Glaucoma: Preventing the visual damage

Most forms of glaucoma cannot be prevented, but early diagnosis and diligent management can avoid life-altering loss of sight.

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An estimated 90% of glaucoma-induced blindness can be prevented, even though the disease is not itself preventable. Just determining that a patient is in trouble is a challenge because glaucoma is typically asymptomatic at its outset—50% of patients are unaware they have the disease. Moreover, the early signs of glaucoma can be very subtle, further delaying the diagnosis. Prompt diagnosis and diligent management are crucial, however. Here’s what you need to know.

What is glaucoma?
A commonly held but oversimplified definition of glaucoma identifies the disease as elevated intraocular pressure (IOP) capable of damaging the optic nerve cells and their fibers, leading to loss of visual field. This definition was found to be inadequate, however, because up to 30% of bona fide glaucoma patients with characteristic optic-nerve and visual-field defects may never manifest any significantly elevated IOP. Newer definitions stress the damage to the visual system and the eye and consider elevated IOP an important risk factor.

The balance between the inflow and outflow of the aqueous fluid of the eye controls IOP. Any imbalance usually occurs where the peripheral cornea and iris meet in what is called “the drainage angle.” Resistance to outflow in this area accounts for a rise of IOP throughout the globe.

In narrow angle glaucoma, the iris is too close to the cornea.

The damage to vision occurs posteriorly at the level of the optic-nerve fibers and their ganglion cells in the retina. Chronic open angle glaucoma (COAG) is the most common form of the disease. Although certain types of glaucoma may be present at birth, COAG usually presents later in life, typically after age 45. Prevalence increases with age.

One concept of glaucoma describes three stages. In stage 1, the disease is undetectable, either by symptoms or by careful clinical exam. Even the most sophisticated monitoring and detection tools cannot document its presence. In stage 2, the patient remains unaware of any problems, but sophisticated tests of visual field and analyses of optic-nerve or retinal-nerve fiber layers can detect damage. In stage 3, both the patient and the doctor are aware of glaucoma’s effects.

Making a prompt diagnosis
We try to detect glaucoma as early in stage 2 as possible. Studies have revealed considerable anatomic damage to retinal ganglion cells even at the beginning of stage 2.

Recently, we have become aware that our ability to measure IOP has been faulty. The standard tonometers by which we routinely measure IOP work by pressing the cornea and determining the resistance of the eye. But these instruments were calibrated on an average of corneas of varying thicknesses. In patients with relatively thin corneas, the instrument meets less resistance and therefore underestimates the pressure of the interior of the eye, sometimes to a significant degree. Conversely, many patients with relatively thick...
corneas who were labeled “ocular hypertensive” were in reality normotensive. Clinicians are now able to measure corneal thickness and compensate for this factor.

Eye doctors can optimize their diagnostic skill by stressing risk factors (see table below). In some varieties of the disease, a genetic defect has been identified.

To diagnose and monitor the progress of glaucoma, eye doctors rely on standard visual acuity and slit-lamp exams, including tonometry, repeated at various intervals. Biomicroscopy of the optic nerve yields a three-dimensional view allowing earlier detection of characteristic changes. The visual-field test, a standard in glaucoma management, is designed to delineate the “island of vision in a sea of darkness.” Threshold visual-field tests determine the dimmest light the patient can see 50% of the time in 24-30 areas of the field of vision. Despite the test’s subjective nature, cooperative patients produce surprisingly reliable results.

For years, eye doctors have made drawings or taken photographs of the optic nerve. Newer modalities can better delineate the anatomy of the nerve or measure the thickness of nerve-fiber layers (which tends to decrease with advancing disease). New versions of visual-field testing are designed to identify defects earlier. However, attempts at earlier detection often come with decreased reliability.

The diagnostic dilemmas presented have resulted in the concept of the “glaucoma suspect”—a patient identified at greater risk of having or developing glaucoma who does not yet have detectable damage. A glaucoma suspect should be monitored more carefully. At times, the clinician and patient may decide that the risk of progression to visual damage is high enough to warrant treatment.

**Preventing damage**

Currently, the only proven way to prevent progressive damage from glaucoma is to lower the IOP to a level at or below a clinician-determined target, customized for each patient. As you seek that goal, keep in mind that IOP varies naturally from hour to hour during the day and from day to day.

The therapeutic choices include various eyedrops (rarely pills), laser treatments, and surgery. Although eyedrops and laser may be equally effective as initial therapy, the culture in the United States in general is to start with eyedrops (see table, facing page), which are chosen on the basis of efficacy, absence of side effects, ease of use, and, increasingly, cost.

An acute attack of narrow angle glaucoma must be treated immediately.

Elderly patients, however, may find it difficult to manage the compliance required of lifelong treatment regimens. Costs vary by insurance coverage, but patients paying out of pocket for a standard glaucoma cocktail of two to three different drugs may have bills of more than $200 per month.

Laser trabeculoplasty involves laser application in the drainage angle, increasing the rate of fluid outflow. The procedure is painless and easy to perform, usually as an office procedure. The effect may be reduced over time, so patients still need to be monitored carefully.

