Review Article - Narrative Review

Taiwan J Ophthalmol 2023;13: 434-442

Access this article online

Quick Response Code:

Website: http://journals.lww.com/TJOP

DOI: 10.4103/tjo.TJO-D-23-00065

Peters anomaly: An overview

Arpita Khasnavis[1,2], Merle Fernandes[2]*

Abstract:

Peters anomaly (PA) is a rare, often bilateral, congenital corneal opacity, usually with a sporadic inheritance pattern, characterized by corneal opacities and irido-corneal or lenticular-corneal adhesions with a defect in the Descemet's membrane, occurring due to anterior segment dysgenesis during fetal development. Due to other ocular and systemic comorbidities, a team comprising pediatric cornea, glaucoma, and strabismus specialists in addition to a pediatrician and geneticist is necessary for the appropriate management of these children. Since the outcome of pediatric penetrating keratoplasty is variable and has a higher chance of failure when accompanied by additional procedures, such as lensectomy and vitrectomy, minimally invasive alternatives are increasingly being offered to these patients. Of note is the recently reported novel procedure: selective endothelialectomy for PA, which avoids the need for a corneal transplant and results in gradual clearing of the corneal opacity over time. In this overview, we aimed to describe the etiology, classification, pathophysiology, histopathology, clinical features, and management of PA.

Keywords:

Congenital corneal opacity, kerato-irido-lenticular dysgenesis, pediatric penetrating keratoplasty, Peters anomaly

Introduction

The first case of Peters anomaly (PA) was described by Dr. Peters, a German ophthalmologist, in 1906,[1] in a child with a shallow anterior chamber, synechiae between the iris and cornea, central corneal leukoma, and a defect in the Descemet's membrane (DM). The characteristic clinical features include a central corneal opacity with a corresponding defect in the posterior stroma, DM, and endothelium with or without iris or lenticular adhesions to the cornea.[2,3] It is a rare congenital disorder that occurs due to anterior segment dysgenesis during development with an incidence of around 1.5/100,000 live births[4] and maybe bilateral in 80% of cases.[2]

Methodology

The authors searched PubMed and Google Scholar in March 2023 with the terms "Congenital corneal opacity," "Peter's Anomaly," and "Peters' Anomaly." This yielded 2970 articles which were sifted for articles in the English language and pertaining to PA. Relevant important articles were cross-referenced from these.

Etiology

The inheritance pattern in PA is usually sporadic, although autosomal dominant and recessive inheritance have been reported from consanguineous marriages.[5] Specific chromosomal abnormalities have also been found in children diagnosed with PA like those in chromosomes 4, 11, 13, and 20.[6]

PA can be associated with mutations or deletions of homeobox genes involved in the development of the anterior segment[7] such as PAX6, PITX2, PITX3, COL4A1, FOXC1, and COL6A3,[8-10] with PAX6 and FOXC1 being the most common gene mutations. PAX6, PITX2, and FOXC1 are associated with neural crest cell (NCC) migration. PITX3 and FOXE3 are important for lens vesicle formation. Mutations in

[1]*Academy for Eye Care Education, L V Prasad Eye Institute, Hyderabad, Telangana, India,* [2]*Cornea and Anterior Segment Service, Shantilal Shanghvi Cornea Institute, L V Prasad Eye Institute, Hyderabad, Telangana, India*

***Address for correspondence:**
Dr. Merle Fernandes, Cornea and Anterior Segment Service, Shantilal Shangvi Cornea Institute, L V Prasad Eye Institute, Kallam Anji Reddy Campus, L V Prasad Marg, Banjara Hills, Hyderabad - 500 034, Telangana, India.
E-mail: merle@lvpei.org

Submission: 01-05-2023
Accepted: 16-07-2023
Published: 20-10-2023

How to cite this article: Khasnavis A, Fernandes M. Peters anomaly: An overview. Taiwan J Ophthalmol 2023;13:434-42.

