Review Article

Pediatric cataract

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Pediatric cataract is a leading cause of childhood blindness. Untreated cataracts in children lead to tremendous social, economical, and emotional burden to the child, family, and society. Blindness related to pediatric cataract can be treated with early identification and appropriate management. Most cases are diagnosed on routine screening whereas some may be diagnosed after the parents have noticed leukocoria or strabismus. Etiology of pediatric cataract is varied and diagnosis of specific etiology aids in prognostication and effective management. Pediatric cataract surgery has evolved over years, and with improving knowledge of myopic shift and axial length growth, outcomes of these patients have become more predictable. Favorable outcomes depend not only on effective surgery, but also on meticulous postoperative care and visual rehabilitation. Hence, it is the combined effort of parents, surgeons, anesthesiologists, pediatricians, and optometrists that can make all the difference.

Key words: Childhood blindness, intraocular lens in pediatric age group, pediatric cataract, visual axis opacification, zonular cataract

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Pediatric cataract managed earlier can have a tremendous impact on the lives of individuals, their families, communities, and the socioeconomic status of the country. Children who are visually impaired need to overcome a lifetime of social, emotional, and economic difficulties. This influences their education, employment, and social life. Approximately 70 million blind-person-years are caused by childhood blindness, of which about 10 million blind-person-years (14%) are due to childhood cataract.[1] India has a huge burden of 280,000–320,000 visually impaired children,[2] leading to an estimated loss of US $3,500 million.[3] The awareness in community, early detection by physician, and prompt management by ophthalmologist can address the problem and achieve the goal of elimination by 2020.

Epidemiology

Pediatric cataract is an treatable leading cause of childhood blindness. It accounts for 7.4%–15.3% of pediatric blindness and a significant avertable disability-adjusted life years.[4,5] The incidence ranges from 1.8 to 3.6/10,000 per year and the median prevalence is about 1.03/10,000 children (0.32–22.9/10,000). The prevalence of childhood cataract is higher in low-income economies (0.63–13.6/10,000) compared to that of high-income economies (0.42–2.05/10,000). There is no difference in the prevalence based on gender or laterality.[6] It has been found that during pregnancy, 67% of the mothers had a history of illness and 22% had taken medications during pregnancy.[7,8] Congenital cataract is associated with ocular abnormalities in 27% of cases and with systemic abnormalities in 22% of cases. The diagnosis of cataract is incidentally made on routine screening in 41% of cases whereas leukocoria and strabismus led to the diagnosis in 24% and 19%, respectively.[9]

Genetics

The eye starts developing at 22 days of gestation. Fibroblast growth factor (FGF) induces migration, differentiation, and is also responsible for the polarity of the lens.[10] Bone morphogenetic protein interacts with FGF during lens induction. The proteins encoded by genes Pax6, Pitx3, c-Maf, and Foxe3 are transcription factors which are crucial for lens development.[11] The mutations are most commonly autosomal dominant, and absence of the function of one copy has a severe effect on lens development.[12] The timing of insult results in involvement of the part of lens developing during that period.

Congenital cataract is hereditary in 8.3%–25% of cases, with 75% being autosomal dominant in inheritance.[13] Autosomal-dominant cataracts have varying amounts of penetrance. Autosomal-dominant cataract includes hyperferritinemia cataract syndrome, Coppock-like, Volkmann-type congenital, zonular with sutural, posterior polar, anterior polar, cerulean, zonular pulverulent, crystalline aculeiform, and myotonic dystrophy 1-like cataracts. Autosomal-recessive cataract includes Warburg micro syndrome, Hallermann–Streiff syndrome, Martsolf syndrome, Smith–Lemli-–Opitz syndrome, Rothmund–Thomson syndrome, Marinesco–Sjogren syndrome, Wilsons disease, and congenital cataract facial dysmorphism and

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neuropathy. X-linked recessive cataract includes Nance–Horan syndrome (NHS) and Norrie's disease.[14,15]

Crystallin and Connexin gene mutations are the most commonly described nonsyndromic inherited cataracts. Alpha-crystallin gene mutation results in nuclear, lamellar, zonular, and posterior polar cataracts.[16] β-crystallin gene mutation presents with variable phenotypic presentations. Anterior polar cataracts are seen with *PAX6* mutations whereas *PITX3* mutations mainly cause posterior polar cataracts.[17] Other genes responsible for major syndromic cataracts include *OCRL* (Lowe syndrome),[18] *GALK117q* (galactosemia),[19] *GLA* (Fabry's disease),[20] and *NHS* (Nance–Horan cataract–dental syndrome).[21] Congenital cataracts with more than 40 genes and loci have been isolated.[22]

Etiology

Idiopathic

Most unilateral and a significant number of bilateral cataracts are idiopathic in nature. The diagnosis of such cases is made after excluding other causes.

Hereditary cataract

Autosomal-dominant cataracts with incomplete inheritance are the most common hereditary cataracts.[13] They are usually nonsyndromic with defect in crystallin and connexin genes.

