the National Institutes of Health, who continue to conduct the major AREDS clinical trials. The continuation of the research also produced the AREDS2 studies that were designed to learn more about the natural history and risk factors of AMD and to evaluate the effect of nutritional supplements on the progression of the disease (AREDS2 also included cataract disease as part of the research). The AREDS studies have shown that specific nutritional supplements can slow the progression of intermediate and late AMD.

The first study, AREDS, included 4,757 participants, ages 55-88 years, with AMD, cataract(s), or both.[11] Of that number, the results of the study are based on 3,640 participants at any one of the three stages of AMD.[11] The study discovered that people with intermediate AMD or those with late AMD in one eye reduced their risk of developing advanced AMD by approximately 25% when treated with a combination of antioxidants (vitamin C, vitamin E, beta-carotene) and zinc + copper, referred to as the AREDS formula (see Box 1).[11] The formula also decreased the risk of central vision loss by 19%.[11]

The researchers continued to monitor participants and 10 years later about 70% of the participants were taking AREDS formula.[11] It was found that those participants were 25%-30% less likely to develop late AMD.[11]

In 2006, the NEI began a second study, AREDS2, to research if adding omega-3 fatty acids or lutein plus zeaxanthin would improve the effectiveness of the AREDS formula. Omega-3 fatty acids are produced by plants, including algae, and are present in fish such as salmon. Lutein and zeaxanthin are carotenoids, a class of yellow, orange, and red fat-soluble pigments, including beta-carotene, that give color to plants such as ripe tomatoes. Additionally, the researchers tested the effect of eliminating beta-carotene that has been indicated in other studies to increase the risk of developing lung cancer in smokers.

A total of 4,203 people, ages 50-85, participated in the AREDS2 research and it only included people with intermediate AMD in both eyes or intermediate AMD in one eye and late AMD in the contralateral eye.[11] The results revealed that adding omega-3 fatty acids or lutein plus zeaxanthin to the AREDS formula provided no increase in benefits in regard to late AMD.[11] However, participants who took antioxidants with lutein plus zeaxanthin minus the beta-carotene (AREDS2 formula) did have an increase in benefit as compared to those taking the AREDS formula (see Box 1).[11]

AREDS and AREDS2 Formulas

Nutrient	AREDS Formula*	AREDS2 Formula
Vitamin C	500 mg	500 mg
Vitamin E	400 IU**	400 IU
Beta-carotene	15 mg	---------
Copper***	2 mg	2 mg
Lutein	----------	10 mg
Zeaxanthin	----------	2 mg
Zinc	80 mg	80 mg

*Not recommended for current or former smokers.
**International units
***Added to avoid zinc-related copper deficiency.
Courtesy of National Institutes of Health, National Eye Institute, Bethesda, MD, 2020.

CURRENT TREATMENT RESEARCH FOR GEOGRAPHIC AMD

Geographic AMD currently has no approved treatment. However, in December 2019, the NEI launched a clinical trial to test the safety of a patient-specific stem cell-based therapy. It is the first clinical trial in the U.S. to use replacement tissues from patient-derived induced pluripotent stem cells (iPSC). Researchers found that the therapy prevented blindness in animal trials.[12]

The therapy involves taking a patient's blood cells and converting them into iPSC that have the potential to form almost any type of cell in the body, hence the term "pluripo-