SOX2, which plays a major role in ocular development, have also been found in PA with other associated ocular defects like microphthalmia/anophthalmia.[11] Peters plus syndrome (PPS) is an autosomal recessive disorder associated with biallelic pathogenic variants in B3GALTL.[12] Due to overlapping genetic mutations between PA and other genetic disorders, for example, Axenfeld–Rieger Syndrome, a genotypic-phenotypic correlation is difficult.[13] Table 1 summarizes the genes associated with PA.[14-16]

Classification

In 1974, Townsend *et al*.[17] subdivided PA into three groups:

1. Cornea with central leukoma only
2. Cornea with central leukoma and corneo-lenticular touch
3. Cornea with central leukoma associated with Rieger mesodermal dysgenesis.

Recently, it has been classified into two types:

- Type I is characterized by irido-corneal adhesion with central corneal opacity. The density of the central corneal opacity is variable.[18] It is usually unilateral with clear peripheral cornea and rarely associated with edema or scleralization.[19] It has a good visual prognosis and is rarely associated with systemic abnormalities
- Type II is characterized by corneo-lenticular touch with the lens directly adherent to the corneal opacity or corneal opacity with cataract. It is usually bilateral and is associated with systemic anomalies.[20,21]

PA can also be associated with glaucoma (20%), microphthalmia (18%), coloboma (6%), rarely sclerocornea, corectopia, iris hypoplasia, irido-corneal endothelial syndrome, aniridia, and persistent fetal vasculature.[22]

The most common syndrome associated with PA is PPS, which is defined as an anomaly with systemic associations such as facial dysmorphism, cleft lip/palate, short stature, brachydactyly, abnormal ears, central nervous system defects, congenital heart defects, genitourinary abnormalities, and intellectual disability.[23] Classic PPS is characterized by a triad of PA, brachydactyly, and short stature.[24]

Pathophysiology

NCCs are a population of multipotent embryonic stem cells that give rise to a wide range of cell and tissue types throughout the body. The migration of the NCCs in three distinctive waves plays an important role in the normal development of the cornea.

The first wave: NCCs migrate into the space between the anterior surface of the lens and the surface ectoderm and eventually form the corneal endothelium.[25,26]

The second wave: NCCs migrate between the corneal epithelium and endothelium to form the keratinocytes of the corneal stroma.[17,27]

The third wave: NCCs migrate to the angle between the endothelium and the anterior edge of the optic cup, thereby forming the ciliary body and iris stroma.

The basic abnormality is probably in the failure of the normal differentiation of the mesoderm into normal endothelium. Matsubara *et al*.[28] hypothesized that a maldevelopment of iris stroma, ciliary stroma, and goniodysgenesis occurs due to developmental disorders of the NCCs sometimes in the 4th–7th week of gestation resulting in the clinical features of PA.

Various developmental mechanisms for PA, including faulty separation of the lens vesicle from the surface ectoderm, primary abnormal migration of NCCs into

Table 1: Summary of genes associated with Peters anomaly

Gene	Location	Function	Abnormality
PAX6	Chr 11p13	Transcription factor	Aniridia, PA, congenital cataract, foveal hypoplasia, microphthalmia, morning glory disc anomaly, and optic nerve hypoplasia
PITX2	Chr 4q25	Transcription factor	Ocular, cardiac, and hearing defects, Axenfeld–Rieger syndrome, type 1; anterior segment dysgenesis 4; and ring dermoid of the cornea
CYPIBI	Chr 2p22	Monooxygenase enzyme	PA
FOXC1	Chr 6p25	Transcription factor	Anterior segment dysgenesis 3 and the type 3 Axenfeld–Rieger syndrome
COL6A3	Chr 2q37.3	Transcription factor	PA
B3GLCT	Chr 13q12.3	Protein-coding gene	PPS, growth retardation, and developmental delay
SOX2	Chr 3q26.3	Oncogene (nonsense mutation causes ocular pathology)	PA, microphthalmia, anophthalmia, cerebral atrophy, growth retardation, and developmental delay
PITX3	Chr 10q25	Transcription factor	Bilateral PA
FOXE3	Chr 1p32	Transcription factor	Microcornea and PA
COL4A1	Chr 13q34	Basement membrane collagen	Bilateral PA