Norrie disease

Norrie disease is an X-linked recessive disorder that manifests as dense congenital cataracts, retinal folds, retinal detachment, vitreous hemorrhage, and bilateral retrolental masses consisting of hemorrhagic vascular and glial tissues. The disease is caused by mutation in the *NDP* gene located on the X chromosome. It is also associated with sensorineural hearing loss and developmental delay.[22]

Nance–Horan syndrome

NHS is an X-linked recessive disorder in which the affected males have bilateral dense stellate or nuclear cataracts, microcornea, anteverted, and simplex pinnae, and dental abnormalities also known as cataract–oto–dental syndrome. The female carriers have punctate opacities aligning with the posterior Y-suture along with small corneal diameter and presenile cataract.[23]

Down's syndrome

Cataract is a common finding in children affected with Down's syndrome, with the incidence ranging from 6% to as high as 50%. They may have other ocular findings such as hyperopia, nystagmus, strabismus, or entropion of eyelids.[24,25]

Lowe syndrome

It is an X-linked multisystem disorder with a classical triad of congenital cataract, mental retardation, and proximal renal tubular dysfunction. In addition, they can have muscle dystonia and debilitating arthropathy. Lifespan rarely exceeds 40 years in such patients.[18]

Metabolic cataract

Galactosemia

Galactosemia is due to mutations in galactokinase (*GALK1*), galactose-1-phosphate uridyltransferase, or uridine diphosphate 1–4 epimerase leading to abnormally high serum level of galactose. The galactitol accumulates and causes osmotic damage to the lens-"oil droplet cataract." It presents as nuclear cataracts but can also manifest as anterior or posterior subscapular cataract. These changes are initially reversible by eliminating galactose from the diet. Galactokinase deficiency causes milder cataracts. It is also associated with vomiting, failure to thrive, jaundice, and mental retardation.[26] Hypoglycemia and hypocalcemia are also associated with cataracts.

Toxoplasma, rubella, cytomegalovirus, herpes, and syphilis infections

Congenital infections such as toxoplasma, rubella, cytomegalovirus, herpes, and syphilis (TORCH) are associated with congenital cataract, with rubella being the most common. Other associated features include pigmentary retinopathy, microphthalmos, glaucoma, iris dystrophy, or chorioretinitis.[27] The incidence of TORCH infections is high in Indian subcontinent and up to 20% of cases may be seropositive.[28] Congenital rubella syndrome has a triad of cataract, sensorineural hearing loss, and heart disease, commonly patent ductus arteriosus. Most cases present with bilateral cataracts, but unilateral cataract may also occur with or without salt and pepper retinopathy [Fig. 1]. ELISA testing for IgM antibody toward a specific antigen of various infections shows high specificity.

Anterior-segment dysgenesis

Peter's anomaly generally presents with central corneal opacity. Peter's anomaly may have irido- or keratolenticular touch. Other ocular anomalies such as posterior embryotoxon microcornea or chorioretinal anomaly may be present.[29] Most cases are sporadic but some may be familial cases with autosomal-recessive inheritance. They may present with lens displaced in anterior chamber or may have anterior polar and nuclear cataracts[30] [Fig. 2].

Preexisting posterior capsular defect

Preexisting posterior capsular defect incidence varies from 2.2% to 6.75%.[31,32] It is seen in association with posterior polar cataract, persistent fetal vasculature (PFV), lenticonus, or lentiglobus. The defect develops due to the developmentally weak posterior capsule or traction of regressing hyaloid artery, resulting in the fluid vitreous hydrating the lens and egress of the lens material into the Berger's space. The preexisting posterior capsular defect presents as a total cataract or sometimes a differential opacity (more white at the center than in the periphery),

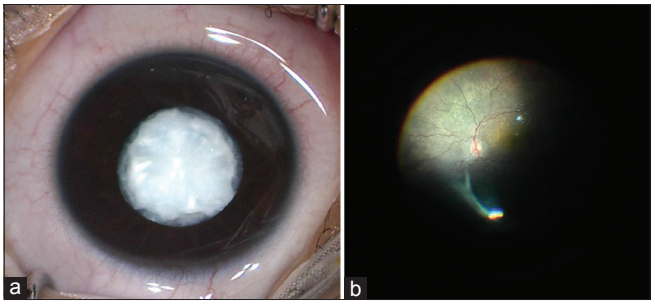

Figure 1: A 4-month-old child with congenital rubella syndrome presenting with (a) Membranous cataract. (b) Salt and pepper retinopathy

white dots on posterior capsule or anterior vitreous, and a fish-tail sign.[31] The diagnosis can be confirmed on ultrasound biomicroscopy[33] [Fig. 3].

Persistent fetal vasculature

PFV presents as eccentric posterior fibrovascular plaque, and hence initially was called "Persistent Hyperplastic Primary Vitreous." The presence of microphthalmos and elongated ciliary body is pathognomonic of PFV.[34] PFV sometimes may be associated with tunica vasculosa lentis, iridohyaloid blood vessels, or persistent pupillary membrane.[35] Anterior PFV generally have good visual prognosis in contrast to patients with posterior PFV in whom visual potential is often limited by coexisting retinal and optic nerve abnormalities[36] [Fig. 4].

