Congenital Glaucoma and Anterior Segment Dysgenesis

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Childhood cataract may occur in association with ocular comorbidities involving cornea, angle structures, and/or iris. These abnormalities may include congenital glaucoma and anterior segment dysgenesis. These associated abnormalities may alter presentation and management of these conditions. In this chapter, we will discuss these two entities in separate section. They have been clubbed together since anterior segment dysgenesis is associated with glaucoma in significant number of cases and few cases of presumed primary congenital glaucoma may actually be secondary to anterior segment dysgenesis.

15.1 Childhood Glaucoma

- Introduction
- Clinical presentation
- Investigations
- Surgical outcomes

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15.1.1 Introduction

Primary congenital glaucoma (PCG) is an anomaly affecting anterior chamber angle which leads to obstruction of aqueous outflow, increased intraocular pressure (IOP) and optic nerve damage [1]. Buphthalmos is an alternate term used to describe congenital glaucoma. Buphthalmos means "bull's eye." It is used to describe visible enlargement of the eyeball and clouding of cornea at birth or early childhood due to uncontrolled glaucoma [2]. High intraocular pressure (IOP) causes increase in axial length and corneal dimensions of the eye, leading to axial myopia, stretched limbus, corneal thinning and visibly enlarged eyeballs. The common causes of buphthalmos include primary congenital glaucoma (PCG), Sturge-Weber syndrome, neurofibromatosis, and aniridia [2].

Incidence of PCG is one in every 10,000–15,000 live births which accounts for 0.01–0.04% of total blindness [3]. It is bilateral in up to 80% of cases and two-third of cases are males. Most cases are sporadic (90%) [4]. However, in the remaining 10%, there appears to be a strong familial component.

15.1.2 Clinical Presentation

PCG patients may present with corneal haze, enlarged eye size, or abnormal eye move-

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ments. Vision loss in these eyes may occur secondary to uncorrected refractive error, corneal opacity, optic nerve damage, amblyopia, and cataracts. Cataract may be congenital/developmental or as a sequalae to glaucoma filtration surgery. Examination findings may be variable.

- Progressive myopia and high astigmatism may be seen.
- Cornea may be hazy and presence of Haab striae is a common finding (Fig. 15.1).
- Cataract may be anterior or posterior subcapsular cataract, total or less commonly cortical or zonular cataract (Fig. 15.2). Cataract may be primary or more commonly secondary to trabeculectomy in 6–58% cases [5–7]. Zonular weakness may be present.
- Bleb in post trabeculectomy eyes with buphthalmos may be thin cystic and rarely associated with other complications (Fig. 15.3).
- Fundus examination may reveal advanced cupping. The cupping may be reversible in small children. Features associated with pathological myopia may be seen.

15.1.3 Investigations

In addition to usual investigations, Ultrasound biomicroscopy (UBM) can be used for measurement of angle to angle and bag diameter and aid in planning of surgery (Fig. 15.4). UBM also helps to assess anterior segment structures, anterior chamber depth (ACD), angle anomalies, abnormal iris insertion, helps assess sulcus-to-sulcus measurement and identifies lax zonules and posterior capsular defect preoperatively [8].

15.1.3.1 Management of Cataract in Buphthalmic Eyes

Apart from the routine challenges encountered, there are additional surgical difficulties faced by surgeon while operating cataract in buphthalmic eyes. These include the following:

- **Corneal haze** may cause difficulty in visualization during surgery (Fig. 15.5). IOP should be controlled before surgery. We recommend use of dye for staining of anterior capsule in such cases. Posterior capsule may also require staining if visibility is low.

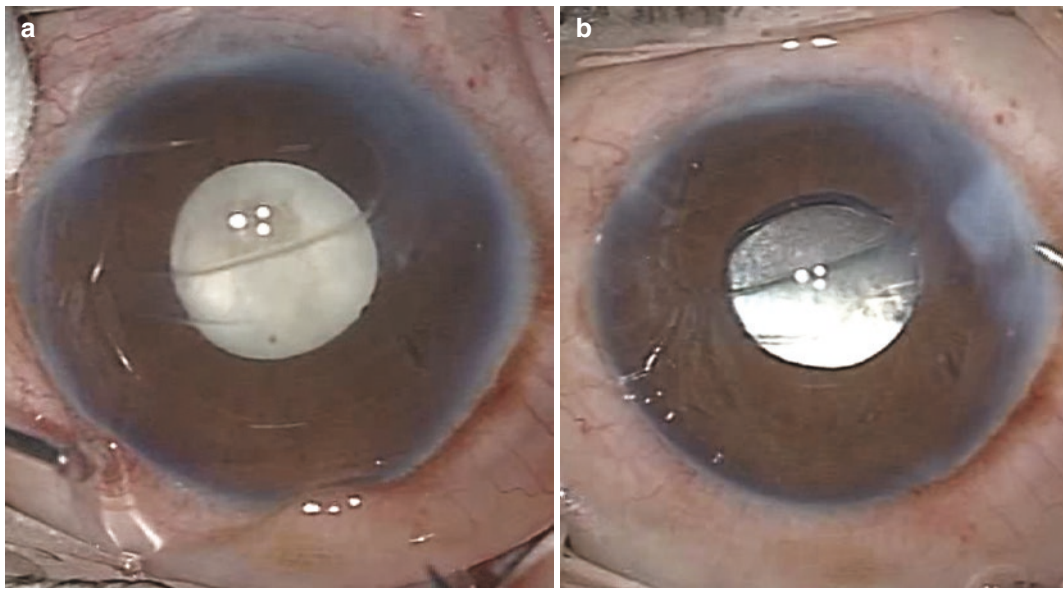

Fig. 15.1 Congenital glaucoma with Haab striae and total cataract. (**a**) Preoperative picture where large superior and multiple peripheral Haab striae. (**b**) Postoperative picture with IOL in situ and more pronounced Haab striae

