Cataract, Senile

Vicente Victor D Ocampo, MD, Consulting Staff, Department of Ophthalmology, Asian Hospital and Medical Center, Philippines
C Stephen Foster, MD, FACS, FACR, FAAO, Clinical Professor of Ophthalmology, Harvard Medical School; Consulting Staff, Department of Ophthalmology, Massachusetts Eye and Ear Infirmary; Founder and President, Ocular Immunology and Uveitis Foundation, Massachusetts Eye Research and Surgery Institution

Updated: May 20, 2009

Introduction

Background

Senile cataract is a vision-impairing disease characterized by gradual, progressive thickening of the lens. It is one of the leading causes of blindness in the world today. This is unfortunate, considering that the visual morbidity brought about by age-related cataract is reversible. As such, early detection, close monitoring, and timely surgical intervention must be observed in the management of senile cataracts. The succeeding section is a general overview of senile cataract and its management.

Pathophysiology

The pathophysiology behind senile cataracts is complex and yet to be fully understood. In all probability, its pathogenesis is multifactorial involving complex interactions between various physiologic processes. As the lens ages, its weight and thickness increases while its accommodative power decreases. As the new cortical layers are added in a concentric pattern, the central nucleus is compressed and hardened in a process called nuclear sclerosis.

Multiple mechanisms contribute to the progressive loss of transparency of the lens. The lens epithelium is believed to undergo age-related changes, particularly a decrease in lens epithelial cell density and an aberrant differentiation of lens fiber cells. Although the epithelium of cataractous lenses experiences a low rate of apoptotic death, which is unlikely to cause a significant decrease in cell density, the accumulation of small scale epithelial losses may consequently result in an alteration of lens fiber formation and homeostasis, ultimately leading to loss of lens transparency. Furthermore, as the lens ages, a reduction in the rate at which water and, perhaps, water-soluble low-molecular weight metabolites can enter the cells of the lens nucleus via the epithelium and cortex occurs with a subsequent decrease in the rate of transport of water, nutrients, and antioxidants.

Consequently, progressive oxidative damage to the lens with aging takes place, leading to senile cataract development. Various studies showing an increase in products of oxidation (eg, oxidized glutathione) and a decrease in antioxidant vitamins and the enzyme superoxide dismutase underscore the important role of oxidative processes in cataractogenesis.

Another mechanism involved is the conversion of soluble low-molecular weight cytoplasmic lens proteins to soluble high molecular weight aggregates, insoluble phases, and insoluble membrane-protein matrices. The resulting protein changes cause abrupt fluctuations in the refractive index of the lens, scatter light rays, and reduce transparency. Other areas being investigated include the role of nutrition in cataract development, particularly the involvement of glucose and trace minerals and vitamins.

Senile cataract can be classified into 3 main types: nuclear cataract, cortical cataract, and posterior subcapsular cataract. Nuclear cataracts result from excessive nuclear sclerosis and yellowing, with consequent formation of a central lenticular opacity. In some instances, the nucleus can become very opaque and brown, termed a brunescent nuclear cataract. Changes in the ionic composition of the lens cortex and the eventual change in hydration of the lens fibers produce a cortical cataract. Formation of granular and plaquelike opacities in the posterior subcapsular cortex often

heralds the formation of posterior subcapsular cataracts.

Frequency

United States

At least 300,000-400,000 new visually disabling cataracts occur annually in the United States, with complications of modern surgical techniques resulting in at least 7000 irreversibly blind eyes. In the Framingham Eye Study from 1973-1975, senile cataract was seen in 15.5% of the 2477 patients examined. The overall rates of senile cataract in general and of its 3 main types — nuclear, cortical, and posterior subcapsular — rapidly increased with age, so that for the oldest age group, 75 years and older, nuclear, cortical, and posterior subcapsular cataracts were found in 65.5%, 27.7%, and 19.7% of the study population, respectively. Nuclear opacities were the most commonly seen lens change.

International

Senile cataract continues to be the main cause of visual impairment and blindness in the world. At least 5-10 million new visually disabling cataracts occur yearly, with modern surgical techniques resulting in 100,000-200,000 irreversibly blind eyes. Published data estimate that 1.2% of the entire population of Africa is blind, with cataract causing 36% of this blindness. In a survey conducted in 3 districts in the Punjab plains, the overall rates of occurrence of senile cataract was 15.3% among 1269 persons examined who were aged 30 years and older and 4.3% for all ages. This increased markedly to 67% for ages 70 years and older. An analysis of blind registration forms in the west of Scotland showed senile cataract as 1 of the 4 leading causes of blindness.

Mortality/Morbidity

Most morbidity associated with senile cataracts occurs postoperatively and is discussed in further detail later. Failure to treat a developing cataract surgically may lead to devastating consequences, such as lens swelling and intumescence, secondary glaucoma, and, eventually, blindness.

- While the risk of dying as a result of cataract extraction is almost negligible, studies have shown an increased risk of mortality in patients who underwent surgery. In a comparison of 167 patients aged 50 years or older who underwent cataract extraction at the New England Medical Center in a period of 1 year to 824 patients who elected 1 of 6 other surgical procedures, it was found that the former had almost twice the mortality of the latter. Further analysis showed no significant correlation between diabetes and increased mortality. In a similar 5-year mortality analysis, patients with cataracts who were younger than 75 years had significantly higher age-specific rates of mortality than would be expected from US life tables.
- These data imply an association between senile cataracts and increased mortality. Meddings et al suggest that senile cataract may be a marker of generalized tissue aging.[1] Hirsch and Schwartz who proposed the concept that senile cataracts reflect systemic phenomena rather than only a localized ocular disease share this view.[2]

Race

Although race has been suggested as a possible risk factor for senile cataract, scarce literature exists to prove this theory. However, it has been observed that unoperated cataracts account for a higher percentage of blindness among blacks compared to whites.

Sex

Studies on the prevalence of senile cataract between males and females have yielded contrasting results.

- In the Framingham Eye Study from 1973-75, females had a higher prevalence than males in both lens changes (63% vs 54.1%) and senile cataract (17.1% vs 13.2%).

- Sperduto and Hiller noted that each of the 3 types of senile lens opacities was found more often in women than in men.[3] In a separate investigation by Nishikori and Yamomoto, the male-to-female ratio was 1:8 with a female predominance in patients older than 65 years who were operated on for senile cataract.[4]
- In a hospital-based, case-control study of senile cataract conducted in Japan, it was observed that an increased risk of cataract was found in males who were presently spending 7 hours or more outdoors and in females with 4 or fewer remaining teeth. However, in another analysis by Martinez et al, no sexual difference was noted in the prevalence of senile cataract.[5]

Age

Age is an important risk factor for senile cataract. As a person ages, the chance of developing a senile cataract increases. In the Framingham Eye Study from 1973-1975, the number of total and new cases of senile cataract rose dramatically from 23.0 cases per 100,000 and 3.5 cases per 100,000, respectively, in persons aged 45-64 years to 492.2 cases per 100,000 and 40.8 cases per 100,000 in persons aged 85 years and older.

Clinical

History

Careful history taking is essential in determining the progression and functional impairment in vision resulting from the cataract and in identifying other possible causes for the lens opacity. A patient with senile cataract often presents with a history of gradual progressive deterioration and disturbance in vision. Such visual aberrations are varied depending on the type of cataract present in the patient.