PA=Peters anomaly, PPS=Peters plus syndrome

the cornea, and intrauterine corneal inflammation and absence of the corneal endothelium and DM, have been described.[29] In severe cases, the lens is also adhered to the cornea, which can cause corneal staphylomas.[30]

Histopathology

The most characteristic corneal feature of PA is the abnormality of both the DM and the endothelium in the area of corneal opacity and thinning or absence of the endothelial basement membrane.[31,32] These findings were also noted on electron microscopy and suggested a developmental abnormality involving the mesodermally derived elements (stroma and endothelium)[33] although subsequently the failure of the lens vesicle to separate from the cornea has been attributed to be the cause.

The endothelial cells are markedly attenuated, and DM in the region of the corneal opacity is immature. Only at the point of iris adhesion, DM and endothelial cells are absent. Aberrant DMs can be found anterior to the original one.

In the anterior layers of the cornea, overlying the posterior defect, disorganization of the corneal epithelium with edematous changes and replacement of the Bowman's layer with pannus is found.[20]

Edematous changes are also seen in the stroma with disorganization of the lamellae in the posterior stroma adjacent to the irido-corneal adhesions. Keratocytes in the anterior stroma were normal with an increase in phagocytic cells in the retrocorneal fibrous layer. Of note were the findings of endothelial cells in zones with absent or attenuated endothelium suggesting that over time, there is a form of endothelial self-repair by probable migration of endothelial cells from the periphery to the center.[1,34]

The peripheral cornea is normal in all aspects. The trabecular meshwork demonstrates changes characteristic of old age such as wide-banded collagen and the presence of phagocytosed pigment granules in the endothelium. Figure 1 shows the typical histopathological findings in a patient with PA.

History and Evaluation

Children with congenital corneal opacities require a careful ocular and systemic examination since the ophthalmologist may be the first physician to diagnose life-threatening systemic associations in patients with PA. A team approach is necessary with the involvement of pediatric cornea and glaucoma specialists, pediatric strabismus specialist, pediatrician or neonatologist, geneticist, and a pediatric anesthetist.

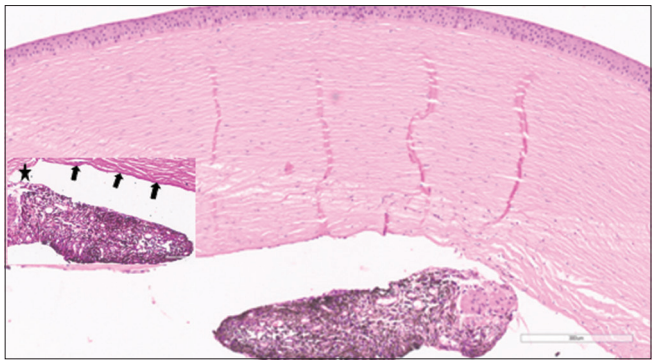

Figure 1: Photomicrograph of anomaly from a penetrating keratoplasty corneal button showed scarred cornea and anterior synechiae formation with a loss of Descemet's membrane (DM) at the site of adhesion: H and E stain; ×10 magnification. Periodic acid–Schiff stain in inset showed magenta color DM (arrow) and absence of DM at the site of peripheral anterior synechiae (arrow); ×24.8 digital scanner magnification. 4–5 layered stratified squamous epithelium. The Bowman's layer is fragmented and replaced by scarred tissue. The stroma has scarring with a loss of lamellar pattern and hyalinized collagen bundles. DM presents with endothelial cells; however, at the site of peripheral anterior synechiae, DM is absent*

