

Uveitis

The uvea is the middle layer in the eye sandwiched between the retina (innermost layer) and the sclera (outermost layer). The uvea contains many blood vessels that carry blood to and from the eye. Uveitis is inflammation of the uvea. Since the uvea nourishes many important parts of the eye, uveitis can damage your sight.

Symptoms can include pain, “floaters,” blurriness, light sensitivity, and redness. Uveitis may develop suddenly with redness and pain or with just a blurring of vision.

Causes of this condition include viruses like mumps, shingles, or herpes simplex; eye injuries; fungi or parasites; autoimmune diseases; and others. In most cases, the cause is unknown.

Uveitis is diagnosed by an examination of the eye. In addition, your ophthalmologist (Eye M.D.) may order blood tests, skin tests, or x-rays and also will want information about your overall health.

There are different types of uveitis:

Iritis

With iritis, the uvea is inflamed near the front of the eye in the iris. Iritis has a sudden onset and may last up to eight weeks.

Cyclitis

Cyclitis affects the muscle that focuses the lens in the middle part of the eye. It develops suddenly and lasts for several months.

Choroiditis

This is an inflammation in the back of the eye. It can develop more slowly than the other forms of uveitis and last longer, although this is variable.

Because uveitis is a serious condition that can cause permanent damage to the eye, it needs to be treated as soon as possible. Eyedrops and pupil dilators reduce inflammation and pain. For more severe inflammation, oral medications or injections may be necessary. If uveitis is associated with other conditions like glaucoma or retinal damage, surgery may be required.

If you have a “red eye” that does not clear up quickly, ocular pain, or other significant symptoms, see your ophthalmologist as soon as possible.

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Stargardt's Disease

Stargardt's disease is an inherited disease that affects the retina, the layer of light-sensitive cells lining the back of the eye. It usually becomes apparent between the ages of eight and 14. Boys and girls are equally susceptible and more than one child in a family can have it.

Stargardt's disease begins with slightly blurry vision that gradually gets worse. By the late 20s, corrected vision is typically about 20/200, the level labeled legally blind. Remaining vision is good enough for most people to live fairly normal lives, though they will not drive, or read without using magnification devices.

A buildup of **lipofuscin** (fatty substance) in retinal cells is thought to cause Stargardt's disease. The buildup typically happens in the central retina, or **macula**, where it resembles beaten bronze. Or it can occur in the side retina where it causes small white flecks. This form is called **fundus flavimaculatus**.

Angiography, a special photograph of the retina, may aid in the diagnosis. Although no specific medical or surgical treatment is available, eyeglasses and magnification help affected people adapt to the disease.

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Retinoschisis

Retinoschisis is a genetic eye disease that splits the retina, the light-sensitive layer of cells lining the back of the eye. It occurs in two forms, one affecting young children, the other affecting older adults. Both forms usually affect both eyes, though one eye may be worse than the other.

Because the disease is inherited on the X chromosome, childhood retinoschisis occurs in boys more than girls. It is usually detected because of poor vision.

Retinoschisis has different affects on the eye and vision depending on the location of the split. If the split retina involves the peripheral (side) retina, peripheral vision is lost. Retinal detachment is another risk associated with retinoschisis. More commonly, retinoschisis affects the **macula**, the area of the retina responsible for central vision. If the split retina is in this location, one loses central vision.

Peripheral retinoschisis is more common in adults and is usually caused by aging. In this case, it usually does not affect vision, but it can cause a retinal detachment. If detected early, a retinal detachment can be treated with surgery or laser therapy.

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Retinal Side Effects From Systemic Medication

The retina is a layer of light-sensing cells that line the back of the eye. As light rays enter your eye, the retina converts the rays into signals that are sent through the optic nerve to your brain, where they are recognized as images.

Certain systemic medications, which affect the entire body rather than one specific location, can sometimes affect the retina and lead to vision loss. If you are taking any of the medications below to treat other conditions, be sure to tell your ophthalmologist (Eye M.D.) so that your eyes can be examined frequently to check for potential damage and vision loss. Other drugs not listed can also have ocular side effects.