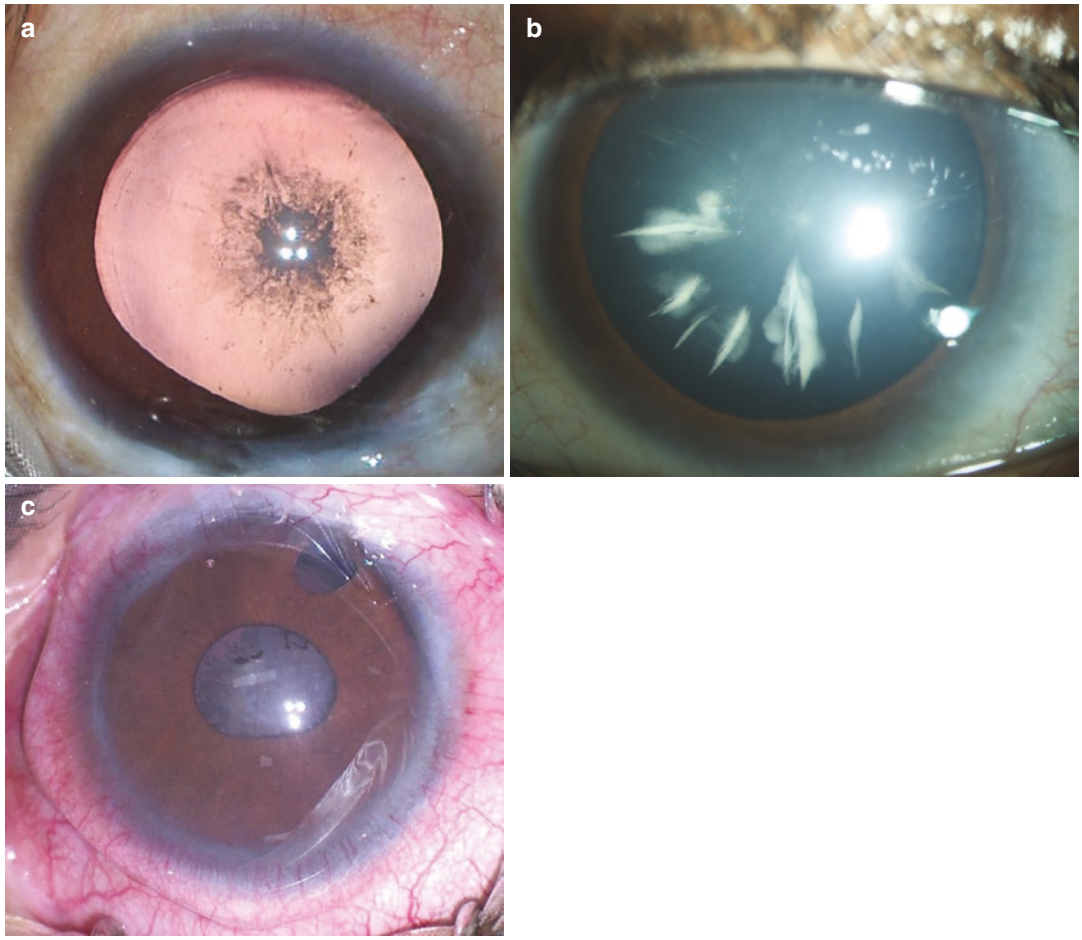

Fig. 15.2 Morphology in cataract with childhood glaucoma post trabeculectomy. (**a**) Posterior subcapsular cataract. (**b**) Cortical cataract. (**c**) Diffuse cataract with large superonasal peripheral iridotomy

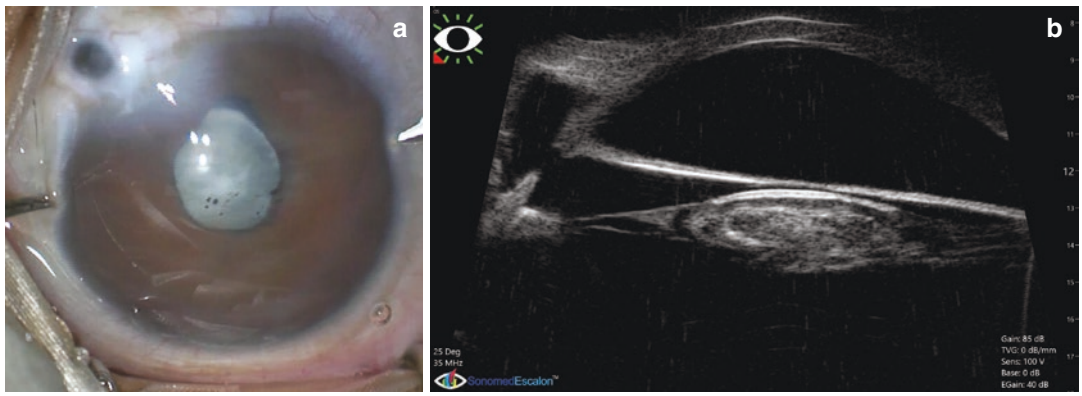

Fig. 15.3 Post trabeculectomy thin cystic bleb with iris prolapse from the ostium and total cataract. (**a**) Clinical picture. (**b**) Ultrasound biomicroscopy of the same showing patent ostium and elevated bleb

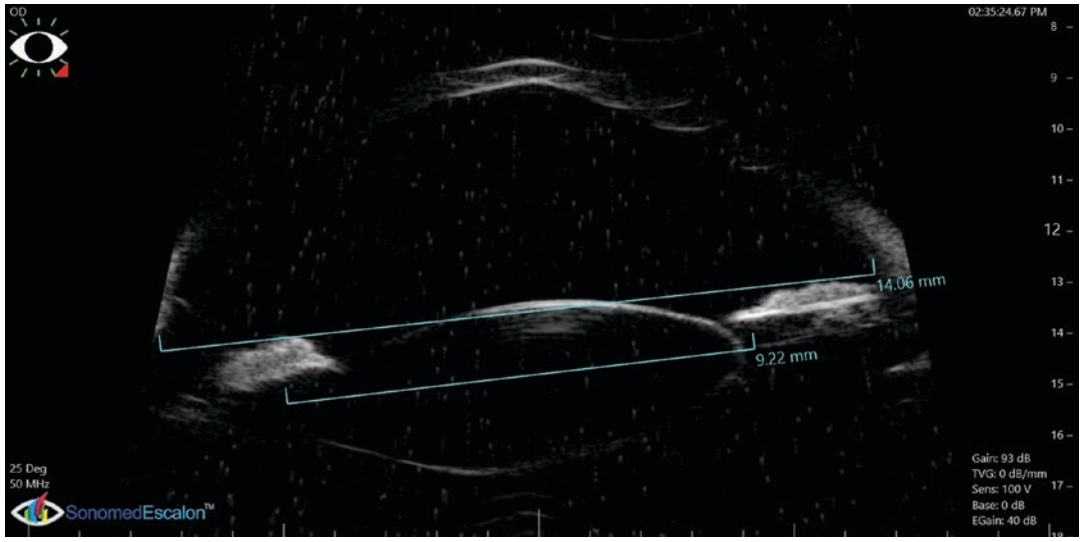

Fig. 15.4 Ultrasound biomicroscopy showing dimensions of buphthalmic eye. Complete white to white examination is not possible in single view. (**a**) Angle to angle distance, (**b**) bag diameter