- Decreased visual acuity
    - Decreased visual acuity is the most common complaint of patients with senile cataract. The cataract is considered clinically relevant if visual acuity is affected significantly. Furthermore, different types of cataracts produce different effects on visual acuity.
    - For example, a mild degree of posterior subcapsular cataract can produce a severe reduction in visual acuity with near acuity affected more than distance vision, presumably as a result of accommodative miosis. However, nuclear sclerotic cataracts often are associated with decreased distance acuity and good near vision.
    - A cortical cataract generally is not clinically relevant until late in its progression when cortical spokes compromise the visual axis. However, instances exist when a solitary cortical spoke occasionally results in significant involvement of the visual axis.
- Glare
    - Increased glare is another common complaint of patients with senile cataracts. This complaint may include an entire spectrum from a decrease in contrast sensitivity in brightly lit environments or disabling glare during the day to glare with oncoming headlights at night.
    - Such visual disturbances are prominent particularly with posterior subcapsular cataracts and, to a lesser degree, with cortical cataracts. It is associated less frequently with nuclear sclerosis. Many patients may tolerate moderate levels of glare without much difficulty, and, as such, glare by itself does not require surgical management.
- Myopic shift
    - The progression of cataracts may frequently increase the diopteric power of the lens resulting in a mild-to-moderate degree of myopia or myopic shift. Consequently, presbyopic patients report an increase in their near vision and less need for reading glasses as they experience the so-called second sight. However, such occurrence is temporary, and, as the optical quality of the lens deteriorates, the second

sight is eventually lost.

- Typically, myopic shift and second sight are not seen in cortical and posterior subcapsular cataracts. Furthermore, asymmetric development of the lens-induced myopia may result in significant symptomatic anisometropia that may require surgical management.

- Monocular diplopia
  - At times, the nuclear changes are concentrated in the inner layers of the lens, resulting in a refractile area in the center of the lens, which often is seen best within the red reflex by retinoscopy or direct ophthalmoscopy.
  - Such a phenomenon may lead to monocular diplopia that is not corrected with spectacles, prisms, or contact lenses.

Physical

After a thorough history is taken, careful physical examination must be performed. The entire body habitus is checked for abnormalities that may point out systemic illnesses that affect the eye and cataract development.

- A complete ocular examination must be performed beginning with visual acuity for both near and far distances. When the patient complains of glare, visual acuity should be tested in a brightly lit room. Contrast sensitivity also must be checked, especially if the history points to a possible problem.
- Examination of the ocular adnexa and intraocular structures may provide clues to the patient's disease and eventual visual prognosis.
  - A very important test is the swinging flashlight test which detects for a Marcus Gunn pupil or a relative afferent pupillary defect (RAPD) indicative of optic nerve lesions or diffuse macular involvement. A patient with RAPD and a cataract is expected to have a very guarded visual prognosis after cataract extraction.
  - A patient with long-standing ptosis since childhood may have occlusion amblyopia, which may account more for the decreased visual acuity rather than the cataract. Similarly, checking for problems in ocular motility at all directions of gaze is important to rule out any other causes for the patient's visual symptoms.
- Slit lamp examination should not only concentrate on evaluating the lens opacity but the other ocular structures as well (eg, conjunctiva, cornea, iris, anterior chamber).
  - Corneal thickness and the presence of corneal opacities, such as corneal guttata, must be checked carefully.
  - Appearance of the lens must be noted meticulously before and after pupillary dilation.
  - The visual significance of oil droplet nuclear cataracts and small posterior subcapsular cataracts is evaluated best with a normal-sized pupil to determine if the visual axis is obscured. However, exfoliation syndrome is appreciated with the pupil dilated, revealing exfoliative material on the anterior lens capsule.
  - After dilation, nuclear size and brunescence as indicators of cataract density can be determined prior to phacoemulsification surgery. The lens position and integrity of the zonular fibers also should be checked because lens subluxation may indicate previous eye trauma, metabolic disorders, or hypermature cataracts.
- The importance of direct and indirect ophthalmoscopy in evaluating the integrity of the posterior pole must be underscored. Optic nerve and retinal problems may account for the visual disturbance experienced by the patient. Furthermore, the prognosis after lens extraction is affected significantly by detection of pathologies in the posterior pole preoperatively (eg, macular edema, age-related macular degeneration).

Causes

Numerous studies have been conducted to identify risk factors for development of senile cataracts. Various culprits have been implicated, including environmental conditions, systemic diseases, diet, and age.

West and Valmadrid stated that age-related cataract is a multifactorial disease with different risk factors associated to the different cataract types.[6] In addition, they stated that cortical and posterior subcapsular cataracts were related closely to environmental stresses, such as ultraviolet (UV) exposure, diabetes, and drug ingestion. However, nuclear cataracts seem to have a correlation with smoking. Alcohol has been associated with all cataract types.

A similar analysis was completed by Miglior et al.[7] They found that cortical cataracts were associated with the presence of diabetes for more than 5 years and increased serum potassium and sodium levels. A history of surgery under general anesthesia and the use of sedative drugs were associated with reduced risks of senile cortical cataracts. Posterior subcapsular cataracts were associated with steroid use and diabetes, while nuclear cataracts had significant correlations with calcitonin and milk intake. Mixed cataracts were linked with a history of surgery under general anesthesia.

- Systemic diseases and senile cataract
    - Senile cataracts have been associated with a lot of systemic illnesses, to include the following: cholelithiasis, allergy, pneumonia, coronary disease and heart insufficiency, hypotension, hypertension, mental retardation, and diabetes.
    - Systemic hypertension was found to significantly increase the risk for posterior subcapsular cataracts. In a related study by Jahn et al, hypertriglyceridemia, hyperglycemia, and obesity was found to favor the formation of posterior subcapsular cataracts at an early age.[8]
    - A possible pathway for the role of hypertension and glaucoma in senile cataract formation was proposed with induced changes in the protein conformational structures in the lens capsules, subsequently causing alterations in membrane transport and permeability of ions, and, finally, increasing intraocular pressure resulting in the exacerbation of cataract formation.
- UV light and senile cataract
    - The association of UV light and development of senile cataract has generated much interest. One hypothesis implies that senile cataracts, particularly cortical opacities, may be the result of thermal damage to the lens.
    - An animal model by Al-Ghadyan and Cotlier documented an increase in the temperature of the posterior chamber and lens of rabbits after exposure to sunlight due to an ambient temperature effect through the cornea and to increased body temperature.[9]
    - In related studies, people living in areas with greater UV exposure were more likely to develop senile cataracts and to develop them earlier than people residing in places with less UV exposure.
- Other risk factors
    - Significant associations with senile cataract were noted with increasing age, female sex, social class, and myopia. Consistent evidence from the study of West and Valmadrid suggested that the prevalence of all cataract types was lower among those with higher education.[6] Workers exposed to infrared radiation also were found to have a higher incidence of senile cataract development.
    - Although myopia has been implicated as a risk factor, it was shown that persons with myopia who had worn eyeglasses for at least 20 years underwent cataract extraction at a significantly older age than emmetropes, implying a protective effect of the eyeglasses to solar UV radiation.
    - The role of nutritional deficiencies in senile cataract has not been proven or established. However, a high intake of the 18-carbon polyunsaturated fatty acids linoleic acid and linolenic acid reportedly may result in

an increased risk of developing age-related nuclear opacity.