Traumatic cataract

In India, the causes of traumatic cataract in children are different from that of Western population. Open-globe injury is three times more frequent than closed-globe injury, with bow and arrow injury being the most common causal agent.[37] The other causes are firecracker, ball, stone, wood, and metal injuries. Cataract is frequently associated with shallow anterior chamber, hyphema, corneal perforation/scarring, iris distortion, posterior synechiae, vitreous hemorrhage, vitreous in anterior chamber, and posterior capsular tear[38] [Fig. 5].

Uveitic cataract

Uveitic cataract in children may be associated with systemic conditions, most commonly juvenile idiopathic arthritis (JIA). JIA is associated with uveitis cataract (71%) due to severe intraocular inflammation.[39] These patients generally present with posterior subcapsular cataract along with posterior synechiae, iris bombe, and even peripheral anterior synechiae. The long-standing cases of JIA present with band-shaped keratopathy and hypotony.[40] JIA-associated uveitis patients have poorer prognosis as compared to non-JIA uveitis patients even after cataract surgery. Masquerade syndrome should always be ruled out in all children presenting with uveitis [Fig. 5].

History taking

The first complaint is often leukocoria, which the parent notices either total or gradual increase in the size of opacity. The second is child not following object close to face or not making eye contact (inability to recognize mother). The parent may also complain of squeezing of eye in bright light, squinting of eyes, small eyes (microphthalmos), large eyes (buphthalmos), and abnormal movement of the eyes (nystagmus). A detailed history is taken that includes asking about the age of onset and duration of symptoms.

Older children may present with difficulty in viewing distant objects, teacher may notice child not being able to read blackboard, or the parent may notice child bringing things very close to face and viewing television at close distance. A similar history in sibling or family member already diagnosed with cataract should be sought and a pedigree chart should be made. The history of fever and rash during pregnancy (TORCH),

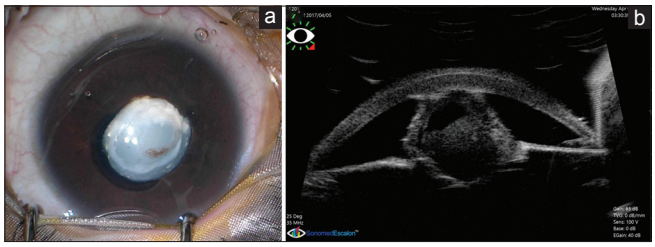

Figure 2: Anterior-segment dysgenesis. (a) A 4-month-old baby with Peter's type 2 anomaly with image showing central corneal opacity with cataractous lens displaced in the anterior chamber. (b) Ultrasound biomicroscopy of the same patient showing keratolenticular touch

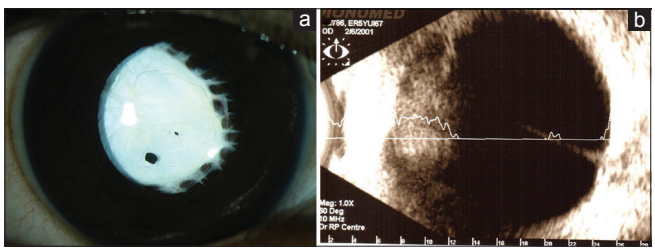

Figure 4: Persistent fetal vasculature. (a) Anterior persistent hyperplastic primary vitreous with prominent ciliary processes and vessels over the lens. (b) Posterior persistent hyperplastic primary vitreous with ultrasonography showing stalk of persistent hyperplastic primary vitreous

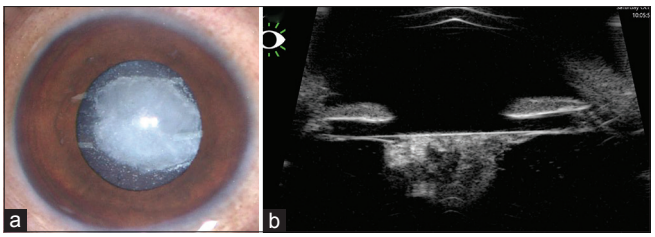

Figure 3: Preexisting posterior capsular defect. (a) Image showing posterior capsular plaque with multiple white dots in the surrounding areas. (b) Ultrasound biomicroscopy showing partially absorbed cataract with large posterior capsular defect