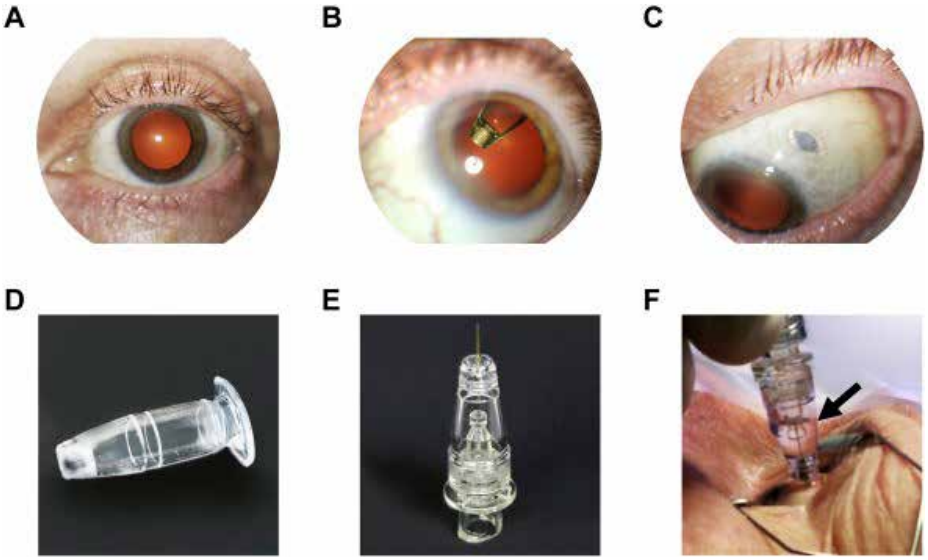

Figure 3: Port delivery system with ranibizumab: (A) Eye with implant in primary position (implant not visible), (B) Eye looking up with implant visible through dilated pupil, (C) Eye looking down to be able to visualize PDS septum, (D) PDS implant, (E) Refill needle, (F) Refill procedure being completed with previous implant contents in the fluid collection reservoir of the refill needle (arrow).

tency."[12] The iPSC are "programmed" to convert to RPE cells which are the cells that die early in the geographic atrophy of AMD.[12] RPE cells support photoreceptor cells in the retina; therefore, when the RPE cells die, the photoreceptor cells also eventually die resulting in blindness. The therapy is aimed at preserving the remaining healthy photoreceptors by replacing dying RPE with iPSC derived RPE.[12]

The iPSC derived RPE are grown in sheets that are one cell thick to replicate their natural anatomy in the eye.[12] The surgeon positions the sheet between the RPE and photoreceptors using a surgical instrument designed specifically for the task.[12] The first clinical trial will involve 12 patients with late AMD who will receive the iPSC derived RPE in one eye and monitored for one year to confirm if the therapy is safe for humans.[12] If safety is confirmed, the next clinical trials will involve more patients to assess the ability of the therapy to prevent blindness and restore patient's vision.

TREATMENTS FOR ADVANCED NEOVASCULAR AMD

Neovascular AMD occurs when abnormally high levels of the vascular endothelial growth factor (VEGF) protein is secreted within the eye that promotes the growth of new abnormal blood vessels. These vessels can bleed causing a hemorrhage underneath the retina or leak fluid lifting the macula from its flat position. The most common treatment to slow the vision loss is anti-VEGF injections that block the abnormal growth of blood vessels.[13] The injections only last for a short period of time so the patient is required to receive monthly injections.[3,13]

Photodynamic therapy (PDT) is a less common treatment that is used in conjunction with anti-VEGF injections.[13] PDT is performed with the use of the photosensitizing drug verteporfin (brand name Visudyne®). When activated by light, verteporfin closes off the leaking new blood vessels and slows their growth.[3] The drug is manufactured as a dark green powder cake.[14] Each vial is reconstituted with 7 mL of sterile water and to achieve the desired dose the required amount of verteporfin is withdrawn from the vial and diluted with 5% dextrose for a total volume of 30 mL for infusion.[14] The full volume is administered intravenously over 10 minutes at a rate of 3 mL per minute.[14] Fifteen minutes after the start of the 10 minute infusion, the physician begins using a nonthermal diode laser to deliver a 689 nm wavelength laser light to photoactivate the verteporfin.[14] The light is delivered to the retina as a circular spot via a fiber optic and a slit lamp using ophthalmic magnification lens.[14] The physician will evaluate the patient three months after treatment; if neovascular leakage is again detected via fluorescein angiography, PDT may be repeated.[14]

A clinical trial that is underway is the Archway trial for a port delivery system, which is now in phase 3 testing. Despite the effectiveness of anti-VEGF agents, studies have shown that the vision outcomes in clinical practice significantly decrease as compared to the outcomes achieved during clinical trials, likely due to a reduction in patient monitoring and a decrease in treatment frequency.[15] The frequent travel burden for clinical practice visits for monitoring and treatment can be difficult to maintain for some patients.

Therefore, the need for a sustainable treatment strategy that meets the requirements for monthly intravitreal anti-VEGF therapy that also reduces the burden on the patient of frequent clinical visits is lacking.