- hydroxychloroquine, an antimalarial drug commonly used in the treatment of systemic lupus erythematosus and rheumatoid arthritis;
- niacin, also known as nicotinic acid or vitamin B₃, used as both a vitamin supplement and a lipid-lowering agent;
- chlorpromazine (Thorazine) and thioridazine, used as antipsychotics;
- amitriptyline and imipramine, used to treat depression, sleep disorders, and neuropathic pain;
- corticosteroids, used to treat inflammatory disorders and for adrenal insufficiency;
- tamoxifen, used in treating breast cancer;
- canthaxanthine, used as an artificial tanning agent, as well as for the treatment of vitiligo and other skin conditions; and
- erectile dysfunction drugs.

Caught early, it is possible to prevent damage and perhaps even to reverse it, depending on the drug and on the particular case. It is not common for eyes to be damaged by these medications, so it is important to continue to take all medications that have been prescribed for you unless your doctor tells you to discontinue them.

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Proliferative Diabetic Retinopathy

Proliferative diabetic retinopathy (PDR) is a complication of diabetes caused by changes in the blood vessels of the eye. If you have diabetes, your body does not use and store sugar properly. High blood sugar levels create changes in the veins, arteries, and capillaries that carry blood throughout the body. This includes the tiny blood vessels in the retina, the light-sensitive nerve layer that lines the back of the eye.

In PDR, the retinal blood vessels are so damaged they close off. In response, the retina grows new, fragile blood vessels. Unfortunately, these new blood vessels are abnormal and grow on the surface of the retina, so they do not resupply the retina with blood.

Occasionally, these new blood vessels bleed and cause a **vitreous hemorrhage**. Blood in the vitreous, the clear gel-like substance that fills the inside of the eye, blocks light rays from reaching the retina. A small amount of blood will cause dark floaters, while a large hemorrhage might block all vision, leaving only light and dark perception.

The new blood vessels can also cause scar tissue to grow. The scar tissue shrinks, wrinkling and pulling on the retina and distorting vision. If the pulling is severe, the macula may detach from its normal position and cause vision loss.

Laser surgery may be used to shrink the abnormal blood vessels and reduce the risk of bleeding. The body will usually absorb blood from a vitreous hemorrhage, but that can take days, months, or even years. If the vitreous hemorrhage does not clear within a reasonable time, or if a retinal detachment is detected, an operation called a vitrectomy can be performed. During a vitrectomy, the eye surgeon removes the hemorrhage and any scar tissue that has developed, and performs laser treatment to prevent new abnormal vessel growth.

People with PDR sometimes have no symptoms until it is too late to treat them. The retina may be badly injured before there is any change in vision. There is considerable evidence to suggest that rigorous control of blood sugar decreases the chance of developing serious proliferative diabetic retinopathy.

Because PDR often has no symptoms, if you have any form of diabetes you should have your eyes examined regularly by an ophthalmologist (Eye M.D.).

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Retinopathy of Prematurity

Retinopathy of Prematurity (ROP) damages premature babies' retinas, the layer of light-sensitive cells lining the back of the eye. ROP usually occurs in both eyes, though one may be more severely affected.

The last 12 weeks of a full-term pregnancy are an especially active time for the growth of the eye. When a baby is born prematurely, blood vessels are not ready to supply blood to the retina. At birth, abnormal new blood vessels form and cause scarring or detachment of the retina. The condition is especially common in very small babies. It is more likely to occur in babies weighing one or two pounds than in babies weighing three pounds or more.

Despite improved medical care, the disease is becoming more common because smaller and sicker infants are surviving. Supplemental oxygen given to premature babies may be part of the cause of ROP, but it is not the only factor as was once thought.

In severe cases, the retina may be extremely scarred and detached. Many cases get better without treatment and only a small number of children go blind. Cryotherapy (freezing) or laser treatments can prevent progression of the disease.

Children with ROP are more likely to develop nearsightedness and **amblyopia** (lazy eye). Eyeglasses, patching, and eye muscle surgery can help these associated problems. Follow-up examinations of severely affected children should continue periodically.

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Retinoblastoma

Retinoblastoma, a malignant tumor that grows in the retina, the layer of light-sensing cells in the back of the eye, can destroy a child's vision and be fatal. Retinoblastoma can occur in one or both eyes, and usually develops in the first year or two of life. It affects children of all races, and occurs in boys and girls equally.

The most common sign is a change in the color of the pupil, which can appear white in reflected light. This phenomenon is referred to as a **cat's eye reflex**. Sometimes the affected eye will cross or turn outward. Retinoblastoma can be hereditary and is more likely to develop in children with a family history of the disease.