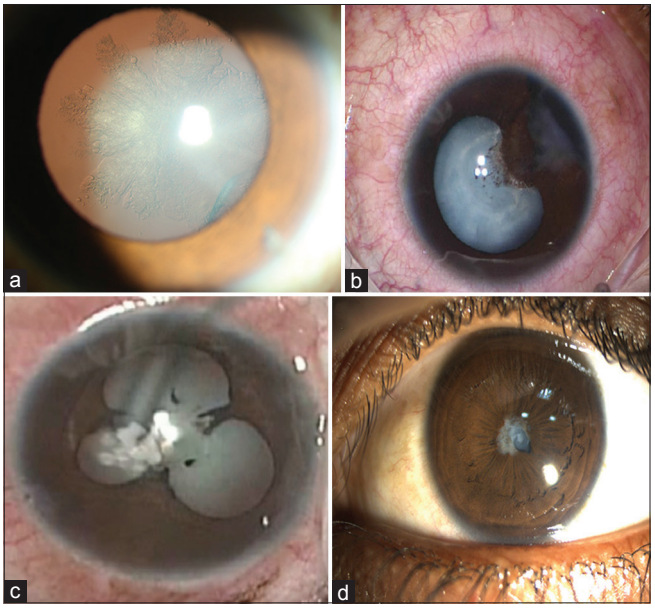

Figure 5: Traumatic and complicated cataracts. (a) Rosette cataract. (b) Traumatic cataract with posterior synechiae and iridodialysis. (c) Festooned pupil in a uveitic patient with complicated cataract. (d) Uveitic cataract with 360° posterior synechiae

consumption of any drugs or alcohol, and trauma in case of unilateral cataract should be looked for. The history of trauma during delivery, preterm delivery (retinopathy of prematurity), failure to thrive, and vomiting (galactosemia) should also be elicited. The delay in normal visual milestones should raise a high degree of suspicion in cases of hereditary cataract. In case of trauma, the mode of injury should be inquired which helps to ascertain the severity of injury.

Systemic examination

Children with cataract often present with systemic illness and syndromic features. Gross general examination may show dysmorphic features with sparse eyebrows, mongoloid slant (Down's syndrome), beaked nose with dental abnormalities (Hallermann–Streiff syndrome), frontal bossing (Lowe syndrome), low-set ears, and prominent parietal and occipital eminences; skin and hair changes such as dry scaly skin over the limbs, abdomen, and scalp with patchy alopecia; skeletal changes such as upper limb flexion deformity of a middle finger, the disproportionate length of upper and lower limbs, and calcaneus valgus deformity (Conradi–Hünermann syndrome), tall stature (Marfan's syndrome), camptodactyly (Beal's syndrome), short stature, and brachydactyly (Weill–Marchesani syndrome). The skull sutures are sometimes irregularly fused, the face shows puffiness (nephrotic syndrome), skin may show pigmentation (xeroderma pigmentosa), and absent eyebrows (Patau's syndrome). Head circumference measurement may show hydrocephalus or microcephaly, both are associated with cataract. Attention-deficit hyperactivity syndrome can be seen in William's syndrome. Subnormal mentation and retardation can be associated with many syndromes. Auscultation may reveal patent ductus arteriosus (rubella) or mitral valve prolapse (Ehler–Danlos syndrome).[18,24,26,27]

Ocular examination

Visual acuity assessment

The most challenging and difficult part is the visual acuity assessment. First, we look for fixation; central fixation suggests that the fovea is the fixing point, steady suggests that there is no component of nystagmus, and maintained suggests that there is no squint.[41] This only gives a gross but essential information regarding the visual acuity. Hence, specialized test has been developed to record the visual acuity in preverbal children. The test which can be used in infants is visual-evoked response, Catford drum, optokinetic nystagmus, and Teller's acuity cards. In children aged 1–2 years, Worth's ivory ball test, Boeck's candy test, the Screening Test for Young Children and Retards, and Cardiff's acuity test can be done. In children aged 2–3 years, miniature toy test, coin test, and LEA symbols® test can be done. In children aged 3–5 years, Allen's picture card, Lippman's HOTV test, and letter test can be done. In children aged more than 5 years, Tumbling E, Landolt's broken ring, Snellen's chart, and LogMAR chart can be used.[42]

Screening for cataract

The child is most comfortable in mother's lap. Hence, first the child is examined with child's head on the parent's shoulder. After assessing the visual acuity and pupillary response, distant direct examination is done to look for anterior segment abnormalities. The eye may show corneal opacity, shallow anterior chamber, peripheral anterior synechiae (Peter's anomaly), microcornea (microcornea-cataract syndrome), posterior synechiae (uveitis), key-hole pupil (iridofundal coloboma), and enlarged ciliary processes with vessels on lens (PFV).

The red reflex screening with direct ophthalmoscope kept at 30 cm and focused on each pupil separately (Bruckner's test) helps in the identification of lenticular opacity.[43] If in doubt, dilated pupil examination with homatropine 2% can be done. The visualization of both the eyes simultaneously with direct ophthalmoscope from 3 ft helps in identifying anisometropia based on the different glows. This also helps in identifying strabismus and asymmetric cataract and checking fixation pattern.[44] If the child is having poor fixation and is not following the object beyond 2 months of age, an urgent referral is indicated.

Refractive errors

The child at birth is mildly hyperopic. This hyperopia initially increases and then starts decreasing.[45] The refraction of a child under 5 years is essentially done under strong cycloplegia. The refractive error beyond 4 D of myopia, 5 D of hyperopia, and 2 D of astigmatism can lead to isometropic amblyopia.[46] Spherophakia and developmental cataract can present with progressively increasing myopia. Subluxation can present with high hyperopia if the lens is shifted away from the pupillary area and the child is essentially aphakic.