Differential Diagnoses

Cataract, Traumatic

Other Problems to Be Considered

Aside from being age related and due to trauma, cataract formation in adult patients also may be due to chronic uveitis, long-term steroid use, or posterior pole pathologies (eg, intraocular tumor, long-standing retinal detachment).

Workup

Laboratory Studies

- Diagnosis of senile cataract is made basically after a thorough history and physical examination are performed. Laboratory tests are requested as part of the preoperative screening process to detect coexisting diseases (eg, diabetes mellitus, hypertension, cardiac anomalies). Studies have shown that thrombocytopenia may lead to increased perioperative bleeding and, as such, should be properly detected and managed before surgery.

Imaging Studies

- Ocular imaging studies (eg, ultrasound, CT scan, MRI) are requested when a posterior pole pathology is suspected and an adequate view of the back of the eye is obscured by the dense cataract. This is helpful in planning out the surgical management and in providing a more guarded postoperative prognosis for the visual recovery of the patient.

Other Tests

- Other special tests can be performed when coexisting ocular diseases are suspected, especially in identifying preoperative visual loss resulting from them. Aside from the routine visual acuity testing, testing for brightness acuity and contrast sensitivity and confrontation visual field testing can be performed to assess visual function. Patients with a history of glaucoma, optic nerve disease, or retinal abnormality should undergo an automated visual field test to document the degree of preoperative field loss.

- In patients suspected of having a macular problem, the following tests may be performed to evaluate macular function: Maddox rod test, photostress recovery test, blue-light entoptoscopy, Purkinje entoptic phenomenon, and visual-evoked response and electroretinography (VER-ERG).
    - In patients with dense cataracts that preclude adequate visualization of the fundus, a Maddox rod test can be used to grossly evaluate macular function with detection of a large scotoma, represented as a loss of the red line, a sign suggestive of a macular pathology.
    - While the photostress recovery test is a semiquantitative estimate of macular function, both blue-light entoptoscopy and Purkinje entoptic phenomenon are subjective means of evaluating macular integrity. The most objective method of measuring macular function is VER-ERG.

- Several measurements should be taken preoperatively, particularly in an anticipated cataract extraction with intraocular lens (IOL) implantation.
    - Careful refraction must be performed on both eyes in planning the IOL to be implanted. The power of the IOL on the operated eye must be compatible with the refractive error of the fellow eye to avoid complications (eg, postoperative anisometropia).
    - An accurate biometry also should be performed to calculate for the IOL power to be used.

- Corneal integrity, specifically the endothelial layer, must be assessed very well through pachymetry and specular microscopy to predict postoperative corneal morbidities (eg, corneal edema, corneal decompensation) and to weigh the risks versus the benefits of performing cataract extraction.

Histologic Findings

Nuclear cataracts are characterized by homogeneity of the lens nucleus with loss of cellular laminations, while cortical cataracts typically manifest with hydropic swelling of the lens fibers with globules of eosinophilic material (morgagnian globules) seen in slitlike spaces between lens fibers. Finally, a posterior subcapsular cataract is associated with posterior migration of the lens epithelial cells in the posterior subcapsular area, with aberrant enlargement of the epithelial cells (Wedl or bladder cells).

Costello et al examined senile cataracts using electron microscopy to highlight differences in the cellular architecture of the various forms of age-related lens changes.[10] Comparisons were made between a typical nuclear cataract with a central opacity and a transparent rim, and a more advanced or mature, completely opaque nuclear cataract. The former was described as having no obvious cell disruption, cellular debris, or changes that could readily account for the central opacity. The fiber cells had intact uniformly stained cytoplasms with well-defined plasma membrane borders and gap junctions. The mature cataract exhibited various types of cell disruption in the perimeter but not in the core of the nucleus in the form of globules, vacuoles, multilamellar membranes, and clusters of highly undulating membranes.

Staging

Clinical staging of senile cataract is based largely on the visual acuity of the patient. A patient who cannot read better than 20/200 on the visual acuity chart is said to have a mature cataract. If the patient can distinguish letters at lines better than 20/200, then the cataract is described as being immature. An incipient cataract is found in a patient who can still read at 20/20 but possesses a lens opacity as confirmed by slit lamp examination.

Treatment

Medical Care

No time-tested and proven medical treatment exists to delay, prevent, or reverse the development of senile cataracts.

Aldose reductase inhibitors, which are believed to inhibit the conversion of glucose to sorbitol, have shown promising results in preventing sugar cataracts in animals. Other anticataract medications being investigated include sorbitol-lowering agents, aspirin, glutathione-raising agents, and antioxidant vitamins C and E.

Surgical Care

The definitive management for senile cataract is lens extraction. Over the years, various surgical techniques have evolved from the ancient method of couching to the present-day technique of phacoemulsification. Almost parallel is the evolution of the IOLs being used, which vary in ocular location, material, and manner of implantation. Depending on the integrity of the posterior lens capsule, the 2 main types of lens surgery are the intracapsular cataract extraction (ICCE) and the extracapsular cataract extraction (ECCE). Below is a general description of the 3 commonly used surgical procedures in cataract extraction, namely ICCE, standard ECCE, and phacoemulsification. Reading books on cataract surgeries for a more in-depth discussion of the topic, particularly with regard to technique and procedure, is also recommended.

- Intracapsular cataract extraction
    - Prior to the onset of more modern microsurgical instruments and better IOLs, ICCE was the preferred method for cataract removal. It involves extraction of the entire lens, including the posterior capsule. In performing this technique, there is no need to worry about subsequent development and management of

capsular opacity. The technique can be performed with less sophisticated equipment and in areas where operating microscopes and irrigating systems are not available.

- However, a number of disadvantages and postoperative complications accompany ICCE. The larger limbal incision, often 160°-180°, is associated with the following risks: delayed healing, delayed visual rehabilitation, significant against-the-rule astigmatism, iris incarceration, postoperative wound leaks, and vitreous incarceration. Corneal edema is a common intraoperative and immediate postoperative complication.
- Furthermore, endothelial cell loss is greater in ICCE than in ECCE. The same is true about the incidence of postoperative cystoid macular edema (CME) and retinal detachment. The broken integrity of the vitreous can lead to postoperative complications even after a seemingly uneventful operation. Finally, because the posterior capsule is not intact, the IOL to be implanted must either be placed in the anterior chamber or sutured to the posterior chamber. Both techniques are more difficult to perform than simply placing an IOL in the capsular bag and are associated with postoperative complications, the most notorious of which is pseudophakic bullous keratopathy.
- Although the myriad of postoperative complications has led to the decline in popularity and use of ICCE, it still can be used in cases where zonular integrity is too severely impaired to allow successful lens removal and IOL implantation in ECCE. Furthermore, ICCE can be performed in remote areas where more sophisticated equipment is not available.
- ICCE is contraindicated absolutely in children and young adults with cataracts and cases with traumatic capsular rupture. Relative contraindications include high myopia, Marfan syndrome, morgagnian cataracts, and vitreous presenting in the anterior chamber.