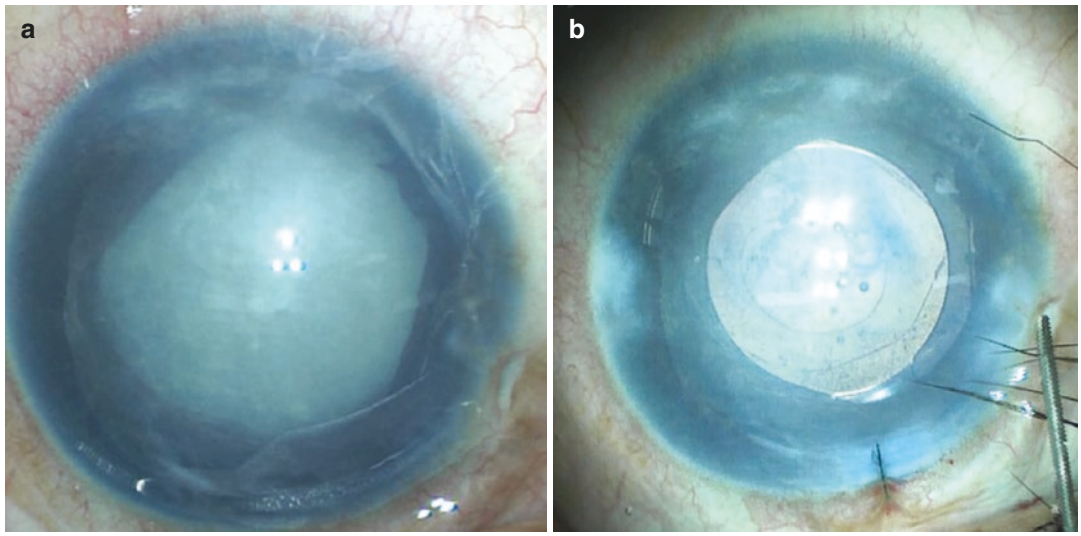

Fig. 15.5 Six-year-old child with congenital glaucoma with corneal haze with atrophic iris. (**a**) Preoperative picture with total cataract. (**b**) Postoperative picture with circular anterior and posterior capsulorhexis and IOL in bag

- **Very deep anterior chamber (AC)** may result in difficult instrumentation. Intraoperatively, anterior chamber depth may show frequent fluctuations due to low scleral rigidity. We use microincision forceps with longer arm compared to utrata's forceps for capsulorhexis. Use of bimanual irrigation and aspiration provides better control to the situation.
- **Zonular weakness:** Phacodonesis, lax lens zonules, liquefied vitreous and, thus, a weak posterior capsular support can lead to inadvertent complications. There is increased risk of vitreous loss in these patients.

- **Wound apposition**: Buphthalmic eyes have thin sclera and cornea. Hydration alone does not provide sufficient wound closure in most cases. We recommend suturing of main wound as well as side port to prevent postoperative shallow AC.
- **IOL power calculation** remains difficult. Post-trabeculectomy buphthalmic eyes have a shift towards with-the rule astigmatism [9, 10]. As most of these eyes are high myopic, IOL power calculation should be done using appropriate IOL formulae e.g., SRK-T for axial lengths >24.5 mm. Many a times, no single IOL power formulae might be able to predict the correct emmetropic power of implant for buphthalmic eyes. Parents should be counseled preoperatively, regarding use of spectacles for distance and near vision.
- Large eye size and bag dimensions that may lead to postoperative intraocular lens (IOL) decentration [11, 12]. In the bag IOL placement with posterior capsulorhexis has been reported to be associated with decentration. The technique of IOL implantation in the sulcus with optic capture with anterior or both anterior and posterior capsule (i.e., optic in bag and haptic in sulcus) may provide satisfactory anatomical outcome (Fig. 15.6) [12].

15.1.4 Surgical Outcomes

Temporary cessation of ocular growth is reported after adequate IOP control in eyes with AL > 22 mm and in children aged 3 months or older [13].

Our experience with 31 eyes of primary congenital glaucoma (post trabeculectomy) with visually significant cataract undergoing lens aspiration surgery showed a mean best corrected visual acuity of 6/60 (Snellen's) at 1 year postoperatively. Reasonably predictable refractive results were obtained in these eyes, provided glaucoma was well controlled [14].

Thus, besides control of IOP, visual rehabilitation of buphthalmic eyes may involve appropriate management for amblyopia, keratoplasty for corneal opacity in addition to timely cataract surgery for visually significant cataract. Buphthalmic eyes undergoing cataract surgery can achieve successful refractive and visual outcomes if careful preoperative planning is carried out regarding the choice of IOL type and IOL power, taking into consideration the adequacy of intraocular pressure control, accurate biometry, assessment of bag size and use of appropriate IOL power formulae.