Nystagmus and head position

Nystagmus develops because the child cannot maintain fixation, rarely the child might acquire a head position that helps the child to get maximum vision with a minimum amplitude of nystagmus (null position). This sensory-type nystagmus develops by 13 weeks of age when cataract is left untreated.[47] The presence of nystagmus is not a contraindication for surgery as the child can still have a working vision if intervened early.

Screening of parent

The screening of parent is essential to rule out hereditary cataracts. Screening of mother can reveal a sutural cataract in a child of NHS with dense nuclear cataract.[48]

Pediatric Cataract Classification

Pediatric cataract can be classified based on onset, morphology [Table 1 and Fig. 6],[49] etiology, and laterality [Table 2]. All these components are included while describing a pediatric cataract. Congenital cataract presents at birth but due to delayed diagnosis and presentation can often be mislabeled, hence morphology of anterior polar, central fetal nuclear, posterior polar, PFV, and sutural cataract confirms to us of the congenital nature. Developmental cataract occurs from infancy to adolescence and hence typically affects a zone involving either infantile or adolescent nucleus or both and sometimes cortical component as well.

Investigations

Ultrasonography (USG) not only helps in intraoperative management, but also in prognosticating a case, especially in cases with unilateral cataract. USG helps us rule out retinal detachment, fundal coloboma, and retinoblastoma.[33] USG in a case of unilateral cataract can diagnose a PFV.[50] Magnetic resonance imaging can be ordered if there is a high degree of suspicion of PFV which is missed on USG. Color Doppler

Table 1: Morphology of cataract

Whole lens	Central	Anterior	Posterior	Miscellaneous
Total	Lamellar	Anterior polar	Mittendorf dots	Punctate lens opacities
Congenital Morgagnian	Central pulverulent	Dot like	Posterior cortical	Sutural
Membranous	Ant egg	Plaque like	Posterior subcapsular	Coralliform
	Nuclear	Anterior pyramidal	Posterior lenticonus	Wedge shaped
	Oil droplet	Anterior subcapsular		Persistent hyperplastic primary vitreous
	Cortical	Anterior lenticonus		
	Coronary			

Table 2: Bilateral and unilateral cataracts

Bilateral cataracts	Unilateral cataracts
Idiopathic	Idiopathic
Hereditary cataracts (autosomal dominant)	Ocular anomalies
Genetic and metabolic diseases	PFV
Down syndrome	Anterior segment dysgenesis
Lowe syndrome	Posterior lenticonus
Galactosemia	Posterior pole tumors
Marfan's syndrome	Traumatic (rule out child abuse)
Trisomy 13-15	Rubella (rarely)
Hypoglycemia	Asymmetric bilateral cataract
Alport syndrome	
Myotonic dystrophy	
Fabry disease	
Hypoparathyroidism	
Maternal infection	
Rubella	
Cytomegalovirus	
Varicella	
Syphilis	
Toxoplasmosis	
Ocular anomalies	
Aniridia	
Anterior segment dysgenesis syndrome	
Corticosteroids	

PFV: Persistent fetal vasculature

also shows flow picture in case of a PFV. X-ray is done in cases of traumatic cataract and computed tomography scan to localize an intraocular foreign body.[51] TORCH profile in case of bilateral congenital cataract can be ordered to look for active infection.[52] Echocardiography in cases such as Marfan's syndrome and rubella may reveal aortic regurgitation, aortic root dilation, patent ductus arteriosus, and atrial septal defect. In cases of JIA and juvenile rheumatoid arthritis needing immunosuppressant medications, a rheumatologist's opinion is sought. A neurologist should be consulted in cases of seizure and gross developmental delay. Nephrology evaluation should be done in cases of nephrotic syndrome and Lowe syndrome.[53]

Prognosis

Congenital cataract has a good prognosis if identified early and surgery is done before 6 weeks. Delayed presentation in adulthood is also being frequently noted because of delayed diagnosis, ignorance, and lack of facilities.[54] The developmental cataract has a better prognosis than congenital cataract.[55] Homocystinuria can be managed using methionine-restricted diet.[56] Factors that may influence the surgical outcome adversely include presence of a unilateral cataract, presence of nystagmus, strabismus, or any ocular defects such as microphthalmos and PFV.[57]

Timing of surgery

The critical period of eye development ranges from 2 months to 6 months of age and emmetropization of an eye is generally achieved by 9 years of age, but the plasticity of the brain can go well beyond the first decade of life.[58] Surgery is advised in visually significant opacities, i.e. more than 3-mm central opacity. Unilateral cataracts should be operated as early as possible before 6 weeks of age and bilateral cataracts should be operated before 8 weeks of age.[59] In symmetric cataracts, the second eye is operated within 1 week of the first eye and in systemically unstable child it can be performed in the same sitting.[60] In visually nonsignificant opacities (blue-dot cataract or small posterior polar), the child can be observed at regular intervals.