- Extracapsular cataract extraction
  - In contrast to ICCE, ECCE involves the removal of the lens nucleus through an opening in the anterior capsule with retention of the integrity of the posterior capsule. ECCE possesses a number of advantages over ICCE, most of which are related to an intact posterior capsule, as follows:
    - A smaller incision is required in ECCE, and, as such, less trauma to the corneal endothelium is expected.
    - Short- and long-term complications of vitreous adherence to the cornea, iris, and incision are minimized or eliminated.
    - A better anatomical placement of the IOL is achieved with an intact posterior capsule.
    - An intact posterior capsule also (1) reduces the iris and vitreous mobility that occurs with saccadic movements (eg, endophthalmodonesis), (2) provides a barrier restricting the exchange of some molecules between the aqueous and the vitreous, and (3) reduces the incidence of CME, retinal detachment, and corneal edema.
    - Conversely, an intact capsule prevents bacteria and other microorganisms inadvertently introduced into the anterior chamber during surgery from gaining access to the posterior vitreous cavity and causing endophthalmitis.
    - Secondary IOL implantation, filtration surgery, corneal transplantation, and wound repairs are performed more easily with a higher degree of safety with an intact posterior capsule.
  - The main requirement for a successful ECCE and posterior capsule IOL implantation is zonular integrity. As such, when zonular support is insufficient or appears suspect to allow a safe removal of the cataract via ECCE, ICCE or pars plana lensectomy should be considered.

- Standard ECCE and phacoemulsification
  - Standard ECCE and phacoemulsification are similar in that extraction of the lens nucleus is performed through an opening in the anterior capsule or anterior capsulotomy. Both techniques also require mechanisms to irrigate and aspirate fluid and cortical material during surgery. Finally, both procedures place the IOL in the posterior capsular bag that is more anatomical than the anteriorly placed IOL.

- Needless to say, significant differences exist between the 2 techniques. Removal of the lens nucleus in ECCE can be performed manually in standard ECCE or with an ultrasonically driven needle to fragment the nucleus of the cataract and then to aspirate the lens substrate through a needle port in a process termed phacoemulsification.
- The more modern of the 2 techniques, phacoemulsification offers the advantage of using smaller incisions, minimizing complications arising from improper wound closure, and affording more rapid wound healing and faster visual rehabilitation. Furthermore, it uses a relatively closed system during both phacoemulsification and aspiration with better control of intraocular pressure during surgery, providing safeguards against positive vitreous pressure and choroidal hemorrhage. However, more sophisticated machines and instruments are required to perform phacoemulsification.
- Ultimately, the choice of which of the 2 procedures to use in cataract extraction depends on the patient, the type of cataract, the availability of the proper instruments, and the degree at which the surgeon is comfortable and proficient in performing standard ECCE or phacoemulsification.

Bell et al reviewed exposure to alpha-adrenergic blockers frequently prescribed to treat benign prostatic hypertrophy (BPH) and their association with serious postoperative adverse effects following cataract surgery.[11] The study included more than 96,000 older men who had cataract surgery over a 5-year period (3.7% had recent exposure to tamsulosin and 7.7% had recent exposure to other alpha-blockers). Exposure to tamsulosin within 14 days of cataract surgery was significantly associated with serious postoperative ophthalmic adverse events (7.5% vs 2.7%; adjusted odds ratio [OR], 2.33; 95% confidence interval [CI], 1.22-4.43), specifically intraoperative floppy iris syndrome and its complications (ie, retinal detachment, lost lens or fragments, endophthalmitis). No significant associations were noted with exposure to other alpha-blocker medications (7.5% vs 8%; adjusted OR, 0.91; 95% CI, 0.54-1.54) or to previous exposure to tamsulosin or other alpha-blockers.

Consultations

Prior to surgery, a thorough preoperative evaluation must be conducted, which would also include a thorough explanation of the procedure to be performed and its accompanying risks.

- Not all senile cataracts require removal at the time of diagnosis. If vision is not impaired significantly and functionally or if the patient is not prepared medically, psychologically, and financially for surgery, periodic consultations are encouraged to assess progression of the cataract.
- Postoperatively, regular follow-up visits are necessary to monitor visual rehabilitation, as well as to detect and address any immediate and late complications arising from the surgery.

Diet

In relation to the surgery, no established dietary restrictions exist that would affect the course of the operation.

Activity

After surgery, the patient is dissuaded from performing activities that would increase the intraocular pressure, especially after undergoing ICCE or standard ECCE. These activities include lifting heavy loads, chronic vigorous coughing, and straining.

Medication

No drug is available that has been proven to prevent the progression of senile cataracts. Medical therapy is used preoperatively and postoperatively to ensure a successful operation and subsequent visual rehabilitation.

Mydriatics

Autonomic drugs used to ensure maximal pupillary dilation preoperatively, which is essential for a successful lens extraction. Short-acting mydriatics often are used. Most commonly used mydriatics are phenylephrine hydrochloride and tropicamide.

Phenylephrine HCl (Neo-Synephrine)

Direct-acting adrenergic agent available in 2.5% and 10% concentrations. Acts locally as potent vasoconstrictor and mydriatic by constricting ophthalmic blood vessels and radial muscles of the iris. Favorably used by many ophthalmologists because of rapid onset and moderately prolonged action, as well as the fact that it does not produce compensatory vasodilation. Most ophthalmologists prefer 2.5% to 10% concentration because of fewer risks of severe adverse systemic effects. Onset of action is within 30-60 min lasting for 3-5 h.

Dosing

Adult

1 gtt 30-60 min prior to surgery, (2.5 or 10%) on affected eye until maximal pupillary dilation achieved

Pediatric

2.5% solution: 1 gtt 30-60 min to affected eye prior to surgery, until maximal pupillary dilation achieved

Interactions

Bretylium may potentiate action of vasopressors on adrenergic receptors, possibly resulting in arrhythmias; MAOIs may significantly enhance adrenergic effects of phenylephrine, and pressor response may be increased 2- to 3-fold; guanethidine may increase pressor response of direct-acting vasopressors, possibly resulting in severe hypertension; concomitant use of 10% ophthalmic solutions and systemic beta-blockers may cause acute hypertension and rupture of congenital cerebral aneurysm; may potentiate cardiovascular depressant effects of potent inhalation anesthetics

Contraindications

Documented hypersensitivity; narrow-angle glaucoma; 10% solution is contraindicated in infants; aneurysms; preexisting cardiovascular diseases; cardiovascular complications, including ventricular arrhythmias and myocardial infarctions

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Exceeding recommended dosages or applying to instrumented, traumatized, diseased, or postsurgical eye or adnexa, or patients with suppressed lacrimation, as during anesthesia, may result in absorption of sufficient drug to produce systemic vasopressor response; significant elevation in blood pressure reported following conjunctival instillation of recommended dosages (10% ophthalmic solution); caution in children of low body weight, elderly persons, and persons with insulin-dependent diabetes, hypertension, hyperthyroidism, generalized arteriosclerosis, or cardiovascular disease; closely monitor patients who develop symptoms of high blood pressure; any mydriatic is contraindicated in patients with glaucoma since it occasionally may increase intraocular pressure; during times of temporary pupillary dilation or when vasoconstriction of intrinsic vessels lower intraocular pressure, may free adhesions; rebound miosis in older persons a day after receiving may occur, but reinstillation of drug may result in reduction of mydriasis; may be of particular importance when using drops prior to cataract surgery; due to a strong action of drug on dilator muscle, older individuals may develop transient pigment floaters in aqueous humor 30-45 min following administration presenting with an appearance similar to anterior uveitis or microscopic hyphema; to prevent pain, a drop of suitable topical anesthetics

may be applied prior to use

Corticosteroids

Help decrease and control inflammatory response following cataract surgery especially in the immediate postoperative period. The most commonly used ophthalmic steroid is prednisolone acetate 1%. Dexamethasone 0.1% ophthalmic solution sometimes is used as an alternative.