With early diagnosis, retinoblastoma treatment is remarkably effective. More than 90% of children survive and many eyes are saved with a combination of medications, radiation therapy, and heat, freezing, or laser treatments. In severe cases, the affected eye is removed.

If a child has had retinoblastoma, there is an increased chance for a second cancer to develop. Children with retinoblastoma should have regular examinations by an ophthalmologist (Eye M.D.) and a pediatric oncologist.

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Retinitis Pigmentosa

Retinitis pigmentosa (RP) describes a group of related diseases that tend to run in families and cause a slow but progressive loss of vision. RP affects the rods and cones of the retina, the light-sensitive nerve layer at the back of the eye, and results in a decline in vision in both eyes. RP usually affects both eyes equally, with severity ranging from no visual problems in some families to blindness at an early age in others. RP gets its name from the fact that one of the symptoms is a clumping of the retinal pigment that can be seen during an eye exam.

The earliest symptom of retinitis pigmentosa, usually noticed in childhood, is night blindness or difficulty with night vision. People with normal vision adjust to the dark quickly, but people with night blindness adjust very slowly or not at all. A loss of side vision, known as “tunnel vision,” is also common as RP progresses. Unfortunately, the combination of night blindness and the loss of peripheral vision can be severe and can lead to legal blindness in many people.

While there is a pattern of inheritance for RP, 40% of RP patients have no known previous family history. Learning more about RP in your family can help you and your ophthalmologist predict how RP will affect you.

Usher’s syndrome, a condition that causes both deafness and blindness, is a form of RP. The incidence of Usher’s syndrome is difficult to determine, but surveys of patients suggest up to 10% of RP patients are deaf. The incidence of Usher’s syndrome is three cases per 100,000. It is the most frequent cause of combined deafness and blindness in adults.

Considerable research is being done to find the hereditary cause of RP. As hereditary defects are discovered, it may be possible to develop treatments to prevent progression of the disease. While developments are on the horizon, particularly in the area of genetic research, there is currently no cure for retinitis pigmentosa.

Nutritional supplements may be of benefit in RP. It has been reported that vitamin A can slow the progression of RP. Large doses of vitamin A are harmful to the body, and supplements of vitamin E alone may make RP worse. Vitamin E is not harmful if taken along with vitamin A or in the presence of a normal diet. Your ophthalmologist (Eye M.D.) can advise you about the risks and benefits of vitamin A and about how much you can safely take.

Despite visual impairment, people with RP can maintain active and rewarding lives through the wide variety of rehabilitative services that are available today. Until there is a cure, periodic examinations by your ophthalmologist will keep you informed of legitimate scientific discoveries as they develop.

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Macular Hole

The macula is the part of the retina responsible for acute central vision, the vision you use for reading, watching television, and recognizing faces. A macular hole is a small, round opening in the macula. The hole causes a blind spot or blurred area directly in the center of your vision.

Most macular holes occur in the elderly. When the **vitreous** (the gel-like substance inside the eye) ages and shrinks, it can pull on the thin tissue of the macula, causing a tear that can eventually form a small hole. Sometimes injury or long-term swelling can cause a macular hole. No specific medical problem is known to cause macular holes.

Vitreotomy surgery, the only treatment for a macular hole, removes the vitreous gel and scar tissue pulling on the macula and keeping the hole open. The eye is then filled with a special gas bubble to push against the macula and close the hole. The gas bubble will gradually dissolve, but the patient must maintain a face-down position for one to two weeks to keep the gas bubble in contact with the macula. Success of the surgery often depends on how well the position is maintained.

With treatment, most macular holes shrink, and some or most of the lost central vision can slowly return. The amount of visual improvement typically depends on the length of time the hole was present.

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Photodynamic Therapy for Age-Related Macular Degeneration

Age-related macular degeneration (AMD) is a deterioration or breakdown of the macula. The macula is a small area at the center of the retina in the back of the eye that allows us to see fine details clearly and perform activities such as reading and driving. In **exudative or “wet” AMD**, abnormal blood vessels can grow in a layer beneath the retina, leaking fluid and blood and creating distortion or a large blind spot in the center of your vision.

Photodynamic therapy (PDT)—an outpatient procedure involving the use of a special light-activated drug—is used to treat some patients with wet AMD. PDT causes fewer visual side effects than other treatments. The benefit of PDT is that it inhibits abnormal blood vessel leakage associated with wet macular degeneration, limiting damage to the overlying retina.