The Archway trial is focused on the efficacy of a Port Delivery System (PDS) of the anti-VEGF drug ranibizumab. Developed by Genentech, a U.S. biotechnology corporation, Ranibizumab is an antibody that inhibits angiogenesis by inhibiting vascular endothelial growth factor A. PDS involves the surgical placement of a permanent refillable ocular port into the affected eye that continuously delivers a set amount of ranibizumab into the vitreous (see Figure 3A, B, D).[15] The implant reservoir can be repeatedly refilled with the drug through a self-sealing septum located in the center of the implant flange (see Figure 3C).[15] Archway is the first successful phase 3 trial of a system that delivers a continuous anti-VEGF agent into the vitreous. Refill-exchanges only need to be completed every six months reducing the frequency of the patient's treatment and travel to the clinical site (see Figure 3E, F).[15] The trial has demonstrated that PDS with ranibizumab has equivalent efficacy to monthly intravitreal injections.[15]

IMPLANTABLE MINIATURE TELESCOPE (IMT) FOR END-STAGE GEOGRAPHIC AND NEOVASCULAR AMD

One of the newest treatment options that offers patients the hope of assisting them to resume normal activities and independence is the IMT that was approved by the U.S. Food and Drug Administration in 2010 (see Figure 4).[16] The IMT has been developed by the VisionCare Ophthalmic Technologies, Inc. as part of Centrasight, a new patient care system focused on treating end-stage AMD. Dr. Isaac Lipshitz developed the IMT that consists of a telescope that is the size of a pea.[17] Simply put it works like the telephoto lens of a camera. Images that enter the eye are magnified two to three times their original size by the telescope.[17,18] The vision is improved because the image is so much larger when it contacts the retina and macula lutea. The goal is to assist the eye in using the parts of the retina that are still healthy to compensate for the damaged cells.[17]

Figure 4: Implantable miniature telescope – 4.4 mm in height and 13.5 mm in diameter.

Patients are carefully screened and must be dedicated to the months of extensive rehabilitation that involves reteaching the brain to learn how to use each eye differently for a task. Patients are required to meet the FDA guidelines to be eligible for the procedure including 75 years of age or older, diagnosed with irreversible geographic or neovascular AMD in both eyes, drug therapy is no longer effective, visual acuity between 20/160 and 20/800, and no previous cataract surgery in the eye that will receive the IMT.[16-18] Other contraindications for surgery include corneal dystrophies and optic nerve disorders.[18]

Potential patients are required to undergo testing with an external telescope that mimics the effects of the IMT to confirm whether vision improvement is possible and if the peripheral vision in the non-operative eye is adequate. This requires several visits to an ophthalmologist that specializes in low vision. Because the IMT replaces the lens, peripheral vision is lost in the operative eye, therefore, the reason for only one eye receiving the telescope.[16,17] The non-operative eye is used to provide peripheral vision to assist the person with activities such as walking. The brain must be "trained" to learn to choose which eye is needed for a specific task such as viewing an image up close or peripherally.[16,17] The postoperative rehabilitation therapy can take up to one year or longer.[16]

Vitreoretinal Systems

Most vitreoretinal systems require single use vitrectomy kits that include a fluid control cartridge, vitrectomy handpiece and tubing, light pipe and cable, scleral trocars and plugs, and infusion canula and tubing. The CST should test the equipment prior to the patient entering the operating room including testing the cutting function of the vitrector. The CST must prime the infusion canula with the solution of the surgeon's preference and confirm that air bubbles are not present prior to the canula being used. The vitrector settings for aspiration, cutting, and infusion must be set on the machine according to the surgeon's preference prior to the patient entering the operating room.

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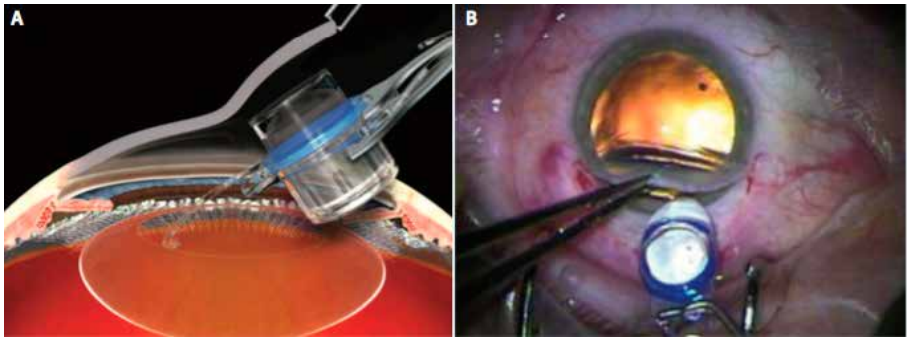

Figure 5: Replacing the lens with the IMT.