An ante- and intranatal history is required with details of parental consanguinity and inquiry about siblings and other family members. Isotretinoin intake in the first trimester of gestation may cause a clinical picture similar to PA with lenticulo-irido-corneal adhesions.[18] A cardiac evaluation, an abdominal ultrasound, neuroimaging, and a hearing assessment are needed to complete the workup of these patients.[35] Visual acuity should be assessed with age-appropriate methods. An examination under anesthesia is usually needed, which necessitates prior evaluation by a neonatologist or pediatrician to rule out other associated congenital malformations as outlined earlier. The role of the pediatric anesthetist cannot be underscored since associated cardiac anomalies or facial dysmorphisms require special considerations.[36]

On examination under anesthesia, with a handheld slit lamp, a corneal opacity of variable density and size is usually noticed. The opacity is usually central, rarely vascularized with relative sparing of the peripheral cornea. The diameter of these lesions and their relation to the undilated pupil with an assessment of the peripheral clear cornea are required as these help guide further management. The presence of irido-corneal or lenticular-corneal adhesions should be assessed. Figure 2 highlights the clinical features of a patient with PA.

In addition, the child may also have nystagmus, glaucoma, cornea plana, sclerocornea, persistent hyperplastic primary vitreous, chorioretinal coloboma, disc coloboma, and retinal dysplasia.[16,20]

Congenital glaucoma in PA is due to developmental anomaly in the trabecular meshwork and the Schlemm's canal.[37] Glaucoma may present in infants or early

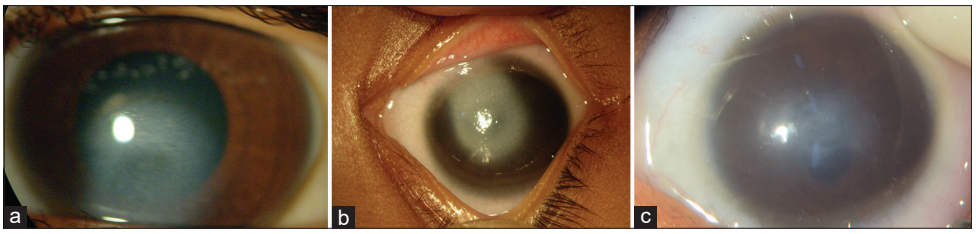

Figure 2: (a) Slit-lamp photograph of a child with unilateral mild corneal opacity (Peters' anomaly Type I) partially obscuring the visual axis with visible details of the anterior chamber. (b) Clinical photograph of an infant under anesthesia with a dense central and peripheral corneal opacity obscuring the visual axis, classified as Peters anomaly Type I. (c) Clinical photograph of an infant with a dense central corneal opacity with iris adhesions without lenticular adhesion (Peters anomaly Type I)

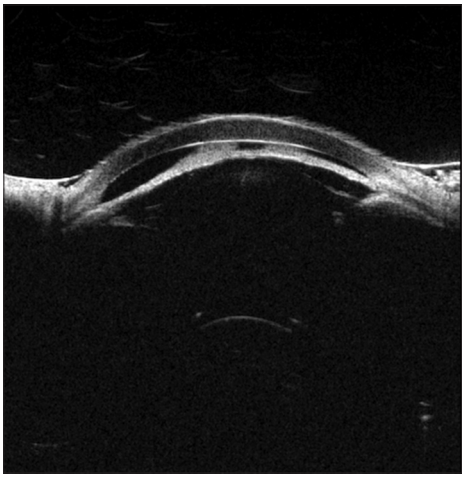

Figure 3: Ultrasound biomicroscopy of the cornea in a patient with peters anomaly Type II, which shows central irido-corneal and lenticular adhesions with a thickened hyperreflective Descemet's membrane

childhood, usually in children with PA Type II, and is extremely difficult to treat, necessitating surgical intervention in most cases.