With PDT, the inactive form of the drug is usually injected into a vein in the arm, where it travels to and accumulates in abnormal blood vessels under the center of the macula. A special low-intensity laser light targeted at the retina activates the drug only in the affected area, damaging the abnormal blood vessels under the retina and leaving normal blood vessels intact.

Patients who are treated with PDT will become temporarily extra sensitive to bright light (photosensitive). Care should be taken to avoid exposure of the skin or eyes to direct sunlight or bright indoor light for several days.

PDT therapy is not effective for treatment of **atrophic or “dry” AMD**, which is caused by aging and thinning of the tissues of the macula. Although photodynamic therapy can preserve vision for many people, it may not stop vision loss in all patients. The abnormal blood vessels may regrow or begin to leak again. Every three months, patients must undergo a repeat examination that includes a **fluorescein angiogram** dye test. Multiple PDT treatments sometimes are necessary.

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Macular Degeneration and Nutritional Supplements

Age-related macular degeneration (AMD) is a disease caused by damage or breakdown of the **macula**, the small part of the eye's retina that is responsible for our central vision. This condition affects both distance and close vision and can make some activities (like threading a needle or reading) very difficult or impossible. Macular degeneration is the leading cause of severe vision loss in people over 65.

Although the exact causes of AMD are not fully understood, a recent scientific study shows that antioxidant vitamins and zinc may reduce the effects of AMD in some people with the disease.

Among people at high risk for late-stage macular degeneration (those with intermediate AMD in both eyes or advanced AMD in one eye), a dietary supplement of vitamins C, E, and beta-carotene, along with zinc, lowered the risk of the disease progressing to advanced stages by about 25% to 30%. However, the supplements did not appear to benefit people with minimal AMD or those with no evidence of macular degeneration.

Light may affect the eye by stimulating oxygen, leading to the production of highly reactive and damaging compounds called **free radicals**. Antioxidant vitamins (vitamins C and E and beta-carotene) may work against this activated oxygen and help slow the progression of macular degeneration.

Zinc, one of the most common minerals in the body, is very concentrated in the eye, particularly in the retina and macula. Zinc is necessary for the action of over 100 enzymes, including chemical reactions in the retina. Studies show that some older people have low levels of zinc in their blood. Because zinc is important for the health of the macula, supplements of zinc in the diet may slow down the process of macular degeneration.

The levels of antioxidants and zinc shown to be effective in slowing the progression of AMD cannot be obtained through your diet alone. These vitamins and minerals are recommended in specific daily amounts as supplements to a healthy, balanced diet.

It is very important to remember that vitamin supplements are not a cure for AMD, nor will they restore vision you may have already lost from the disease. However, specific amounts of certain supplements do play a key role in helping some people at high risk for advanced AMD to maintain their vision. You should speak with your ophthalmologist (Eye M.D.) to determine if you are at risk for developing advanced AMD and to learn if supplements are recommended for you.

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Ocular Histoplasmosis Syndrome

Ocular histoplasmosis syndrome (OHS) is a major cause of visual impairment in the eastern and central United States, where 90% of adults have been exposed to *Histoplasma capsulatum*. This common fungus is found in molds from soil enriched with bat, chicken, or starling droppings and yeasts from animals.

Although the fungus is not found directly in the eye, people with OHS usually test positive for previous exposure to *Histoplasma capsulatum*.

Histoplasmosis is usually mistaken for a cold. The symptoms are very similar. The body's immune system normally overcomes the infection in a few days. Generally, "histo spots," or small scars in the retina, do not affect vision, but for unknown reasons, some people can have ocular complications years or decades later.

Doctors believe that the histoplasmosis spores travel from the lungs to the eye where they settle in the choroid, the layer of tiny blood vessels that provide blood and nutrients to the retina, the light-sensing layer of cells lining the back of the eye.

Ocular histoplasmosis can affect vision when fragile, abnormal blood vessels grow under the retina. These abnormal blood vessels form a lesion known as a **choroidal neovascularization (CNV)**. If left untreated, the CNV lesion can turn into scar tissue and replace the normal retinal tissue in the macula.

The only proven treatment for OHS is a form of laser surgery called **photocoagulation**. The laser's small, powerful beam of light destroys the abnormal blood vessels as well as a small amount of the retinal tissue. Other treatments, including steroids and intraocular injections, are sometimes used. Treatment is not necessary unless the new vessels are in the macula, the part of the retina responsible for acute central vision.