The surgical procedure involves removing the lens, such as what is performed during cataract surgery, and replacing the lens with the IMT (see Figures 5 and 6). The initial incision is made superiorly and is approximately 12 mm in length; it is like the incision made for an extracapsular cataract incision, except the incision is longer.[19] A second 7 mm capsulorhexis is made temporally to facilitate performing the phacoemulsification and viscoelastic is injected to protect the endothelium.[19] However, not all surgeons use two incisions. A second option is to make only one 12 mm scleral tunnel incision and for a keratome to be inserted into the tunnel to create the incision for the phacoemulsification and then inject the viscoelastic to fill the anterior chamber and to coat the endothelium for protection.[19] The cornea is lifted and the IMT is inserted. When the IMT is in position it protrudes through the pupil 0.1 to 0.5 mm with a clearance of approximately 2.5 mm from the device to the endothelium of the cornea.[19] The last steps of the procedure involve performing an iridectomy – viscoelastic is removed, and the incision or incisions are closed with several sutures.[19]

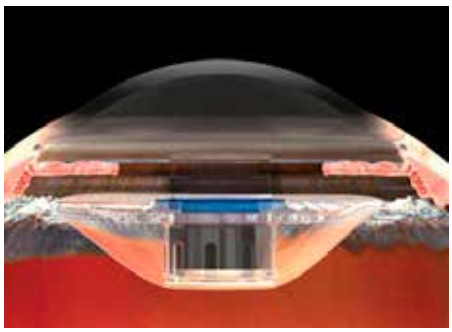

Figure 6: Implanted IMT.

Postoperatively, the patient is prescribed a regimen of eye drops with the addition of atropine dilating drops for one month. It needs to be communicated to the patient because of the dilation and corneal edema, the initial postoperative vision may be blurred and therefore, not to be immediately disappointed with the surgical outcome.[18] The patient will require wearing glasses and visually assistive equipment postoperatively on a permanent basis.[10] Additionally, driving is contraindicated.[18]

Studies have reported statistically significant improvement of vision in the eyes of patients with the telescope as compared to control patients. Approximately 90% of patients demonstrated two or more lines of improvement when reading the visual acuity chart, and 67% of patients were able to see three or more lines on the chart postoperatively as compared to preoperatively.[18]

The primary complication from surgery is damage to the inner endothelial cells of the cornea. During the procedure, a small incision is made, and a small portion of the cornea is lifted upwards to allow removal of the lens and implantation of the IMT (see Figure 5). The corneal cells can be damaged causing postoperative swelling and fluid retention that affects the vision.[17] In some instances, this may lead to a corneal transplantation procedure having to be performed.

SUBRETINAL HEMORRHAGE – COMPLICATION OF NEOVASCULAR AMD

There are instances where patients with neovascular AMD can experience a subretinal hemorrhage. The complication is addressed by the surgeon performing a vitrectomy. After placement of an incise drape over the operative eye and the opening in the drape is made by the surgeon using straight Tenotomy scissors, the eyelids are retracted with a self-retaining eyelid retractor. The operating microscope is brought into place and the surgeon performs a sclerotomy. For a 23-, 25-, or 27-gauge procedure, the sclerotomy is placed 3 to 4 mm from the limbus.[20] The trocar or blade is inserted until it is seen through the pupil and withdrawn and the canula remains in the sclerotomy.[20] An infusion line is placed onto the canula to deliver balanced salt solution through a vitreoretinal system (see Box 2). The system is setup and managed by the CST during the procedure and run by the surgeon through footswitch controls to power the surgical instrumentation including intraocular cautery,

There are three stages of AMD – early, intermediate, and late. The stage is determined according to the size and number of drusen present.[3] Patients can have AMD in one eye only or both. Additionally, one eye could have a later stage as compared to the contralateral eye.[3]

laser probes, light pipes, soft-tip drainage needles, viscous fluid packs, and vitrectomy probes.