Staging

Disease severity has been classified into mild, moderate, and severe forms.

Mild: corneal opacity with normal iris or lens.[38]

Moderate: central irido-corneal adhesions or iris defects such as atrophy or abnormal vasculature with sclerocornea, microphthalmos, aniridia, and coloboma.[21]

Severe: corneo-lenticular adhesion or corneal staphyloma.

Investigations

Ultrasound biomicroscopy

Since routine slit-lamp examination precludes visualization of the anterior segment structures in the presence of dense corneal opacities, ultrasound biomicroscopy (UBM) has proven to be an invaluable tool to identify the iris, lens, and ciliary body relationship with the cornea [Figure 3]. A clinic-pathologic correlation of UBM findings showed a good correlation with the histopathological examination and resulted in a change in the clinical diagnosis in almost 1/3rd of cases.[39] However, it requires sedation or examination under anesthesia, and despite being safe and painless, requires a skilled technician for execution and expertise to interpret the scans. A classification scheme on UBM has been described, which would be useful to determine the management options and prognosis of cases with congenital corneal opacities.[40]

According to UBM image classification, PA can be classified into three types [Table 2], which correlates well with the clinical description of anterior segment dysgenesis by Waring[29] and can also predict the outcome of surgical intervention.[40]

Spectral-domain optical coherence tomography

Anterior segment optical coherence tomography (OCT) is a valuable investigation for clinical assessment of the extent of corneal opacity in children with anomaly providing a greater resolution than UBM while being a noninvasive and fast technique. It has been used in the clinic even in neonates as young as 2 days[41] or under anesthesia[42] and has helped to modify the surgical plan in 21% of patients with PA. Integrated intraoperative OCT can be used during lamellar keratoplasty in children with PA.[43,44]

Ultrasound B-scan

Ultrasound B-scan is an essential tool for the evaluation of the posterior segment in the eyes where the posterior segment details cannot be appreciated due to severe corneal opacity.

Differential Diagnosis

The traditional differential diagnosis for congenital corneal opacity included sclerocornea, trauma, ulcer, mucopolysaccharides, anomaly, congenital hereditary endothelial dystrophy, and corneal dermoid (acronym: STUMPED). However, several conditions were not included in this simple mnemonic, and hence, this is largely not used by pediatric corneal surgeons.

Table 2: Ultrasound biomicroscopy image classification of Peters anomaly[41]

Type	Feature	Surgical outcome
I	Corneal DM and endothelial defect	Good outcome postpenetrating keratoplasty
II	Type 1 + irido-corneal adhesions	Variable outcome post-PKP, may or may not have a good outcome
III	Type II + kerato-lenticular involvement	Usually poor outcome. May need cataract surgery combined with PKP

UBM=Ultrasound biomicroscopy, DM=Descemet's membrane, PKP=Penetrating keratoplasty

A new classification of congenital corneal opacity was proposed by Ken Nischal.[45] According to this classification, neonatal corneal opacity can be classified as primary and secondary.

Primary neonatal corneal opacities are present since birth, and the causes are corneal dystrophies such as congenital hereditary endothelial dystrophy, posterior polymorphous corneal dystrophy, congenital hereditary stromal dystrophy, X-linked endothelial corneal dystrophy; dermoid (limbal and central), isolated peripheral sclerocornea.

The secondary causes of neonatal corneal opacification can be congenital or acquired.

Congenital causes include kerato-irido-lenticular dysgenesis, where either the lens fails to separate from the cornea, the lens separated but fails to form thereafter, or the lens fails to form altogether. Based on this classification, Peters anomaly Type II falls under secondary congenital corneal opacification, where the lens fails to separate from the cornea, and Peters anomaly Type I is possibly a mechanical cause, where the iris adheres to the cornea with the lens being normal.

Acquired causes include infections and trauma, and metabolic causes such as mucopolysaccharidoses and cystinosis.