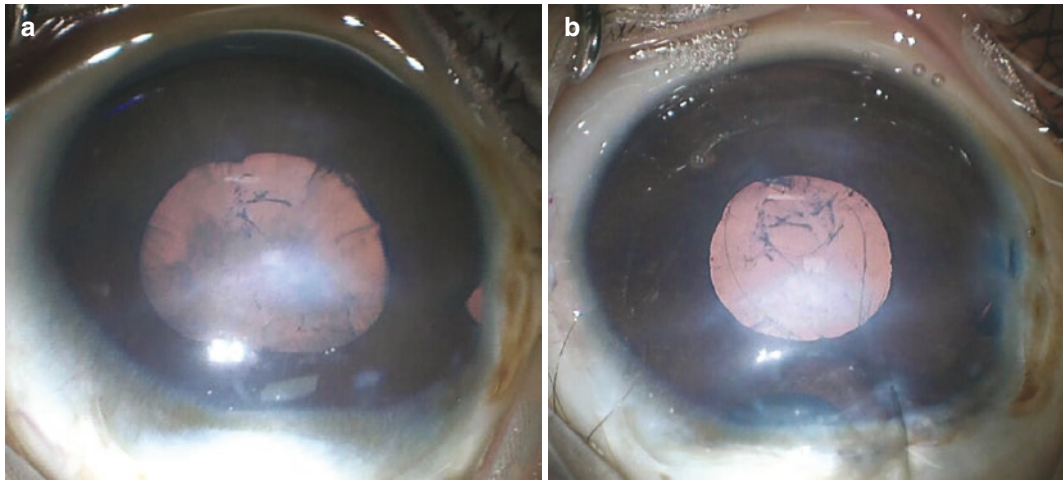

Fig. 15.6 Fifteen-month-old child with congenital glaucoma with corneal haze with Haab striae and large cornea (buphthalmos). (**a**) Preoperative picture with Anterior and posterior subcapsular cataract. (**b**) Postoperative picture with IOL in sulcus with optic capture with anterior and posterior capsulorhexis (bag complex) for better centration

15.2 Anterior Segment Dysgenesis

Anterior segment dysgenesis (ASD) is a group of disorders arising from abnormal development in cornea, iris, lens and angle structures. This includes Axenfeld's anomaly, Rieger's anomaly, Axenfeld-Rieger syndrome (ARS), Peters anomaly, sclerocornea, aniridia, posterior keratoconus, and iridogoniodysgenesis. It occurs due to abnormalities in neural crest differentiation and migration. Various classifications are used for describing ASD depending either on their clinical features or area of involvement [15–17]. Lens abnormalities are not uncommon in cases with ASD. Townsend, Font, and Zimmerman have classified ASD based on involvement as Descemet layer defect alone or associated with lens abnormalities or with iris stromal abnormalities. This involvement of lens suggested the effects of primary mesenchymal defect on the development of lens [17].

15.2.1 Embryology

The surface ectoderm invaginates and forms lens vesicle in the embryonic cup at sixth week of gestation. Then, neural-crest-derived tissue migrates in three waves beneath this surface ectoderm. The surface ectoderm forms the corneal epithelium. The three waves forms endothelium, corneal stroma, and iris stroma. Any arrest in the development of these layers may affect further development of anterior chamber leading to different presentations of ASD [18].

15.2.2 Genetics

Many genes are involved in the ASD with variable degree of penetrance. Forty percent cases occur due to involvement of PITX2 (4q25) and FOXC1 (6p25). Typically, PITX2 disruption is associated with ARS with ocular and dental abnormalities, and FOXC1 is associated with ARS with hearing or cardiac abnormalities. Others associated with ARS include PAX6 (11p13) and FOXO1A (13q14) [19, 20]. ARS has autosomal dominant inheritance pattern in 70% cases. In Peter's anomaly, rare cases have been attributed to PITX2, FOXC1, and PAX6 mutations, but the majority of cases are sporadic [21–23].

15.2.3 Clinical Features

- Axenfeld-Rieger syndrome
- Axenfeld anomaly presents as posterior embryotoxon (Fig. 15.7) (anteriorly displaced

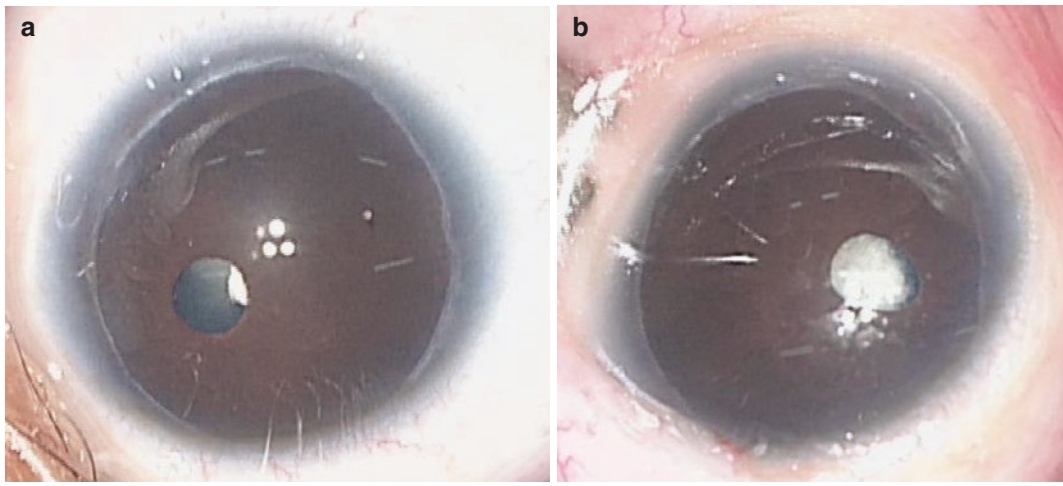

Fig. 15.7 Mild variant of Axenfield-Rieger syndrome (**a**, **b**) Posterior embryotoxon in 9-month-old child with cataract in right and left eye, respectively. Also notice presence of corectopia in both eyes

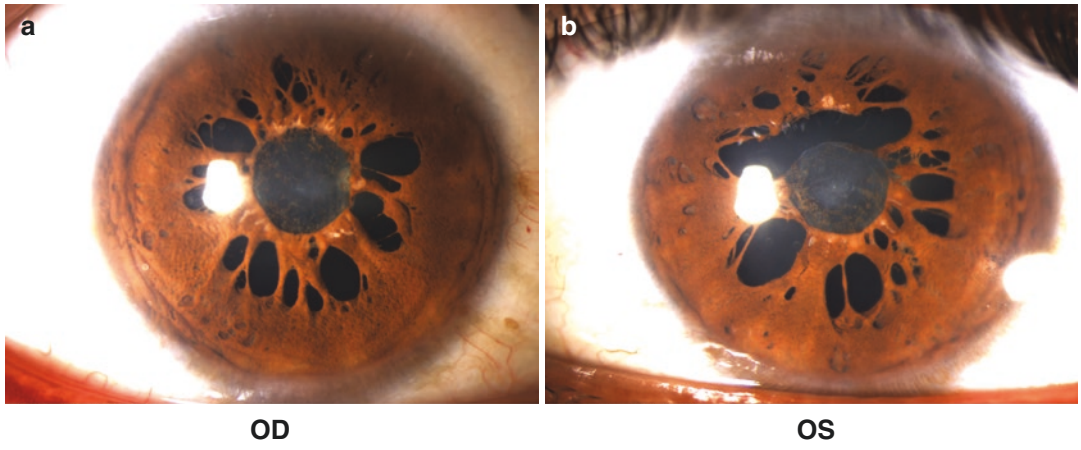

Fig. 15.8 (**a**, **b**) Rieger Anomaly-polycoria and iris atrophy in 7-year-old girl in right and left eye respectively