Although only a very small number of people infected with the histoplasmosis virus develop OHS, if you have been exposed to histoplasmosis, you should be sensitive to any changes in your eyesight, and you should monitor your vision using an **Amsler grid test** at home.

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Lattice Degeneration

Lattice degeneration is a condition that causes thinning and weakening of the peripheral retina, the light-sensitive layer of cells lining the back of the eye, which can lead to a retinal tear.

The **vitreous**, a clear, gel-like substance that fills the inside of the eye, is contained in a sac loosely attached to the retina. As one ages, the vitreous takes on a more fluid consistency, and the sac sometimes separates from the retina. In lattice degeneration, there are places where the sac is strongly attached to the retina and pulls on it. This pulling weakens the retina and creates “lattice” lesions, which look like white, crisscrossing lines on the retina.

If part of the vitreous sac becomes detached from the retina, the friction and pulling at the attachment site can create a tear in the retina. Lattice degeneration can sometimes cause **retinal detachments** when holes or tears in the lattice formation permit vitreous fluid to flow under the retina.

Fortunately, most people with lattice degeneration do not develop a retinal detachment. Preventive treatment of lattice degeneration is indicated in some cases, but usually, the ophthalmologist (Eye M.D.) will only need to monitor the condition. If you have a history of lattice degeneration, you should be aware of the symptoms of retinal tears and detachment.

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Macular Edema

Macular edema is the swelling of the **macula**, the small area of the retina responsible for central vision. The edema is caused by fluid leaking from retinal blood vessels. Central vision, used for reading and other close, detail work, is affected.

Because the macula is surrounded by many tiny blood vessels, anything that affects them, such as a medical condition affecting blood vessels elsewhere in the body or an abnormal condition originating in the eye, can cause macular edema.

Retinal blood vessel obstruction, eye inflammation, and **age-related macular degeneration** have all been associated with macular edema. The macula may also be affected by swelling following cataract extraction, although typically this resolves itself naturally.

Treatment seeks to remedy the underlying cause of the edema. Eyedrops, injections of steroids or other, newer medicines in or around the eye, or laser surgery can be used to treat macular edema. Recovery depends on the severity of the condition causing the edema.

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Nonproliferative Diabetic Retinopathy

If you have diabetes mellitus, your body does not use and store glucose properly. Over time, diabetes can damage blood vessels in the retina, the nerve layer at the back of the eye that senses light and helps to send images to the brain. The damage to retinal vessels is referred to as diabetic retinopathy.

Nonproliferative diabetic retinopathy (NPDR), commonly known as background retinopathy, is an early stage of diabetic retinopathy. In this stage, tiny blood vessels within the retina leak blood or fluid. The leaking fluid causes the retina to swell or to form deposits called **exudates**.

Many people with diabetes have mild NPDR, which usually does not affect their vision. When vision is affected, it is the result of macular edema or macular ischemia, or both.

Macular edema is swelling or thickening of the macula, a small area in the center of the retina that allows us to see fine details clearly. The swelling is caused by fluid leaking from retinal blood vessels. It is the most common cause of visual loss in diabetes. Vision loss may be mild to severe, but even in the worst cases, peripheral (side) vision continues to function. Laser treatment can be used to help control vision loss from macular edema. Newer treatments are being investigated.

Macular ischemia occurs when small blood vessels (capillaries) close. Vision blurs because the macula no longer receives sufficient blood supply to work properly. Unfortunately, there are no effective treatments for macular ischemia.

A medical eye examination is the only way to discover any changes inside your eye. If your ophthalmologist (Eye M.D.) finds diabetic retinopathy, he or she may order color photographs of the retina, a special test called fluorescein angiography, or optical coherence tomography (OCT) to find out if you need treatment.

If you have diabetes, early detection of diabetic retinopathy is the best protection against loss of vision. You can significantly lower your risk of vision loss by maintaining strict control of your blood glucose and visiting your ophthalmologist regularly. People with diabetes should schedule examinations at least once a year. Pregnant women with diabetes should schedule an appointment in their first trimester, because retinopathy can progress quickly during pregnancy. More frequent medical eye examinations may be necessary after a diagnosis of diabetic retinopathy.

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Macular Dystrophy

Macular dystrophy is a hereditary condition in which the **macula** degenerates. The macula is the part of your retina responsible for acute central vision, the vision one uses to read, watch television, and recognize faces.