When the clinical situation is deteriorating despite eyedrops, laser, or both, ophthalmologists may recommend surgery to create an alternative route for fluid drainage. A trabeculectomy or, in refractory cases, a tube shunt may be required.

**Special types of glaucoma**

Various other types of glaucoma exist. Etiologies vary. Some are structurally based; others are genetic in origin.

**Narrow angle glaucoma:** In some patients, especially those who are farsighted and have eyes that are physically shorter from front to back, the iris may be anatomically very close to the cornea. The drainage angle is then said to be narrow. If the iris suddenly blocks the drainage angle, the IOP rises precipitously, and an acute attack of narrow angle glaucoma occurs.

The patient may experience pain and blurred vision, see halos around lights, and notice that the eye is red. The pupil becomes fixed and mid-dilated. The patient may even develop nausea and vomiting. An acute attack is a medical emergency and must be treated immediately.

Narrow angle glaucoma is the only variant that can be prevented. If a critically narrow angle is noted by the ophthalmologist before symptoms develop, laser iridotomy will be

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**Risk factors for glaucoma**

- Elevated intraocular pressure
- Advancing age
- Family history of glaucoma
- Ethnic background (specifically African American, Asian, and Hispanic)
- Diabetes
- Nearsightedness
- Hypertension
recommended. The laser is used to punch a small hole in the peripheral iris. This acts as a safety valve, deflating the pressure behind the iris and allowing the iris to fall away from the cornea. Prophylactic laser iridotomy usually confers lifelong protection. Note that eyedrops for the treatment of other forms of glaucoma generally will not prevent narrow angle disease or an acute attack.

A variant of narrow angle glaucoma, chronic angle closure, results from small, repeated, intermittent, and self-limited episodes of angle closure that, over time, scar and close off the drainage angle. These episodes may be induced by any situation causing dilation of the pupil. This serious type of glaucoma frequently requires surgery to control elevated IOP unless it is caught in time and treated with laser iridotomy.

In narrow angle glaucoma patients who have not been treated with iridotomy, any medication with sympathomimetic effects may cause acute or intermittent angle closure. Sympathomimetics comprise an array of drugs, including common anticholinergics, antihistamines, and even OTC cold remedies. Patients with narrow angles who require such medications should be monitored carefully or undergo prophylactic iridotomy. Medications with sympathomimetic effects do not increase IOP in patients with open angle glaucoma. A few patients may have a combination of both types of glaucoma. In providing care for patients who report using eyedrops for glaucoma, primary-care physicians should be aware that most glaucoma patients being treated with eye-drops do not have the narrow angle variety. Nevertheless, before prescribing a sympathomimetic medication, check with the glaucoma patient’s eye specialist to determine type.

Pigmentary glaucoma: As the obverse to narrow angle disease, some irises have a more posterior attachment, i.e., farther from the cornea but closer to the lens and its supporting filaments, the zonules. The almost constant movement of the pupil throughout the day causes the posterior iris surface to chafe against the zonules. Iris pigment granules are released into the aqueous humor producing a condition called “pigment dispersion syndrome.”

These patients are usually younger, myopic, and male. In as many as 30%-50% of patients with pigment dispersion syndrome, the accumulation of pigment in the drainage angle over time causes elevated IOP, or pigmentary glaucoma. It is treated much like COAG, but a previous finding of pigment dispersion syndrome will alert the physician to monitor these patients more carefully to prevent visual damage.

Exfoliation glaucoma: There exists a fascinating condition known as exfoliation syndrome, involving the accumulation of certain protein precursors within the eye. It is commonly described as the presence of what appears to be “dandruff” collecting as a ring on the lens surface just within the pupil. This abnormal material may eventually accumulate in the iris and especially in the trabecular meshwork, the sieve that lines the drainage angle, where it causes elevated IOP.

Approximately 10% of exfoliation syndrome patients will go on to develop exfoliation glaucoma. Interestingly, this abnormal material can be found throughout the body. Researchers in Iceland have recently discovered the gene believed to be responsible for exfoliation syndrome.

Other types: Glaucoma may also occur as a result of intraocular inflammation or trauma, either acute or delayed. Some
patients with significant ocular injuries may appear to have recovered only to develop glaucoma decades later. Certain individuals are genetically predisposed to a rise in IOP as the result of steroid use. Steroids may be administered as eyedrops, sprays, inhalers, creams, pills, or injection. The glaucoma usually is the result of chronic or long-term use but may occur as early as several weeks after the initiation of steroids. The elevated IOP is usually reversible when steroids are stopped, but chronic glaucoma damage has been known to occur. Being aware of preceding events may permit earlier diagnosis and effectively prevent glaucoma damage.

**Protecting vision**

True prevention of glaucoma is possible in narrow angle disease (laser iridotomy), some cases due to traumatic injury (face protectors for appropriate sports), and steroid-induced glaucoma (cessation of medication). But in general, we cannot prevent glaucoma.

The need for routine eye exams cannot be overemphasized. Even with treatment, we probably cannot stop progression of the disease completely. But by lowering the IOP, we can slow it down. Practically, with diligence and cooperative patients, we can and do slow down the disease enough that our patients can live out their lives without the visual disability, even blindness, that might otherwise occur.