Schwalbe line) and iris strands adhered to the anteriorly displaced Schwalbe line. Rieger anomaly includes posterior embryotoxon, pseudopolycoria, and iris atrophy (Fig. 15.8) while Rieger syndrome is Rieger anomaly along with systemic findings including facial bone defects, hypertelorism, telecanthus, maxillary hypoplasia, dental abnormalities (microdontia and hypodontia), umbilical abnormalities or pituitary involvement. Thus, they are now considered as a spectrum of disorder termed as Axenfeld-Rieger syndrome (Fig. 15.9). It may vary from subtle changes in the angle to severe ocular changes. Systemic involvement may also include cardiac and endocrine system. Fifty percent cases with ARS are associated with glaucoma [24, 25].

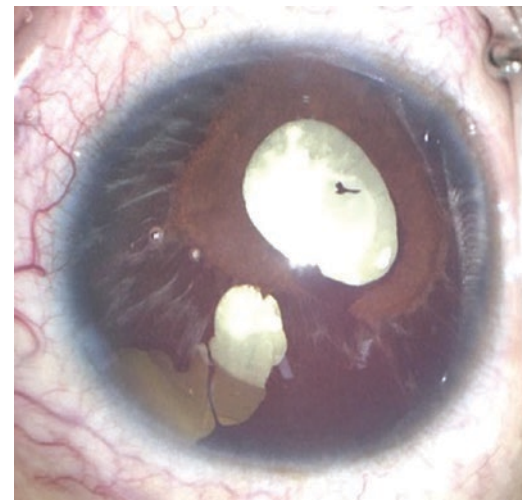

Fig. 15.9 Severe variant of Axenfield-Rieger syndrome—posterior embryotoxon with corectopia, iris atrophy and polycoria along with total cataract

- Peters anomaly
- Peter syndrome is characterized by a shallow anterior chamber, synechiae between iris and cornea and central corneal opacity. It occurs due to defect in endothelium, Descemet membrane and posterior stroma due to the defect in the migration of the neural crest cells. This syndrome can vary in severity with ocular findings ranging from unilateral mild central corneal opacity to severe bilateral microphthalmia, corneal opacification, cataract, and glaucoma. Eighty percent of cases have bilateral presentation. The Peters anomaly has been further divided into type I and type II. Type I Peters anomaly is categorized by central corneal opacity and iridocorneal adhesions (Fig. 15.10). Type II Peters anomaly has a more severe phenotype with corneal opacity and lens involvement with iridocorneal touch with or without cataract (Fig. 15.11). The Peters plus syndrome includes the anterior segment findings with systemic developmental anomalies. These include craniofacial dysmorphism, cleft lip/palate,