Management

Since the opacity in PA lies in the visual axis, there is a significant risk of sensory deprivation amblyopia, and hence, any intervention should happen in the 1st year of life. In addition, almost 50% of patients develop glaucoma. Thus, the main aim of management is to improve vision, prevent amblyopia, and control intraocular pressure (IOP). In addition, genetic counseling may be necessary, if a recessive or dominant pattern is found, to determine the risk in future pregnancies. The management options consist of medical management or surgery.

A management algorithm has been proposed by Elbaz *et al*.[46] based on their analysis of the varied phenotypic presentation in 80 eyes of 54 patients. Observation, medical, or surgical intervention has been recommended based on the severity and location of the corneal opacity and associated kerato-lenticular touch.[46]

Medical management

Medical management is not the definitive management for PA. The options include using phenylephrine to dilate the pupil while awaiting surgical management to prevent amblyopia, occlusion therapy depending on the density of corneal opacity and laterality of the eye involved,[20] and anti-glaucoma medications to control the IOP in cases with congenital glaucoma.[37] In a series of 15 eyes of nine patients with PA who did not undergo a penetrating keratoplasty (PKP), Yoshikawa *et al*.[38] observed that in four eyes, the opacity was noted to regress over time. Hence, observation may be considered in patients with mild opacity with a close watch for the development of amblyopia.

Surgical management

The goal of surgical intervention is to provide better visual acuity and prevent the development of amblyopia and squint. The type of intervention required depends on the age at presentation, laterality, and the extent of corneal involvement, i.e. its depth, central or peripheral corneal involvement, lenticular involvement, associated retinal pathologies, and the IOP.[2] These include peripheral iridectomy, PKP with or without cataract extraction, Descemet's stripping automated endothelial keratoplasty (DSAEK), and selective endothelialectomy for PA (SEPA).[47]

Peripheral optical iridectomy

It is a safe and simple procedure, proposed as an alternative to PK in children with central corneal opacity.[48,49] Besides being an easier and shorter procedure with minimal postoperative care, no dependency on mydriatics, and less chance of developing glaucoma, this is a useful procedure when there is a dearth of corneal tissues for PKP. However, visual recovery is dependent on early refraction and spectacle correction accompanied by amblyopia therapy. Spierer *et al*.[49] have shown a significant improvement in visual acuity, particularly in bilateral PA as opposed to unilateral disease. Table 3 summarizes the outcome of iridectomy in PA.

Pediatric penetrating keratoplasty

Pediatric PKP can be challenging due to technical difficulties of the surgical procedure, detrimental effects of sensory deprivation amblyopia, higher rate of rejection and failure, and difficult postoperative evaluation and compliance.

Table 4 summarizes the anatomical and functional outcomes of PKP for PA. Graft clarity is highly variable

Table 3: Summary of the literature on peripheral iridectomy in Peters anomaly

Author	Number of eyes	Duration of FU (months)	Visual acuity (LogMAR)		Complications	Outcome
			Preoperative	Postoperative		
Jünemann *et al*.[48]	20	42	PL+	1–1.4 (*n*=9)	Glaucoma (*n*=2) Phthisis bulbi (*n*=1)	
Spierer *et al*.[49]	29	41.6±43.8	2.5±0.3	1.8±0.6	Glaucoma (*n*=5)	PKP (*n*=2) Repeat iridectomy (*n*=1) Kpro (*n*=1)

FU=Follow-up, PKP=Penetrating keratoplasty, Kpro=Keratoprosthesis, PL=Perception of light

ranging from 39% to 90%. The main causes of poor outcome following PKP were rejection, glaucoma, and infection. In addition, visual acuity improved following PKP; however, very few studies[50] reported vision better than 20/100. Good outcomes post-PK in patients with PA are seen in patients with mild-to-moderate disease, between 2 and 12 months of age, compliant to treatment with intensive postoperative care with frequent follow-ups with early suture removal.[50]