Prednisolone acetate 1% (AK-Pred, Pred Forte)

Topical anti-inflammatory agent for ophthalmic use. A good glucocorticoid that, on the basis of weight, has 3-5 times anti-inflammatory potency of hydrocortisone. Glucocorticoids inhibit edema, fibrin deposition, capillary dilation, and phagocytic migration of acute inflammatory response as well as capillary proliferation, deposition of collagen, and scar formation. Indicated for treatment of steroid-responsive inflammation of palpebral and bulbar conjunctiva, cornea, and anterior segment of the globe.

Dosing

Adult

During initial 24-48 h postoperative period may be used frequently even up to q1h, followed by tapered dosing to bid/qid

Pediatric

Not established

Interactions

None reported

Contraindications

Documented hypersensitivity; viral, fungal, mycobacterial, or tubercular infections

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Do not discontinue therapy prematurely; administration beyond 20 mL should be made by physician with aid of magnification, such as slit-lamp biomicroscopy, and where appropriate, fluorescein staining; if signs and symptoms fail to improve after 2 d, reevaluate patient; prolonged use may result in glaucoma with damage to optic nerve, defects in visual acuity, and fields of vision, and in posterior subcapsular cataract formation; if drops are used for 10 d or longer, monitor intraocular pressure periodically; prolonged use may suppress host immune response and, thus, increase hazard of secondary ocular infection; fungal infections of cornea are prone to develop coincidentally with long-term use; suspect fungal invasion in any persistent corneal ulceration where a corticosteroid has been used or is in use; obtain fungal cultures when appropriate

Dexamethasone (Ocu-Dex)

Decreases inflammation by suppressing migration of polymorphonuclear leukocytes and reducing capillary permeability.

Dosing

Adult

2 gtt into conjunctival sac q1h while awake and q2h at night; gradually reduce to q3-4h, then to tid/qid

Pediatric

Not established

Interactions

None reported

Contraindications

Documented hypersensitivity; viral, fungal, mycobacterial, or tubercular infections

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Prolonged use may increase hazard of secondary ocular infection; suspect fungal invasion in any persistent corneal ulceration where a corticosteroid has been used or is in use; obtain fungal cultures when appropriate

Antibiotics

Broad-spectrum antibiotic ophthalmic solutions often are used prophylactically in immediate postoperative period. A number of topical antibiotics are used depending on surgeon's preference, but, generally, medications are active against both gram-positive and gram-negative organisms.

Ciprofloxacin (Ciloxan)

Active against a broad spectrum of gram-positive and gram-negative organisms. Bactericidal action results from interference with enzyme DNA gyrase needed for bacterial DNA synthesis. In vitro and clinical studies have shown it to be active against following organisms: gram-positive (ie, *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Streptococcus viridans*) and gram-negative (ie, *Haemophilus influenzae*, *Pseudomonas aeruginosa*, *Serratia marcescens*). Other organisms have been found to be susceptible in vitro but have yet to be established firmly by clinical studies.

Dosing

Adult

1-2 gtt in the eye(s) tid/qid for 1-2 wk as postoperative prophylaxis following cataract extraction

Pediatric

<18 years: Not recommended
>18 years: Administer as in adults

Interactions

Specific drug interaction studies not conducted with ophthalmic ciprofloxacin; systemic administration of some

quinolones has been shown to elevate plasma concentrations of theophylline; interfere with metabolism of caffeine; enhance effects of anticoagulant warfarin and derivatives; associated with transient elevations in serum creatinine in patients receiving cyclosporine concomitantly

Contraindications

Documented hypersensitivity; viral, mycobacterial, and fungal eye infections; avoid coadministration with steroid combinations after uncomplicated removal of a foreign body from cornea

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

A white crystalline precipitate located in superficial portion of corneal defect may occur (onset starts in 1-7 d); precipitate usually is cleared within 2 wk and does not adversely affect clinical course or outcome; do not use in ocular infections that may become systemic; superinfections may occur with prolonged or repeated antibiotic therapy

---

Erythromycin (E-Mycin)

Indicated for infections caused by susceptible strains of microorganisms and for prevention of corneal and conjunctival infections.

Dosing

Adult

Apply 0.5-inch (1.25 cm) ribbon 2-8 times/d depending on severity of infection

Pediatric

Apply as in adults

Interactions

None reported

Contraindications

Documented hypersensitivity; viral, mycobacterial, or fungal infections of eye; patients using steroid combinations after uncomplicated removal of a foreign body from cornea also should avoid using this product

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Do not use topical antibiotics to treat ocular infections that may become systemic; prolonged or repeated antibiotic therapy may result in bacterial or fungal overgrowth of nonsusceptible organisms and may lead to a secondary infection; take appropriate measures if superinfection occurs

Nonsteroidal anti-inflammatory ophthalmics

Used for pain and inflammation associated with cataract surgery.

Nepafenac ophthalmic (Nevanac)

Nonsteroidal anti-inflammatory prodrug for ophthalmic use. Following administration, converted by ocular tissue hydrolases to amfenac, an NSAID. Inhibits prostaglandin H synthase (cyclooxygenase), an enzyme required for prostaglandin production. Indicated for treatment of pain and inflammation associated with cataract surgery.

Dosing

Adult

Shake well before use
Apply 1 gtt to affected eye(s) tid; initiate 1 d prior to cataract surgery and continue on day of surgery and for 2 wk postsurgery

Pediatric

Not established

Interactions

Concomitant use with topical corticosteroids may increase potential for healing problems

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

May slow or delay healing; may cause keratitis; long-term use may cause epithelial breakdown, corneal thinning, corneal erosion, and corneal ulceration or perforation, which may threaten vision; frequent adverse effects (5-10%) include capsular opacity, decreased visual acuity, foreign body sensation, increased intraocular pressure, and a sticky sensation

Follow-up

Further Inpatient Care

- Most cataract surgeries are performed on an outpatient basis, especially with the onset of phacoemulsification performed under topical anesthesia. Often, patients are discharged from the clinic as soon as they have recovered from the emotional stress of the procedure. Patients are sent home on topical steroids and antibiotics either separately or in combination. An eye shield is placed on the newly operated eye and removed a few hours later.

Further Outpatient Care

- On the first postoperative day, visual acuity should be consistent with the refractive state of the eye, the clarity of the cornea and media, and the visual potential of the retina and optic nerve. Mild edema of the eyelid may be

evident, as well as some conjunctival injection. The cornea is normally clear with minimal edema and striae. The anterior chamber should be deep with mild cellular reaction. It is important to check whether the posterior capsule is intact and whether the IOL is positioned properly. The red reflex must be strong and clear and the intraocular pressures should be within normal limits. Transient intraocular pressure elevations may be observed and attributed to retained viscoelastic.

- Significant improvement of these initial findings is to be expected in subsequent postoperative evaluation as the ocular inflammation subsides typically within 2 weeks. Topical steroids and antibiotics are tapered accordingly. Refraction is believed to be stable at the sixth to eighth postoperative week, at which time corrective lenses can be prescribed. Significant postoperative astigmatism can be addressed by suture removal by the sixth week as guided by keratometry or corneal topography.