Anesthesia

General anesthesia with laryngeal mask airway or endotracheal tube can be used for surgery. Opioids are associated with adverse effects such as vomiting and respiratory depression. The subtenon block leads to a reduced requirement for postoperative analgesia, prolonged time to first rescue analgesia, reduced incidence of oculo-cardiac reflex, and better parent satisfaction scores. The subtenon block and topical lignocaine are safe alternatives to intravenous fentanyl for perioperative analgesia.[61]

Preoperative Evaluation

Examination under anesthesia

Examination under anesthesia is carried out to record the essential parameters for intraocular lens (IOL) power calculation. The immaturity of an infant's organ system and thermoregulation put them at greater risk from general anesthesia. The most life-threatening risk to an infant is postoperative apnea. The child preferably is taken up for surgery after the 1st month of birth or 44 weeks of gestational age in preterm infants as they are prone to apnea in postoperative period.[62]

Biometry

Axial length

Predicting axial length (AL) growth and hence the myopic shift is difficult after pediatric cataract surgery.[63] AL increases rapidly in the first 6 months (0.46 mm/month), then has a relatively slower (infantile phase) growth (0.15 mm/month) till 18 months, followed by a slow (juvenile phase) growth (0.10 mm/month).[64] The absolute error in children is higher compared to adult population.[65] AL measurement is better estimated with immersion A-scan than indentation A-scan.[66] In spite of this disadvantage, indentation method is more commonly used than immersion AL (82.4% vs. 17.6%). Hence, A-scan reading with maximum anterior chamber depth should be taken.[66]

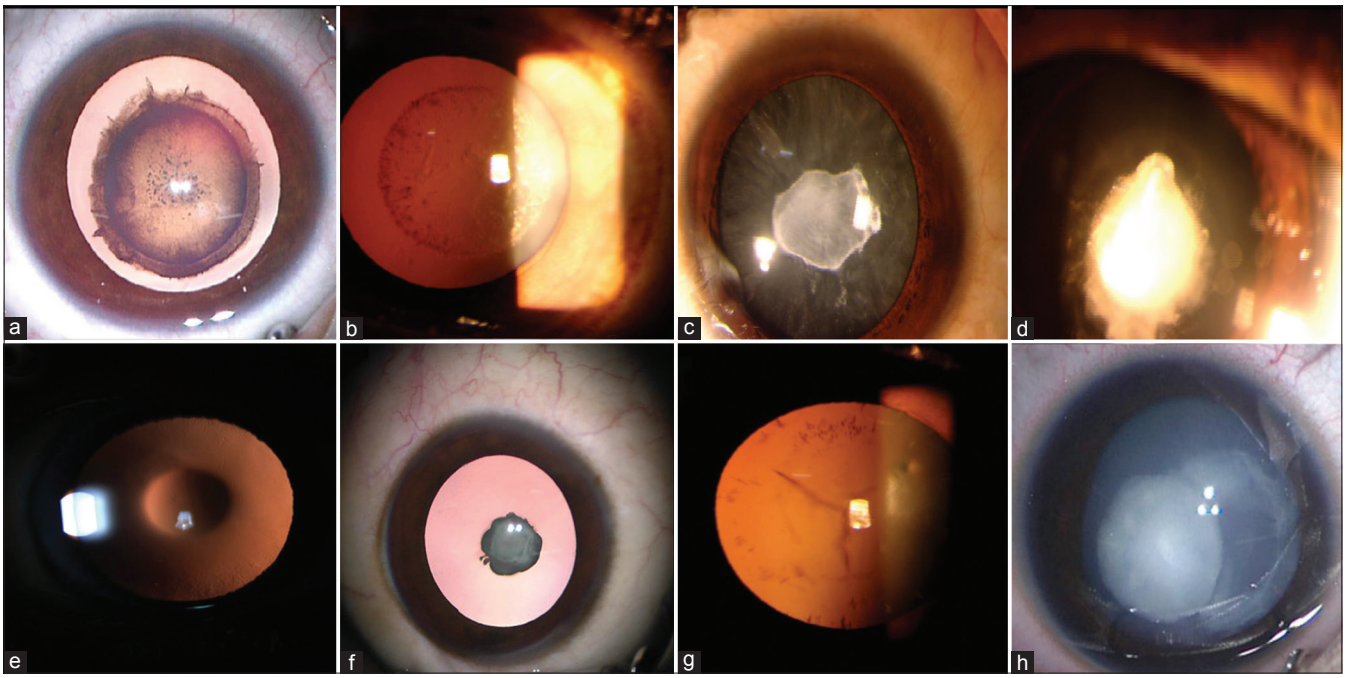

Figure 6: Morphology of pediatric cataract. (a) Zonular cataract with riders. (b) Cataract pulverulenta. (c). Anterior polar cataract (plaque type). (d) Anterior polar cataract (pyramidal type). (e) Posterior lenticonus showing oil droplet sign. (f) Posterior polar cataract. (g) Sutural cataract. (h) Morgagnian cataract

Keratometry

Keratometry values are obtained using auto keratometer. Keratometry also steeply reduces in the first 6 months (−0.40 D/month), −0.14 D/month in the next 6 months, and − 0.08 D/month in the second year, reaching the adult range at about 3 years of age.[64] Keratometry readings without speculum are preferred.[67]

Optical biometry can also be used in older children.