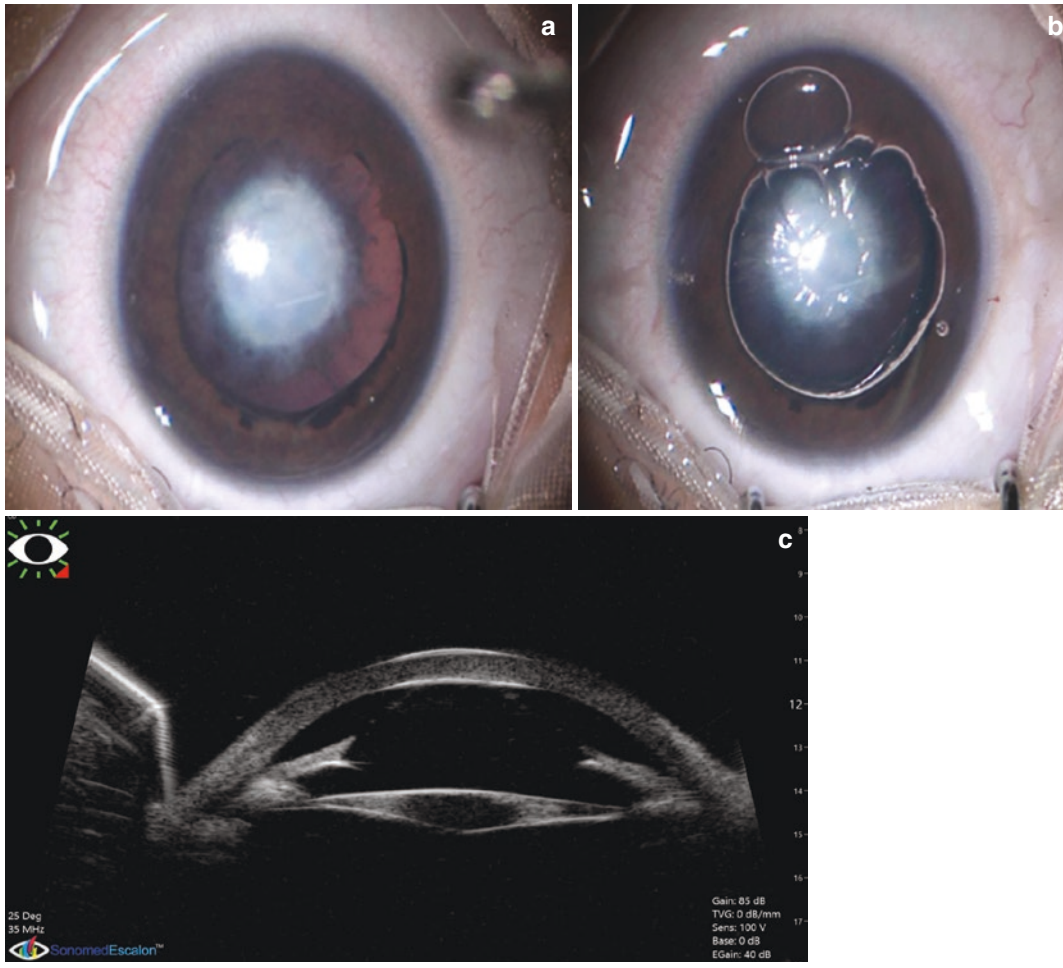

Fig. 15.10 Peter's anomaly type 1 in 2-month-old child. (**a**) Small corneal opacity with iridocorneal adhesions with cataract. (**b**) Intraoperative picture after ingestion of air in anterior chamber, irregular air bubble is seen due to iridocorneal adhesions. (**c**) Ultrasound biomicroscopy of the same showing fine central iridocorneal adhesions

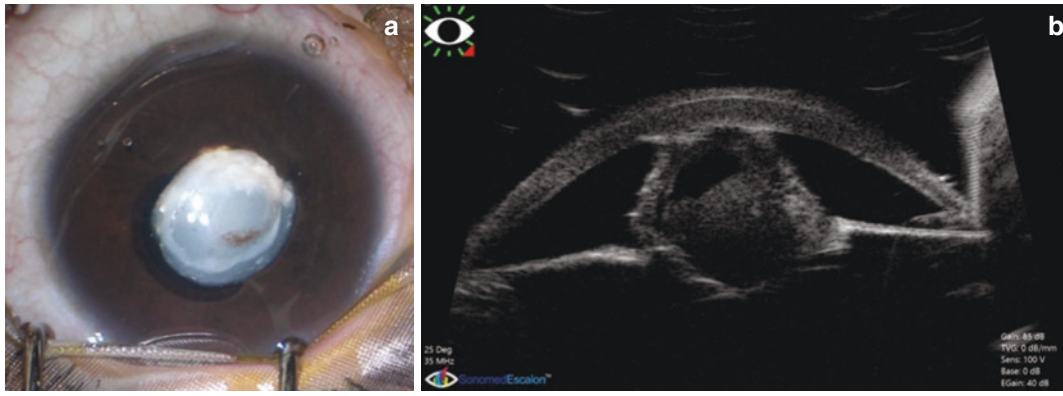

Fig. 15.11 Peter's anomaly type 2 in 4-month-old child. (**a**) Central corneal opacity with total cataract. (**b**) Ultrasound biomicroscopy of the same showing iridolenticular adhesions with anterior displacement of lens

short stature, brachydactylyl, ear abnormalities, and mental retardation [26, 27].

- Aniridia
  Aniridia is a rare congenital disorder characterized by iris hypoplasia along with other abnormalities of the eye [28]. Ocular abnormalities include dry eye, aniridia associated keratopathy (AAK) (Fig. 15.12), angle abnormalities, glaucoma, cataract, foveal hypoplasia, optic nerve hypoplasia, nystagmus, or strabismus [29–31]. Cataract morphology may be anterior or posterior subcapsular, lamellar, cortical, total or a combination of the above [28] (Fig. 15.13). Zonular weakness may be seen and ectopia lentis may be associated in some patients [28]. This can be managed with placement of capsular tension ring in mild cases (Fig. 15.14). Anterior polar or pyramidal cataract may be associated with aniridia along with remnants of persistent fetal vasculature [32] (Fig. 15.15).

15.2.4 Differential Diagnosis

The differential diagnosis of ASD includes obstetric trauma, congenital glaucoma, intrauterine infections like rubella, herpes simplex virus and bacterial infections, iridocorneo-endothelial syndrome (Fig. 15.16), metabolic diseases like mucopolysaccharidosis, mucolipidoses and tyrosinosis, congenital hereditary endothelial dystrophy, congenital hereditary stromal dystrophy and dermoids.

15.2.5 Investigations

Apart from usual investigations ultrasound biomicroscopy may allow us to preoperatively assess the area beneath the corneal opacity. It helps us to determine the area of corneal opacity, depth of opacity, presence of iris adhesion, anterior chamber depth and angle details in the involved area. The lens can be visualized and observed for kerato-lenticular adhesion or presence of any tilting of the lens (Figs. 15.10b, 15.11b, and 15.13d, e). This can help us in the planning of the cataract surgery and a better outcome.

15.2.6 Surgical Pearls

Patients with ASD should be screened for glaucoma and managed appropriately. They require optimization of visual function which includes refractive error prescription and tinted contact lenses for photophobia. This is important for prevention and treatment of amblyopia. Few patients may also require surgery for corneal opacity, lens abnormality or glaucoma management. Various challenges may be involved in the cataract surgery in cases with ASD.

- Corneal abnormalities: Corneal opacity or AAK may cause difficulty in visualization. Staining of the anterior capsule enhances its visualization during capsulorhexis. Other methods like use of illumination techniques

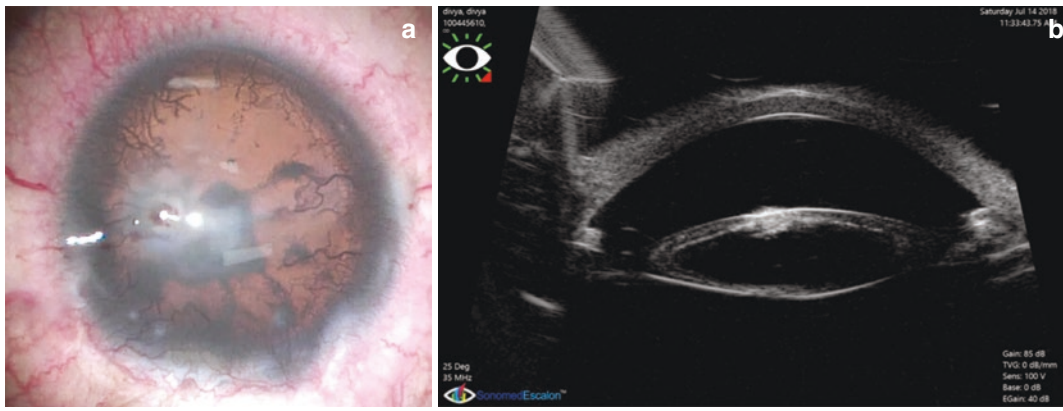

Fig. 15.12 Aniridia associated keratopathy with corneal opacity with 360° pannus. (**a**) Clinical picture. (**b**) Ultrasound biomicroscopy of the same showing anterior subcapsular cataract, not clearly seen clinically