Inpatient & Outpatient Medications

- During the postoperative period, the patient is prescribed topical 1% prednisolone acetate, which is applied every hour for the first day, then tapered depending on the inflammatory state of the eye. Studies have shown that topical ketorolac tromethamine provides adequate postoperative control of intraocular inflammation without the risk of increased intraocular pressure, which may be associated with steroid use. A broad-spectrum topical antibiotic also is given 4-6 times a day for 1-2 weeks.

Deterrence/Prevention

- Age is believed to be the most significant risk factor for senile cataract and, as such, it is almost inevitable to develop some degree of lens opacity as one becomes older. No study has established firmly whether avoidance of some of the risk factors for senile cataract (eg, UV exposure, hypercholesterolemia, diabetes mellitus) will lessen the chance of developing a senile cataract.

Complications

- The following are the major intraoperative complications encountered during cataract surgery:
    - Shallow or flat anterior chamber
    - Capsular rupture
    - Corneal edema
    - Suprachoroidal hemorrhage or effusion
    - Expulsive choroidal hemorrhage
    - Retained lens material
    - Vitreous disruption and incarceration into wound
    - Iridodialysis
    - Retinal light toxicity

- The following are the major immediate postoperative complications encountered during cataract surgery often seen within a few days or weeks after the operation:
    - Flat or shallow anterior chamber due to wound leak
    - Choroidal detachment
    - Pupillary block
    - Ciliary block
    - Suprachoroidal hemorrhage

- Stromal and epithelial edema
- Hypotony
- Brown-McLean syndrome (peripheral corneal edema with a clear central cornea most frequently seen following ICCE)
- Vitreocorneal adherence and persistent corneal edema
- Delayed choroidal hemorrhage
- Hyphema
- Elevated intraocular pressure (often due to retained viscoelastic)
- Cystoid macular edema - Studies have shown that diclofenac was more effective than topical steroids in preventing CME.[12]
- Retinal detachment - Significant risk factors include axial length greater than 25 mm, age younger than 65 years, and intraoperative complications.[13]
- Acute endophthalmitis
- Uveitis-glaucoma-hyphema (UGH) syndrome

- The following are the major late postoperative complications seen weeks or months after cataract surgery:
  - Suture-induced astigmatism
  - Pupillary capture
  - Decentration and dislocation of the IOL
  - Corneal edema and pseudophakic bullous keratopathy
  - Chronic uveitis
  - Chronic endophthalmitis
  - Wrong power of IOL used

- At any stage of the postoperative recovery of the eye, a risk of noninfectious endophthalmitis and infectious endophthalmitis exists. Noninfectious endophthalmitis is believed to be a multifactorial process or an interindividual variable response to a common factor as a hypersensitivity reaction. Treatment may range from the use of topical, transseptal, or oral steroids to the explantation of the intraocular lens.

- Although of low incidence, infectious endophthalmitis may lead to severe vision loss and blindness.[14] *Staphylococcus epidermidis* is the most common isolated organism, and rupture of the posterior capsule is one of the most common risk factors.[14] Of late, a significant increase in the incidence of gram-positive bacteria in bacterial isolates from postoperative eyes suspected of having endophthalmitis has been observed. Furthermore, a significant increase in resistance to ciprofloxacin has occurred. Seemingly, the spectrum of bacteria causing postcataract endophthalmitis is changing partly because of an increased resistance to mainstay antibiotics in the treatment of endophthalmitis.

Prognosis

- In the absence of any other accompanying ocular disease prior to surgery, which would affect significantly the visual outcome, such as macular degeneration or optic nerve atrophy, a successful uncomplicated standard ECCE or phacoemulsification carries a very promising visual prognosis of gaining at least 2 lines in the Snellen distance vision chart. The main cause of visual morbidity postoperatively is CME. A major risk factor affecting visual prognosis is the presence of diabetes mellitus and diabetic retinopathy.

Patient Education

- To date, no established guidelines are available for the prevention of senile cataracts. Education programs are geared toward early detection and surgical intervention when vision is impaired functionally. With the advent of phacoemulsification, patients are advised against delaying lens extraction to the point when the cataract is hard and mature and the likelihood of postoperative complications increases.
- For excellent patient education resources, visit eMedicine's Eye and Vision Center. Also, see eMedicine's patient education article Cataracts.

Miscellaneous

Medicolegal Pitfalls

- Tasked with the pivotal role of restoring vision to a functionally impaired patient, the cataract surgeon must now also confront issues that may ultimately jeopardize the medical practice, for example, malpractice litigation, administrative sanctions in the form of expulsion from participation in third-party reimbursement plans, revocation or cancellation of hospital privileges, and, ultimately, limitation or loss of the license to practice. In addition, cut-throat competition coupled with profound changes in the eye care delivery system makes it necessary for the surgeon to be aware of the legal intricacies affecting increased productivity and income creation.
- The onset of office setting phacoemulsification under topical anesthesia performed in less than 30 minutes ironically has turned the art of cataract surgery into an industry. Remaining productive, creative, and fairly paid is both a concern and a challenge to the modern-day cataract surgeon. As such, the surgeon must be well informed of what is and what is not legal, to thrive and remain in business. Rabin grouped the current legal concerns of the ophthalmologist into 3 categories: (1) conventional malpractice liability, (2) surveillance for regulatory compliance, and (3) practice productivity and income creation.[15]

References

1. Meddings DR, Marion SA, Barer ML, Evans RG, Green B, Hertzman C, et al. Mortality rates after cataract extraction. *Epidemiology*. May 1999;10(3):288-93. [Medline].
2. Hirsch RP, Schwartz B. Increased mortality among elderly patients undergoing cataract extraction. *Arch Ophthalmol*. Jul 1983;101(7):1034-7. [Medline].
3. Sperduto RD, Hiller R. The prevalence of nuclear, cortical, and posterior subcapsular lens opacities in a general population sample. *Ophthalmology*. Jul 1984;91(7):815-8. [Medline].
4. Nishikiori T, Yamamoto K. Epidemiology of cataracts. *Dev Ophthalmol*. 1987;15:24-7. [Medline].
5. Martinez GS, Campbell AJ, Reinken J, Allan BC. Prevalence of ocular disease in a population study of subjects 65 years old and older. *Am J Ophthalmol*. Aug 1982;94(2):181-9. [Medline].
6. West SK, Valmadrid CT. Epidemiology of risk factors for age-related cataract. *Surv Ophthalmol*. Jan-Feb 1995;39(4):323-34. [Medline].
7. Miglior S, Marighi PE, Musicco M, Balestreri C, Nicolosi A, Orzalesi N. Risk factors for cortical, nuclear, posterior subcapsular and mixed cataract: a case-control study. *Ophthalmic Epidemiol*. Jun 1994;1(2):93-105. [Medline].
8. Johns KJ, Feder RS, Rosenfeld SI, et al. Lens and cataract. In: *American Academy of Ophthalmology Basic and Clinical Science Course*. Vol. 11. 1999-2000.

9. Al-Ghadyan AA, Cotlier E. Rise in lens temperature on exposure to sunlight or high ambient temperature. *Br J Ophthalmol*. Jun 1986;70(6):421-6. [Medline].

10. Costello MJ, Oliver TN, Cobo LM. Cellular architecture in age-related human nuclear cataracts. *Invest Ophthalmol Vis Sci*. Oct 1992;33(11):3209-27. [Medline].