Intraocular lens power calculation

IOL power calculation is multifactorial. The eye is dynamic in nature with constant change in children. Hence, predicting the right power for the child's eye is difficult and often confusing. There is a trend toward implantation of IOL in young children and infants, especially in the developing countries where postoperative care and follow-up are difficult.[68] The IOL power depends on various factors which include the age of presentation, morphology of cataract, visual acuity at presentation, time of development of cataract (congenital/developmental), biometry at presentation, unilateral or bilateral cataract, and refractive status of the fellow eye.[69] IOL can be implanted in eyes with AL >17 mm and corneal diameter >10 mm. The initial desired refractive outcome after IOL implantation is moderate hypermetropia to avoid undesired myopic shift in adulthood. Dahan and Drusedau suggested undercorrection of 20% in children <2 years and 10% in children between 2 and 8 years.[70] Prost suggested 20% undercorrection between 1 and 2 years of age, 15% undercorrection between 2 and 4 years, and 10% between 4 and 8 years of age.[71] Enyedi suggested postoperative target refraction to be used for IOL power calculation according to age (age + postoperative refraction = 7).[72] Intraoperative aphakic refraction or aberrometry can be used to calculate the IOL power.[73] SRK/T and the Holladay 2 formulae have been shown to have the least predictive error.[74]

Ultrasound biomicroscopy

Ultrasound biomicroscopy is a noninvasive imaging technique that helps in identifying anterior PFV, posterior capsular defect, and posterior polar cataract preoperatively.[75] It also helps us in post-traumatic cases to look for cyclodialysis, subluxation, foreign body localization in anterior segment, and rupture of posterior capsule.[76]

Choice of intraocular lens

Hydrophobic acrylic lenses with square edges inhibits lens epithelial cell (LEC) migration and posterior capsular opacification formation and are hence preferred in pediatric age group.[77] The "Perfect" IOL would have a hydrophilic anterior surface and a hydrophobic posterior surface.[78] Multifocal IOLs may help in establishing stereopsis in unilateral cases, but the contrast and brightness of the images get compromised.[79] There may be significant myopic shift and subsequent refractive error, hence they are more suitable to teenagers.

Surgery

The main aim of treatment is to clear the visual axis followed by postoperative visual rehabilitation. Surgery in children differs from that of adults as they have lower corneal and scleral rigidity, very elastic anterior capsule, soft lens, and well-formed vitreous. Superior incision is preferred which allows wound to be protected by eye lid and by Bell's phenomenon, in trauma-prone childhood years. Either scleral or clear corneal incision can be used, and it has been shown to be insignificant with respect to astigmatism caused by the incision.[80] Two side ports 180° apart gives 360° movement. Preservative-free adrenaline (1:100,000) is injected for pupillary dilation and Trypan blue dye (0.06%) under air stains the capsule. A cystitome is used to give nick on the anterior lens capsule. The anterior capsulorhexis is completed with the help of utrata or

intravitreal 23G forceps through 2.2-mm incision thereafter. Two-incision push–pull technique has been shown to produce consistent size of capsulorhexis and reduced chances of run off.[81] Multiquadrant hydrodissection (at least three quadrants) is the preferred method in pediatric group, following which bimanual lens aspiration is completed to remove the lens matter. This is followed by partially underfilling the anterior chamber with high-viscosity, cohesive viscoelastic substance. The high incidence of visual axis opacification (VAO) necessitates primary management of the posterior capsule. The cystitome is used to give nick over the posterior capsule sideways and posterior capsulorhexis is completed using intravitreal forceps.[82] Anterior vitrectomy breaks the scaffold for proliferating LECs and metaplastic pigment cells, thus preventing VAO formation. Posterior capsulotomy is must for all patients <6 years of age. Vitrectomy can be deferred after 5 years of age. In children older than 8 years (with mental retardation or with nystagmus) who may not cooperate for laser capsulotomy on a slit lamp, posterior capsulorhexis is mainly required.[41] Inserting the IOL in the bag when posterior capsulorhexis is already made is difficult. After making a 2.75-mm entry wound, IOL is inserted by pushing the leading haptic against the back surface of anterior capsule and then pushing down the trailing haptic followed by tucking of the trailing haptic into the bag. This is a safe method and results in no complications related to faulty IOL implantation.[83] The capsulorhexis can be performed using vitrectomy cutter, but the tensile strength is less than curvilinear capsulorhexis.

In cases of children presenting with subluxation or anterior dislocation, intralenticular aspiration of a lens with anterior-chamber IOL and peripheral iridectomy is preferred.[84] Iris and scleral fixations of IOL are emerging as the methods of aphakic correction.[85] In traumatic cataracts, posterior capsulorhexis with posterior capture of optic is preferred to avoid VAO.[86] Optic capture can also be helpful in cases of capsulorhexis run off. Peripheral iridectomy should be done in aphakic patients. Peripheral iridectomy can also be considered in uveitic and traumatic cataracts depending on the presentation. In children, the surgical incisions need to be sutured using 10-0 monofilament nylon because of the increased risk of anterior-chamber collapse and endophthalmitis. Side ports can be left sutureless if there is no leak.