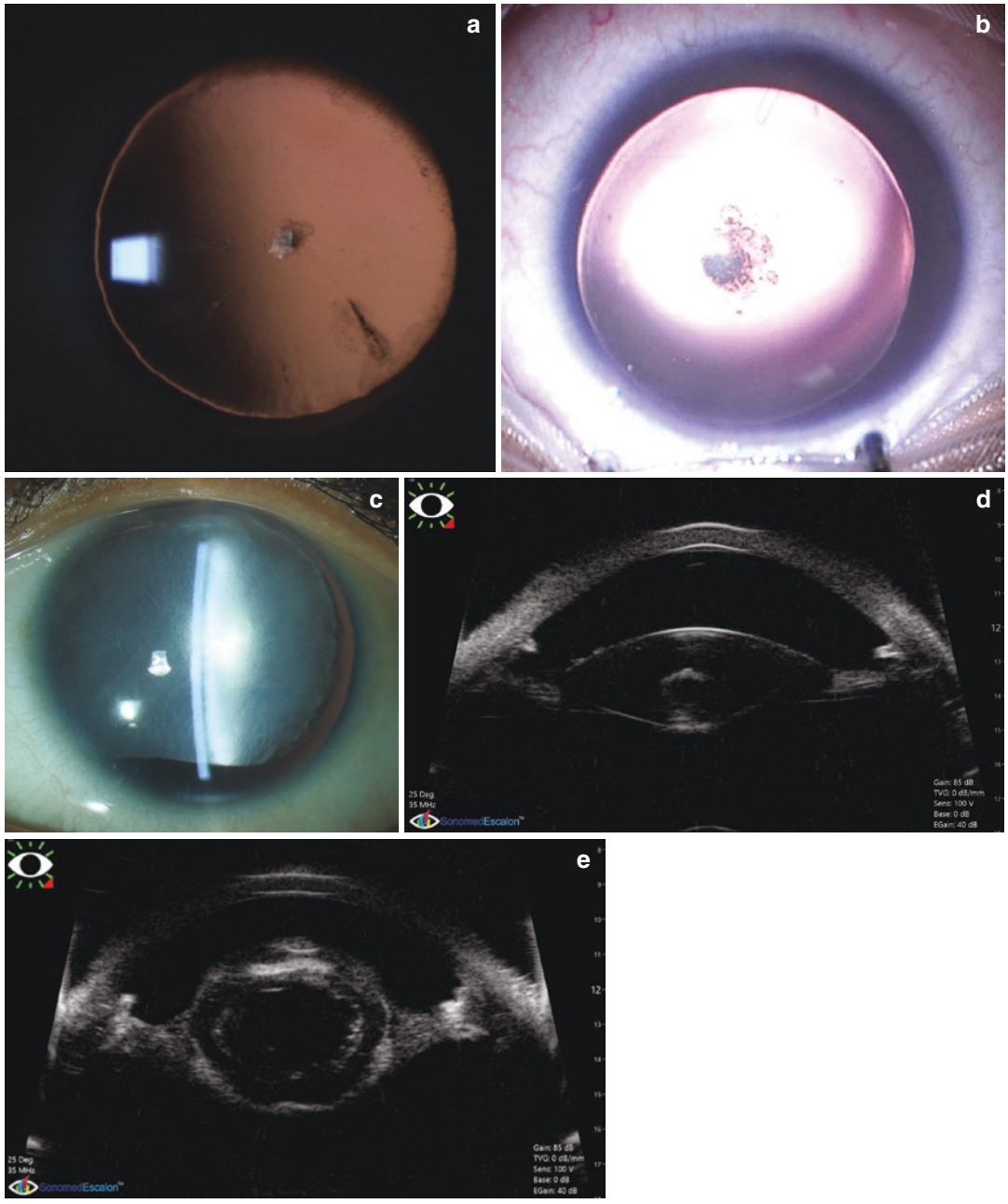

Fig. 15.13 Aniridia with cataract. (**a**) Clinical picture of insignificant anterior polar with cortical cataract. (**b**) Clinical picture of posterior subcapsular cataract. (**c**) Clinical picture of total cataract with inferior notching due to zonular laxity (Pseudo-coloboma). (**d**) UBM of aniridia patient showing minimal cataract and remnant of iris stump clearly with no subluxation. (**e**) UBM of aniridia patient with anterior polar and zonular cataract with zonular laxity causing increase in lens globularity

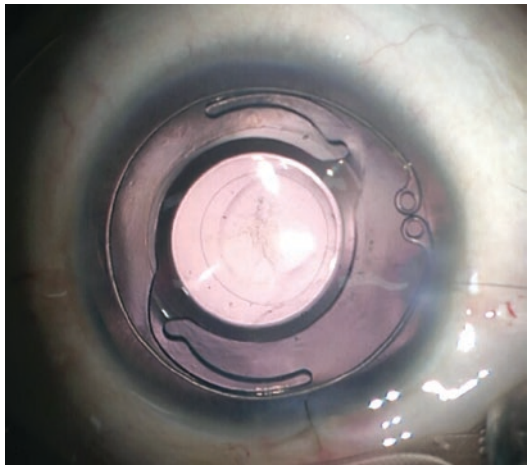

Fig. 15.14 Postoperative picture of aniridia with mild subluxation. Notice anterior and posterior capsulorhexis with well-centered IOL in bag with capsular tension ring