After the infusion line is connected, a vitrector probe is used to cut and remove the vitreous gel. Tissue plasminogen activator (tPA) is also injected posterior to the retina to create a small bubble or bleb of fluid that assists in disintegrating the hemorrhage.[20] The peripheral retina is assessed by applying scleral depression to identify retinal tears or defects that may require laser treatment. Next, a fluid-gas exchange is completed to displace the subretinal hemorrhage from the central retina. By injecting an air bubble into the posterior segment, the intraocular fluid is removed, leaving the air bubble to temporarily maintain the eye pressure.[20]

Two types of gases can be used to maintain eye pressure and displace the hemorrhage – perfluoropropane (C3F8) and sulfurhexafluroide (SF6). Using a 60cc syringe, the agent of surgeon's choice is drawn up and mixed with sterile air to fill the syringe. The mixture is slowly injected through the infusion line until the correct eye pressure is achieved and maintained. The canula is removed and because the incisions are only 0.5 mm in length the surgical wound is self-sealing. If suture is required, 7-0 or 8-0 polyglactin 910 suture is used to prevent the gas bubble from leaking through the canula incision.[20]

Postoperatively, patients often experience minimal pain, but can experience redness and irritation for two to three weeks. Antibiotic and steroid eye drops are prescribed to reduce the risk of infection and inflammation. Because of the gas, vision may be blurry one to three weeks postoperatively. Because of the gas bubble, the patient is instructed to lay face down or to one side for one to five days while taking 10-15-minute breaks to exercise the neck muscles, eat, and address other essential activities.[20] Postoperatively, the eye will regenerate the aqueous fluid while the air or gas bubble is being absorbed. The air bubble can be present for about

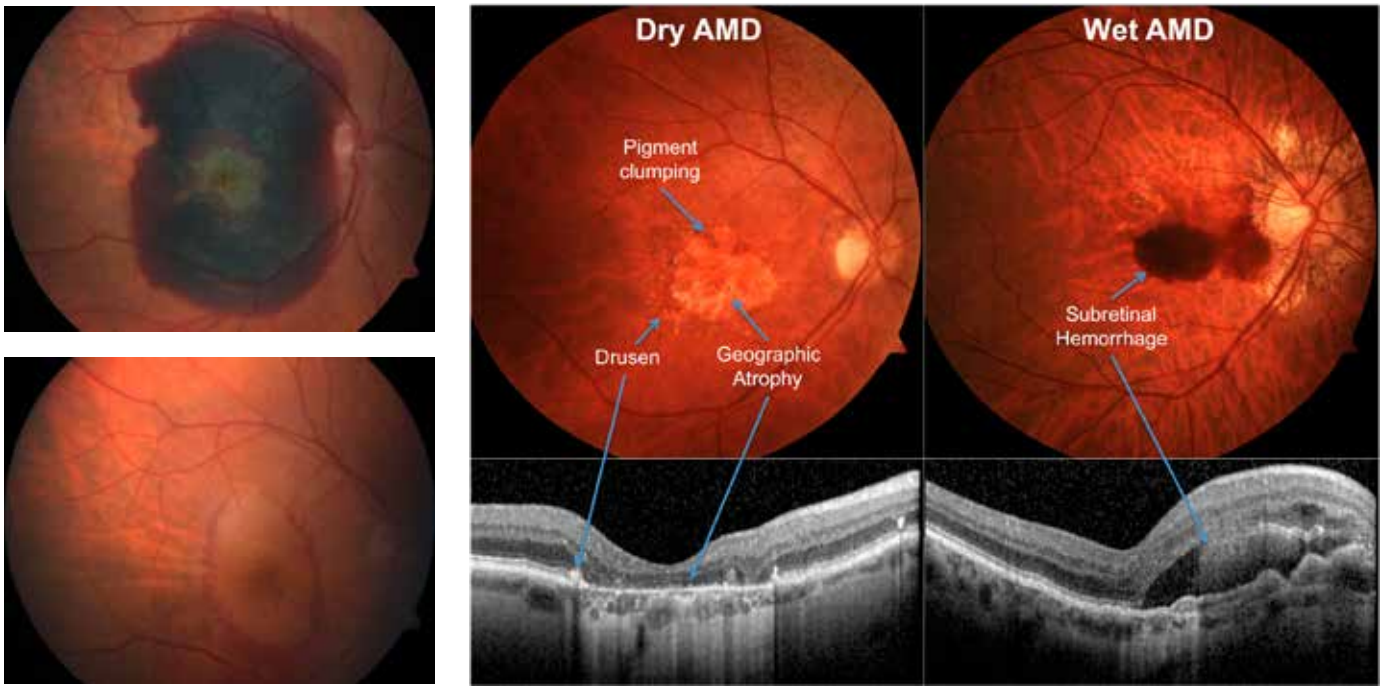

Image 1 is a patient with a large submacular hemorrhage. Image 2 is two months after surgical intervention; minimal hemorrhage remains in the macula due to displacement.