Postoperative treatment

Pediatric eyes due to immaturity of the blood–aqueous barrier have more intraocular reaction, which includes anterior-chamber cells, flare, fibrinous reaction, pupillary membrane formation, and posterior synechiae formation. Patients are prescribed topical steroids (prednisolone acetate 1% six times/day) and cycloplegics (preferably homatropine 2%) postoperatively. Heparin surface-coated IOL in uveitic cases and subconjunctival injection of dexamethasone with or without triamcinolone are known to reduce postoperative inflammation.[87] Postoperative single injection of hydrocortisone 5 mg/kg and dexamethasone 0.1 mg/kg has been shown to be equally effective, without increase in intraocular pressure seen with depot steroid and hyphema seen with heparin. Difluprednate 0.05% four times a day has shown safety and efficacy profiles like prednisolone acetate 1% in children aged 0–3 years undergoing cataract surgery.[88] Topical steroids should be tapered over 6–8 weeks. Patients are followed up 1 month after surgery and every 3 months thereafter.

Visual rehabilitation and amblyopia therapy

Surgery is immediately followed by prescription of glasses and amblyopia therapy even before removal of suture. The suture removal and repeat refraction is done at 1 month postoperatively and again at 3 months. The parent must ensure that the child wears the prescribed glasses and occlusion therapy (for unilateral or asymmetrical cases). Contact lenses are more suitable in children with unilateral aphakia as spectacles cause aniseikonia. According to the Infant Aphakia Treatment Study, primary IOL implantation and contact lens did not show a significant difference in terms of final visual acuity in infants <7 months of age. The children who had IOL implantation had more adverse events and required more reoperations to clear visual-axis opacities. Hence, the use of contact lens is a good alternate to IOL implantation in such children.[89]

In younger children, glasses are prescribed for near. In schoolgoing children, executive bifocal glasses with near add are prescribed after correcting for distance. In children <12 months of age, occlusion is given where both eyes are occluded depending on age (6-month-old child is given occlusion half of the waking hours per day in alternate eyes). A child aged >1 year is given occlusion in the better eye more than worse eye (3-year-old child is given occlusion 8 waking hours per day for 3 days in better eye and 1 day in the worse eye).[90] The worse eye is occluded to avoid development of occlusion amblyopia in the better eye. Patients are followed up every 4–6 weeks initially and every 3 months thereafter to see for response of treatment. Occlusion is gradually tapered off once the desired effect is achieved. Levodopa, carbidopa, and citicoline have shown good results in modulating the plasticity.[91]

Complications

Glaucoma following cataract surgery is observed in 10%–25% of children. Younger age at surgery and presence of microphthalmia are high-risk factors.[92] Intraocular pressure may be controlled on medication or may require surgical intervention. VAO is the most common complication after pediatric cataract surgery with or without IOL surgery and may cause visual deprivation amblyopia.[93] In a thick VAO, posterior capsulotomy combined with anterior vitrectomy is required [Fig. 7]. Postoperative reaction is seen more commonly in preexisting complicated and traumatic cataract. This can result in posterior synechia formation, leading to seclusio pupillae, iris bombe, and subsequent secondary angle-closure glaucoma and VAO. Improvement in surgical techniques, frequent use of topical steroids, and cycloplegics in the postoperative period have significantly decreased the complications.[94] Posterior optic capture and in-the-bag IOL fixation technique decrease the incidence of anterior optic capture. Retinal detachment is rare but more common in eyes with PFV.[95]

Conclusion

Pediatric cataract surgery has evolved over the years and has become more safe and predictable. Early identification, immediate referral, and appropriate management have favorable outcomes.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have

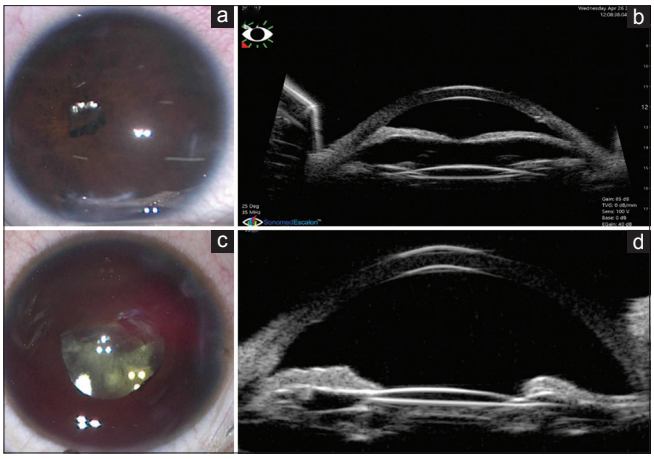

Figure 7: Visual axis opacification. (a) A 6-month-old child with 1-mm pupil with pupillary membrane. (b) Ultrasound biomicroscopy showing membrane only anterior to intraocular lens which was tackled with anterior route surgery. (c) A 2-year-old child with thick posterior capsular opacification. (d) Ultrasound biomicroscopy showing thick membrane behind the intraocular lens which was removed using pars plana membranectomy

given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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