Posterior lamellar keratoplasty

PA involves the posterior part of the stroma, DM, and the endothelium. Hashemi *et al*.[59] performed DSAEK in two children with PA Type II in an attempt to replace the dysfunctional endothelium and reduce the corneal haze while providing the benefits of a closed chamber procedure without sutures. However, their reliance on clinical assessment without ASOCT resulted in an underestimation of the overlying corneal haze, which persisted in one patient. The challenges of DSAEK in patients with PA included poor visualization, difficulty in scoring the DM due to stronger adhesion between the DM and stroma, and shallow anterior chamber with irido-corneal adhesion, with the possible need for rebubbling.

Selective endothelialectomy in peters anomaly

Since the peripheral corneal endothelium is normal in PA type I with abnormal/absent endothelial cells in the periphery, a minimally invasive endothelial removal was performed by Soh and Mehta in a 21-month-old child with unilateral PA type I.[60] This resulted in excellent anatomical recovery with improvement in visual acuity from 20/960 preoperatively to 20/30 over 1 year. A similar anatomic outcome was observed over a 16-week follow-up in an 8-year-old child with bilateral PA, who underwent this procedure in the worse eye.[61] The largest study of SEPA (either alone or combined with optical iridectomy or lensectomy), performed on 34 eyes of 28 patients, showed that almost 85% of the eyes had a partial or complete clearing of the central visual axis with a significant improvement in postoperative vision.[62] The authors recommend this procedure for patients with mild-to-moderate PA with <7 mm involvement, which allows the repopulation of the area of denuded endothelium by healthy endothelial cells from the periphery. The restoration of corneal clarity is noted as early as 1 month after surgery but continues to clear even up to 18 months; hence, a close follow-up with attention to visual acuity and institution of amblyopia therapy is essential. The risk factors for failure included the severity of PA, glaucoma, and microcornea.

Associated congenital glaucoma

Congenital glaucoma occurs in 50%–70% of the patients with anomaly and is considered one of the most difficult glaucomas to manage.[37,63,64] Glaucoma surgery with medical management may help in IOP control in children with anomaly. Surgical procedures that can be performed include trabeculectomy, diode laser cyclophotocoagulation, trabeculectomy, goniotomy, Molteno shunt implantation, cyclodialysis, and cyclocryotherapy.[37]

Management of strabismus and amblyopia in Peters' anomaly

Visual outcome in children with the anomaly is poor despite early surgical intervention due to the presence of various ocular pathologies, including glaucoma, and the requirement of numerous surgeries such as PKP, lensectomy, vitrectomy, peripheral iridectomy, and glaucoma surgery. Significant asymmetry in the central corneal opacity between the two eyes may cause amblyopia, nystagmus, and sensory strabismus.[22] Seventy-two percent of the patients can have strabismus, with esotropia (54%) being the most common. Amblyopia therapy is successful in patients with a milder form of the disease.

Conclusion

The importance of a detailed clinical examination and the integrated comanagement involving pediatric ophthalmologist, cornea and glaucoma specialists, pediatrician, and geneticist cannot be underscored in the approach to children with congenital corneal opacities like anomaly. While the outcome of PKP in patients with PA is variable and fraught with complications, novel minimally invasive techniques like SEPA have shown promising results. Early and aggressive amblyopia therapy is essential to restore vision.

Acknowledgment

The authors would like to acknowledge Dr. Dilip Misra for providing the histopathology image and Dr. Muralidhar Ramappa for the UBM image.