11. Bell CM, Hatch WV, Fischer HD, Cernat G, Paterson M, Gruneir A, et al. Association between tamsulosin and serious ophthalmic adverse events in older men following cataract surgery. *JAMA*. May 20 2009;301(19):1991-6. [Full Text].

12. Asano S, Miyake K, Ota I, Sugita G, Kimura W, Sakka Y, et al. Reducing angiographic cystoid macular edema and blood-aqueous barrier disruption after small-incision phacoemulsification and foldable intraocular lens implantation: multicenter prospective randomized comparison of topical diclofenac 0.1% and betamethasone 0.1%. *J Cataract Refract Surg*. Jan 2008;34(1):57-63. [Medline].

13. Szijarto Z, Schvoller M, Poto L, Kuhn F, Kovacs B. Pseudophakic retinal detachment after phacoemulsification. *Ann Ophthalmol (Skokie)*. Jun 2007;39(2):134-9. [Medline].

14. Cao X, Liu A, Zhang J, Li Y, Jie Y, Liu W, et al. Clinical analysis of endophthalmitis after phacoemulsification. *Can J Ophthalmol*. Dec 2007;42(6):844-8. [Medline].

15. Rabin SM. Medicolegal concerns of cataract surgeons. In: *Management and Care of the Cataract Patient*. 1992:331-8.

16. Belkin M, Jacobs DR, Jackson SM, Zwick H. Senile cataracts and myopia. *Ann Ophthalmol*. Jan 1982;14(1):49-50. [Medline].

17. Bellows JG, Bellows RT. Crosslinkage theory of senile cataracts. *Ann Ophthalmol*. Feb 1976;8(2):129-35. [Medline].

18. Bilge AH, Aykan U, Akin T, Unsal U. Review of sterile, postoperative, anterior segment inflammation following cataract extraction and intraocular lens implantation. *Eur J Ophthalmol*. Mar-Apr 2005;15(2):224-7. [Medline].

19. Bunce GE, Kinoshita J, Horwitz J. Nutritional factors in cataract. *Annu Rev Nutr*. 1990;10:233-54. [Medline].

20. Burgess CA, Sowers M. Systemic hypertension and senile cataracts: an epidemiologic study. *Optom Vis Sci*. Apr 1992;69(4):320-4. [Medline].

21. Charakidas A, Kalogeraki A, Tsilimbaris M, Koukoulomatis P, Brouzas D, Delides G. Lens epithelial apoptosis and cell proliferation in human age-related cortical cataract. *Eur J Ophthalmol*. Mar-Apr 2005;15(2):213-20. [Medline].

22. Chatterjee A, Milton RC, Thyle S. Prevalence and aetiology of cataract in Punjab. *Br J Ophthalmol*. Jan 1982;66(1):35-42. [Medline].

23. Chylack LT Jr. Mechanisms of senile cataract formation. *Ophthalmology*. Jun 1984;91(6):596-602. [Medline].

24. Eckerskorn U, Hockwin O, Muller-Breitenkamp R, Chen TT, Knowles W, Dobbs RE. Evaluation of cataract-related risk factors using detailed classification systems and multivariate statistical methods. *Dev Ophthalmol*. 1987;15:82-91. [Medline].

25. Emery JM, Wilhelmus KA, Rosenberg S. Complications of phacoemulsification. *Ophthalmology*. Feb 1978;85(2):141-50. [Medline].

26. Fujiwara H, Takigawa Y, Suzuki T, Nakata K. Superoxide dismutase activity in cataractous lenses. *Jpn J Ophthalmol*. 1992;36(3):273-80. [Medline].

27. Ghafour IM, Allan D, Foulds WS. Common causes of blindness and visual handicap in the west of Scotland. *Br J Ophthalmol*. Apr 1983;67(4):209-13. [Medline].

28. Gibson JM, Rosenthal AR, Lavery J. A study of the prevalence of eye disease in the elderly in an English community. *Trans Ophthalmol Soc U K*. 1985;104 (Pt 2):196-203. [Medline].

29. Gibson JM, Shaw DE, Rosenthal AR. Senile cataract and senile macular degeneration: an investigation into possible risk factors. *Trans Ophthalmol Soc U K*. 1986;105 (Pt 4):463-8. [Medline].

30. Hirneiss C, Neubauer AS, Kampik A, Schönfeld CL. Comparison of prednisolone 1%, rimexolone 1% and ketorolac tromethamine 0.5% after cataract extraction: a prospective, randomized, double-masked study. *Graefes Arch Clin Exp Ophthalmol*. Aug 2005;243(8):768-73. [Medline].

31. Hollows F, Moran D. Cataract--the ultraviolet risk factor. *Lancet*. Dec 5 1981;2(8258):1249-50. [Medline].

32. Hu TS, Lao YX. An epidemiologic survey of senile cataract in China. *Dev Ophthalmol*. 1987;15:42-51. [Medline].

33. Imbach P, Odavic R, Bleher EA, Bucher U, Deubelbeiss KA, Wagner HP. [Autologous bone marrow reimplantation in children with advanced tumor. First experiences of feasibility]. *Schweiz Med Wochenschr*. Feb 24 1979;109(8):283-7. [Medline].

34. Jacques PF, Chylack LT Jr, McGandy RB, Hartz SC. Antioxidant status in persons with and without senile cataract. *Arch Ophthalmol*. Mar 1988;106(3):337-40. [Medline].

35. Javitt JC, Taylor HR. Cataract and latitude. *Doc Ophthalmol*. 1994-1995;88(3-4):307-25. [Medline].

36. Kador PF. Overview of the current attempts toward the medical treatment of cataract. *Ophthalmology*. Apr 1983;90(4):352-64. [Medline].

37. Kahn HA, Leibowitz HM, Ganley JP, Kini MM, Colton T, Nickerson RS, et al. The Framingham Eye Study. I. Outline and major prevalence findings. *Am J Epidemiol*. Jul 1977;106(1):17-32. [Medline].

38. Kamei A. Characterization of water-insoluble proteins in normal and cataractous human lens. *Jpn J Ophthalmol*. 1990;34(2):216-24. [Medline].

39. Kamei A. Glutathione levels of the human crystalline lens in aging and its antioxidant effect against the oxidation of lens proteins. *Biol Pharm Bull*. Sep 1993;16(9):870-5. [Medline].

40. Katoh N, Sasaki K, Shibata T, Obazawa H, Fujiwara T, Kogure F, et al. Case-control study of senile cataract in Japan: a preliminary report. *Jpn J Ophthalmol*. 1993;37(4):467-77. [Medline].

41. Ke Y, Jiang J, Chen P, Weng Y, Yang Y. Phacoemulsification and posterior chamber intraocular lens implantation. *Zhonghua Yan Ke Za Zhi*. Mar 1996;32(2):85-91. [Medline].

42. Kini MM, Leibowitz HM, Colton T, Nickerson RJ, Ganley J, Dawber TR. Prevalence of senile cataract, diabetic retinopathy, senile macular degeneration, and open-angle glaucoma in the Framingham eye study. *Am J Ophthalmol*. Jan 1978;85(1):28-34. [Medline].

43. Knekt P, Heliövaara M, Rissanen A, Aromaa A, Aaran RK. Serum antioxidant vitamins and risk of cataract. *BMJ*. Dec 5 1992;305(6866):1392-4. [Medline].