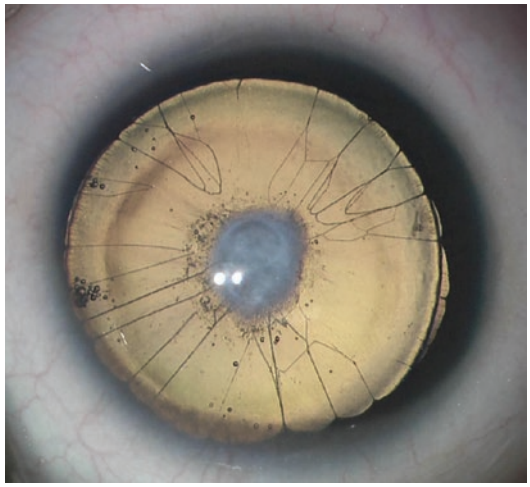

Fig. 15.15 Aniridia with anterior polar cataract with remnant of persistent fetal vasculature

like transcorneal oblique illumination or endoscope assisted surgery can help in better visualization but are time-consuming methods with a greater learning curve [33–35]. Image-guided surgery using femtosecond laser for cataract surgery in peter syndrome has also been recently used [36]. However, the depth and height of the femtosecond laser should be

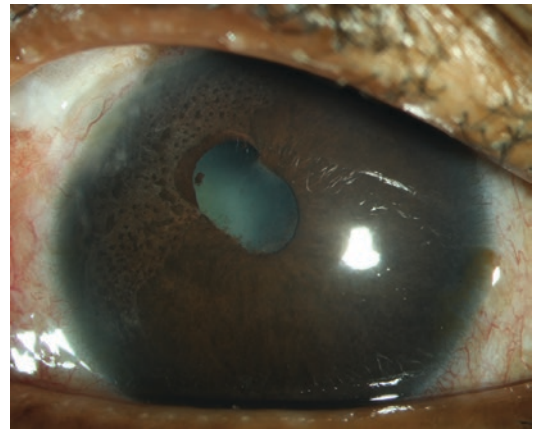

Fig. 15.16 Sixteen-year-old girl with Cogan Reese syndrome. Notice atophic iris and corectopia with iris nodules

cautiously adjusted to avoid damage to the endothelium.

- Presence of kerato-lenticular adhesion increases difficulty in surgical maneuvering. There may be risk of Descemet and endothelial damage during release of the keratolenticluar adhesions and difficulty to achieve appropriate size regular capsulorhexis. We have also noticed "irregular air bubble" in anterior chamber as a sign of presence of adhesions when they are not clearly visible (Fig. 15.17a, b).
- Iris abnormalities like corectopia, polycoria, iridocorneal adhesion, posterior synechiae between iris and lens may require anterior segment reconstruction may be required with synechiae release, anterior chamber formation or pupilloplasty along with the cataract surgery. Aniridia patients require use of tinted glasses or contact lens. Iris prosthetic devices may be used [37]. There is risk of secondary glaucoma, corneal decompenstion, band shaped keratopathy and device displacement [38] (Fig. 15.18).
- Zonular weakness: Aniridia cases have been reported with zonular laxity and lens subluxation. Use of capsular tension ring in cases with mild zonular laxity may give more desirable anatomical outcomes.

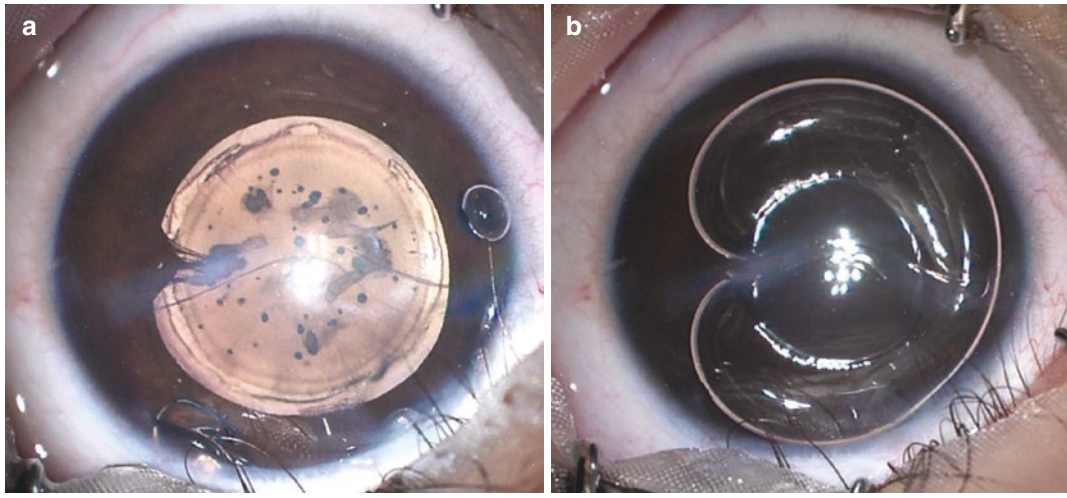

Fig. 15.17 Irregular air bubble sign in Anterior segment dysgenesis with glaucoma. (**a**) Corneal opacity with iridocorneal adhesions with Haab striae with anterior capsular pigments and zonular cataract. (**b**) Intraoperative picture after ingestion of air in anterior chamber, irregular air bubble is seen. Iridocorneal adhesions which were not clearly seen preoperatively are enhanced

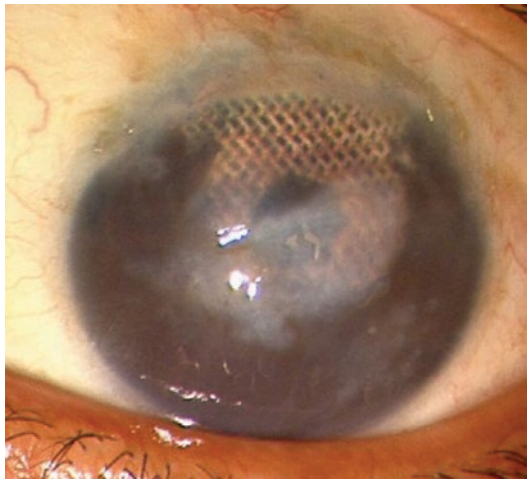

Fig. 15.18 One year postoperative picture of patient operated with Iris implant (outside center) with acquired aniridia (traumatic) with band shaped keratopathy and corneal decompensation

- Glaucoma management in cases with ASD is of importance and may include medical management, surgical management or both. Thus a regular follow-up with monitoring of visual acuity and the intraocular pressure is crucial.

Challenges in surgery in patients with ASD have to be carefully dealt with, in order to achieve satisfactory visual outcome. In addition to cataract surgery, glaucoma management is of utmost importance in these cases.

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