Table 4: A literature review of penetrating keratoplasty for Peters anomaly

Author	Number of eyes/ patients Unilateral/bilateral	Duration of FU (months)	Final VA (Snellen/ LogMAR)	Additional procedures	Complications	Graft clarity
Zaidman *et al.*[50]	30 eyes/24 patients 24/0	78.9	>3 years 20/20–20/50 (*n*=7; 29%) 20/60–20/100 (*n*=6; 25%) 20/200–CF (*n*=9; 38%) HM (*n*=2; 8%) <3 years VA not assessed	Cataract surgery (*n*=6)	Rejection (*n*=5) Corneal ulcer (*n*=2) RD (*n*=1)	90 (PA type 1)
Rao *et al.*[51]	40 eyes/32 patients 24/8	22.8±34.8	NA	Synechiolysis (*n*=18; 45%) Lensectomy + vitrectomy (*n*=6; 15%) Spontaneous lens expulsion (*n*=1)	Rejection (*n*=15) Corneal ulcer (*n*=7) Endothelial decompensation (*n*=1)	43 (2 years)
Yang *et al.*[52]	72 eyes/47 patients 11/36	130.8	>20/100 (*n*=7; 10%) 20/200–20/400 (*n*=14; 19%) < CF (*n*=52; 71%)	Lensectomy + vitrectomy (*n*=25)	Graft failure (*n*=44) Cataract (*n*=15) Glaucoma (*n*=14) RD (*n*=16) Phthisis (*n*=7)	39
Basdekidou *et al.*[53]	14 eyes/14 patients 14/0	31.28	>20/100 (*n*=2) CSM (*n*=8) Poor fixation (*n*=3)		Rejection (*n*=4) Cataract (*n*=4) Glaucoma (*n*=1)	78.6
Chang *et al.*[54]	21 eyes	NIA	20/400–20/200 (*n*=3) CF-HM (*n*=5) LP (*n*=5) NLP (*n*=8)	Cataract surgery (*n*=3)	NIA	NIA
Chang *et al.*[55]	22 eyes 14/7	90.6	2.344±0.668	Lensectomy + vitrectomy (*n*=4)	Rejection (*n*=9) Glaucoma (*n*=10) Phthisis (*n*=1) RD (*n*=1) Scleral thinning (*n*=1)	48
Lin *et al.*[56]	37 eyes 11/16	18±3	>20/260 (*n*=18) <20/260 (*n*=12)	Lensectomy/ vitrectomy (*n*=3)	Rejection (*n*=8) Glaucoma (*n*=7) Cataract (*n*=5)	73
Donoso Rojas *et al.*[57]	27 eyes	122.4	0.97±0.78	Lensectomy + vitrectomy (*n*=15)	Failure (*n*=8) Glaucoma (*n*=8) Corneal ulcer (*n*=4) Anterior staphyloma (*n*=4)	45.5
Elbaz *et al.*[58]	36 eyes 20/20	75.8±52.9	PA I: 1.0±0.6 (*n*=25) LP (*n*=2) NLP (*n*=2) PA II: 1.4±1.3 (*n*=12) LP (*n*=2) NLP (*n*=3)	Lensectomy/ vitrectomy (*n*=7)	Rejection (*n*=7) LSCD (*n*=5) Glaucoma (*n*=4) RCM (*n*=4) Corneal ulcer (*n*=1) RD (*n*=6)	67.6

FU=Follow-up, VA=Visual acuity, CF=Counting fingers, HM=Hand movements, LP=Light perception, NLP=No light perception, NIA=No information available, CSM=Central steady maintained, PA I=Peters anomaly type I, PA II=Peters anomaly type II, RD=Retinal detachment, LSCD=Limbal stem cell deficiency, RCM=Retrocorneal membrane, NA=Not available, LogMAR=Logarithm of the minimum angle of resolution

Declaration of patient consent

The authors certify that they have obtained all appropriate consent forms from the legal guardians of the patients. In the form, the guardians have given the consents for the images and other clinical information of the patients to be reported in the journal. The guardians understand that the names and initials of the patients will not be published and due efforts will be made to conceal the identity, but anonymity cannot be guaranteed.

Data availability statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

Financial support and sponsorship

This study was funded by Hyderabad Eye Research Foundation, Hyderabad, India.

Conflicts of interest

The authors declare that there are no conflicts of interests in this paper.

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