Symptoms of macular dystrophy can range from minimal vision loss and disturbance of color vision to profound loss of reading and night vision. The most common types of macular dystrophies, which tend to appear early in life, are **Best's disease**, **Stargardt's macular dystrophy**, and **bull's eye maculopathy**.

Considerable research is directed toward finding the hereditary cause of many types of macular dystrophies. With further research, it may be possible to develop medical treatments to prevent or slow the progression of macular dystrophy.

Low-vision devices can help affected individuals continue with many of the activities of daily life.

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Vitreotomy Surgery

Vitreotomy is a type of eye surgery used to treat disorders of the retina (the light-sensing cells at the back of the eye) and vitreous (the clear gel-like substance inside the eye). It may be used to treat a severe eye injury, diabetic retinopathy, retinal detachments, macular pucker (wrinkling of the retina), and macular holes.

During a vitrectomy operation, the surgeon makes tiny incisions in the sclera (the white part of the eye). Using a microscope to look inside the eye and microsurgical instruments, the surgeon removes the vitreous and repairs the retina through these tiny incisions. Repairs include removing scar tissue or a foreign object if present.

During the procedure, the retina may be treated with a laser to reduce future bleeding or to fix a tear in the retina. An air or gas bubble that slowly disappears on its own may be placed in the eye to help the retina remain in its proper position, or a special fluid that is later removed may be injected into the vitreous cavity.

Recovering from vitrectomy surgery may be uncomfortable, but the procedure often improves or stabilizes vision. Once the blood- or debris-clouded vitreous is removed and replaced with a clear medium (often a saltwater solution), light rays can once again focus on the retina. Vision after surgery depends on how damaged the retina was before surgery.

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Toxoplasmosis

Toxoplasmosis is a common parasitic infection. When contracted by a pregnant woman, toxoplasmosis can pose serious risks to the unborn baby. Simple precautions can reduce the chance of infection.

Pregnant women should avoid handling litter boxes and eating raw meat because the parasite may originate in cat feces or undercooked meat. If acquired during the first trimester of pregnancy, the infection can be devastating to an infant.

Toxoplasmosis affects the retina, the light-sensitive cells lining the back of the eye. Both eyes can be involved. If the infection settles in the **macula**, the area of the retina responsible for central vision, good vision can be lost forever.

When toxoplasmosis heals, it leaves a scar. The infection may recur years later, sometimes near the previously infected area. Swelling that fights the infection may cause floating spots, red, painful eyes, and poor vision.

Treating toxoplasmosis with oral medications can be very effective. Pyrimethamine and sulfa drugs are the classic choices for treatment with antibiotics, although some doctors add or substitute clindamycin. Occasionally, treatment with steroids, laser therapy, or cryotherapy (freezing) is prescribed.

Screening tests can identify women of childbearing age who are at risk of passing the infection to an unborn child.

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Myopic Degeneration

Myopic degeneration is a condition characterized by progressive stretching of the eye that damages the retina, the layer of light-sensitive cells that lines the back of the eye. People with severe nearsightedness (high myopia) are at greater risk for myopic degeneration.

Myopic degeneration commonly occurs during young adulthood and can lead to a gradual decrease in central vision. Vision can decrease more abruptly in a small percentage of patients. Although central vision may be lost, side (peripheral) vision usually remains unaffected. Remaining sight can still be very useful, and with the help of low vision optical devices, people with this condition can continue many of their normal activities.

The causes of myopic degeneration are not clearly understood, but they may include biomechanical abnormalities or hereditary factors. The biomechanical theory assumes that the retina, in a myopic eye, is stretched over a larger than normal area because the eye is longer in shape than is normal. Over time, the outer coat of the eye, known as the sclera, also stretches in response to forces like internal eye pressure. This stretching of the sclera is thought to lead to retinal degeneration. In the hereditary theory, the retinal changes are thought to be an unavoidable, inherited process.

Loss of central vision can occur if abnormal vessels grow directly under the center of the retina in an area known as the macula. This is called **choroidal neovascularization**. Early diagnosis and treatment can minimize the amount of vision loss. People with myopic degeneration should have their vision monitored by an ophthalmologist (Eye M.D.) on a regular basis. Using an **Amsler grid** to monitor vision at home is also helpful in detecting early growth of these abnormal vessels.

Patients with myopic degeneration have an increased risk of developing peripheral retinal tears and retinal detachment. If a patient experiences new flashes of light, “floaters,” “curtains” or “veils,” or loss of vision, he or she should see an ophthalmologist immediately.

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