Images 1, 2 and 3 are provided by Dr. Sandeep Shah Retina Vitreous Center Patient Database

a week, SF6 bubble present for about 30 days, and C3F8 present for up to 60 days.[20] Patients can resume normal activities in approximately two to four weeks.[20]

Complications of vitrectomy depend on age, presence of other medical conditions, and issues related to AMD. Complications include challenges with normal eye movement, hemorrhaging, infection, increased risk for cataract formation, lens damage, ocular hypertension, and retinal detachment.[20] Additionally, a small percentage of patients may not experience an improvement in vision. The patient is seen by the surgeon the same day of the procedure or next day to examine the eye and address complications, if present.

The eyes are an astonishing organ. So much can be discovered by looking inside the eye. Surgeons can see systemic illness through the eye such as diabetes, aneurysms, high blood pressure, high cholesterol, lupus, certain types of cancers, and other conditions. But like most of the human body, the eyes' function can be taken for granted.

ORGANIZATIONS THAT SUPPORT RESEARCH OF AMD AND PROVIDE INFORMATION

American Academy of Ophthalmology
P.O. Box 7424
San Francisco, CA 94120-7424
(415) 561 – 8500
www.aao.org

American Foundation for the Blind
1401 South Clark Street
Suite 700
Arlington, VA 22202
(215) 502 – 7600
www.afb.org

American Optometric Association
243 North Lindberg Boulevard
St. Louis, MO 63141-7881
(314) 991 – 4100
www.aoa.org

Macular Degeneration Association
5969 Cattleridge Boulevard
Suite 100
Sarasota, FL 34232
(855) 962 – 2852
www.macularhope.org

Macular Degeneration Partnership
6222 Wilshire Boulevard
Suite 260
Los Angeles, CA 90048
(310) 623 – 4466
www.amd.org

National Eye Institute
Information Office
31 Center Drive MSC 2510
Bethesda, MD 20892-2510
(301) 496 – 5248
www.nei.nih.gov

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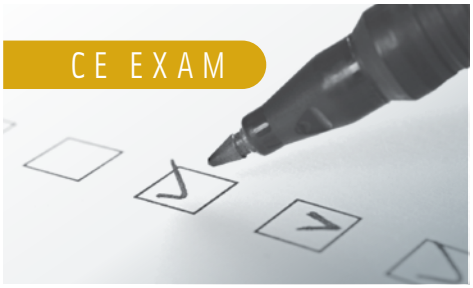

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Age-Related Macular Degeneration

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1. Which structure of the eye is continuous with the optic nerve?
A. Iris
B. Lens
C. Retina
D. Cornea

2. Which eye structure contains only cones?
A. Suspensory ligaments
B. Fovea centralis
C. Cornea
D. Lens

3. Which of the following doubles the risk for developing AMD?
A. Gender
B. Race
C. Genetics
D. Smoking

4. Which of the following is usually an early sign of geographic AMD?
A. Drusen
B. Pigment changes
C. Vision loss
D. Bruch's

5. Which type of AMD is most common?
A. Early
B. Late
C. Neovascular
D. Geographic

6. Which dye is used during an angiogram?
A. Iodine
B. Fluorescein
C. Barium sulfate
D. Gadolinium

7. Which exam cannot be used if the patient has cataracts?
A. Optical coherence tomography
B. Fluorescein angiogram
C. Dilated eye exam
D. Amsler grid

8. Which procedure is performed in conjunction with an implantable miniature telescope implantation?
A. Keratoplasty
B. Cataract extraction
C. Scleral buckle
D. Iridectomy

9. What is lost in the eye that receives the implantable miniature telescope?
A. Night vision
B. Central vision
C. Peripheral vison
D. Color vision

10. Which type of cell can potentially convert to most type of cells in the body?
A. RPE
B. Endothelial
C. Cartilage
D. iPSC

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