44. Lee SM, Lin SY, Li MJ, Liang RC. Possible mechanism of exacerbating cataract formation in cataractous human lens capsules induced by systemic hypertension or glaucoma. *Ophthalmic Res*. 1997;29(2):83-90. [Medline].

45. Lu M, Taylor A, Chylack LT Jr, Rogers G, Hankinson SE, Willett WC, et al. Dietary fat intake and early age-related lens opacities. *Am J Clin Nutr*. Apr 2005;81(4):773-9. [Medline].

46. Lydahl E. Infrared radiation and cataract. *Acta Ophthalmol Suppl*. 1984;166:1-63. [Medline].

47. Moffat BA, Landman KA, Truscott RJ, Sweeney MH, Pope JM. Age-related changes in the kinetics of water transport in normal human lenses. *Exp Eye Res*. Dec 1999;69(6):663-9. [Medline].

48. Mozaffarieh M, Heinzl H, Sacu S, Wedrich A. Clinical outcomes of phacoemulsification cataract surgery in diabetes patients: visual function (VF-14), visual acuity and patient satisfaction. *Acta Ophthalmol Scand*. Apr 2005;83(2):176-83. [Medline].

49. Packer M, Fishkind WJ, Fine IH, Seibel BS, Hoffman RS. The physics of phaco: a review. *J Cataract Refract Surg*. Feb 2005;31(2):424-31. [Medline].

50. Papamatheakis DG, Demers P, Vachon A, Jaimes LB, Lapointe Y, Harasymowycz PJ. Thrombocytopenia and the risks of intraocular surgery. *Ophthalmic Surg Lasers Imaging*. Mar-Apr 2005;36(2):103-7. [Medline].

51. Recchia FM, Busbee BG, Pearlman RB, Carvalho-Recchia CA, Ho AC. Changing trends in the microbiologic aspects of postcataract endophthalmitis. *Arch Ophthalmol*. Mar 2005;123(3):341-6. [Medline].

52. Robertson JM, Donner AP, Trevithick JR. A possible role for vitamins C and E in cataract prevention. *Am J Clin Nutr*. Jan 1991;53(1 Suppl):346S-351S. [Medline].

53. Sack R, Cohen J. Comparative diets of a idiopathic senile cataract and normal population: dietary risk factors in cataractogenesis. *Metab Pediatr Syst Ophthalmol*. 1987;10(1):9-13. [Medline].

54. Shaikh MR, Janjua MZ. Morphological and morphometrical study of human lens in senile cataract. *J Pak Med Assoc*. May 1997;47(5):141-4. [Medline].

55. Sharma YR, Vajpayee RB, Honavar SG. Sunlight and cortical cataract. *Arch Environ Health*. Sep-Oct 1994;49(5):414-7. [Medline].

56. Sommer A. Cataracts as an epidemiologic problem. *Am J Ophthalmol*. Mar 1977;83(3):334-9. [Medline].

57. Sommer A, Tielsch JM, Katz J, Quigley HA, Gottsch JD, Javitt JC, et al. Racial differences in the cause-specific prevalence of blindness in east Baltimore. *N Engl J Med*. Nov 14 1991;325(20):1412-7. [Medline].

58. Steinkuller PG. Cataract: the leading cause of blindness and vision loss in Africa. *Soc Sci Med*. 1983;17(22):1693-702. [Medline].

59. Street DA, Javitt JC. National five-year mortality after inpatient cataract extraction. *Am J Ophthalmol*. Mar 15 1992;113(3):263-8. [Medline].

60. Tseng SH, Tang MJ. Na,K-ATPase in lens epithelia from patients with senile cataracts. *J Formos Med Assoc*. Sep 1999;98(9):627-32. [Medline].

61. Tseng SH, Yen JS, Chien HL. Lens epithelium in senile cataract. *J Formos Med Assoc*. Feb 1994;93(2):93-8. [Medline].

62. Worgul BV, Merriam GR Jr, Medvedovsky C. Cortical cataract development--an expression of primary damage to the lens epithelium. *Lens Eye Toxic Res*. 1989;6(4):559-71. [Medline].

63. Wu TT, Amini L, Leffler CT, Schwartz SG. Cataracts and cataract surgery in mentally retarded adults. *Eye Contact Lens*. Mar 2005;31(2):50-3. [Medline].

Keywords

senile cataract, age-related cataract, vision loss, visual deficit, blindness

Contributor Information and Disclosures

Author

Vicente Victor D Ocampo, MD, Consulting Staff, Department of Ophthalmology, Asian Hospital and Medical Center, Philippines
Vicente Victor D Ocampo, MD is a member of the following medical societies: American Academy of Ophthalmology, Philippine Academy of Ophthalmology, and Philippine Ocular Inflammation Society
Disclosure: Nothing to disclose.

Coauthor(s)

C Stephen Foster, MD, FACS, FACR, FAAO, Clinical Professor of Ophthalmology, Harvard Medical School; Consulting Staff, Department of Ophthalmology, Massachusetts Eye and Ear Infirmary; Founder and President, Ocular Immunology and Uveitis Foundation, Massachusetts Eye Research and Surgery Institution
C Stephen Foster, MD, FACS, FACR, FAAO is a member of the following medical societies: Alpha Omega Alpha, American Academy of Ophthalmology, American Association of Immunologists, American College of Rheumatology, American College of Surgeons, American Federation for Clinical Research, American Medical Association, American Society for Microbiology, American Uveitis Society, Association for Research in Vision and Ophthalmology, Massachusetts Medical Society, Royal Society of Medicine, and Sigma Xi
Disclosure: Nothing to disclose.

Medical Editor

Richard W Allinson, MD, Associate Professor, Department of Surgery, Texas A&M University Health Science Center; Senior Staff Ophthalmologist, Scott and White Clinic
Richard W Allinson, MD is a member of the following medical societies: American Academy of Ophthalmology, American Medical Association, and Texas Medical Association
Disclosure: Nothing to disclose.

Pharmacy Editor

Simon K Law, MD, PharmD, Assistant Professor of Ophthalmology, Jules Stein Eye Institute; Chief of Section of Ophthalmology Surgical Services, Department of Veterans Affairs Healthcare Center, West Los Angeles
Simon K Law, MD, PharmD is a member of the following medical societies: American Academy of Ophthalmology, American Glaucoma Society, and Association for Research in Vision and Ophthalmology
Disclosure: Nothing to disclose.

Managing Editor

J James Rowsey, MD, Former Director of Corneal Services, St Luke's Cataract and Laser Institute, Florida
J James Rowsey, MD is a member of the following medical societies: American Academy of Ophthalmology, American Association for the Advancement of Science, American Medical Association, Association for Research in Vision and Ophthalmology, Florida Medical Association, Pan-American Association of Ophthalmology, Sigma Xi, and Southern Medical Association

Disclosure: Nothing to disclose.

CME Editor

Lance L Brown, OD, MD, Ophthalmologist, Affiliated With Freeman Hospital and St John's Hospital, Regional Eye Center, Joplin, Missouri
Disclosure: Nothing to disclose.

Chief Editor

Hampton Roy Sr, MD, Associate Clinical Professor, Department of Ophthalmology, University of Arkansas for Medical Sciences
Hampton Roy Sr, MD is a member of the following medical societies: American Academy of Ophthalmology, American College of Surgeons, and Pan-American Association of Ophthalmology
Disclosure: Nothing to disclose.

Further Reading