SECONDARY ACQUIRED GLAUCOMA IN CHILDHOOD

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Childhood glaucoma is a heterogenous group of diseases, which all share the final common pathway of ocular hypertension, and pressure related damage to the ocular structures in form of optic neuropathy, and progressive visual field loss. It is estimated to cause significant percentage of blindness in children, from 1.2% in UK to 3-7% in India[1-3].

Being a heterogenous group of disorders, it was classified by overlapping and variably defined nomenclature, which denoted an age of onset rather than the underlying mechanism until an international collaboration on childhood glaucoma for the World Glaucoma Association (WGA) meeting in July 2013, gave the definitions of childhood glaucoma, glaucoma suspect, and a new childhood glaucoma classification system which is the most widely followed at present (Figure 1)[4]. The Childhood Glaucoma Research Network (CGRN) is an international consortium of clinicians and scientists who specialize in treating children with glaucoma.

According to CGRN classification, Childhood age is based on national criteria:

US: younger than 18 years

EU, UK, UNICEF: 16 years or younger

GLAUCOMA

IOP related damage to the eye; at least 2 criteria are required for the diagnosis:

1. IOP > 21 mm Hg; however, investigator discretion is required if there is data of examination under anesthesia alone due to variable effects of anesthesia on all methods of IOP assessment.
2. Optic disc cupping: progressive increase in cup-disc ratio.
3. Cup disc asymmetry of ≥ 0.2 when the optic discs are of similar size, or there is focal rim thinning.

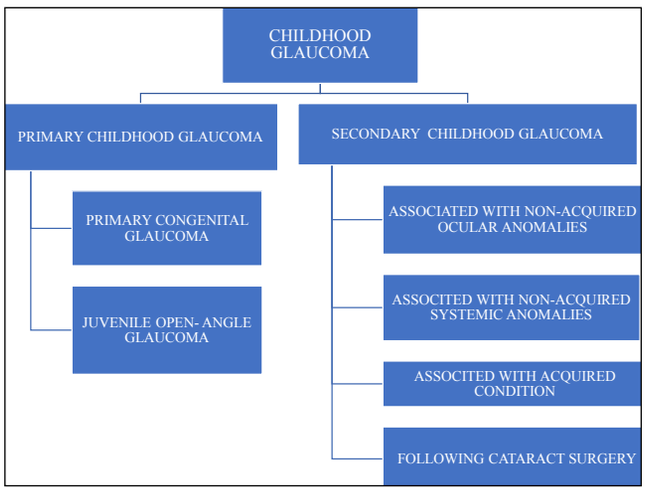

Figure 1: *Childhood Glaucoma Research Network (CGRN) classification*

4. Corneal findings: Haab's striae or diameter ≥ 11 mm in newborn, > 12 mm in a child < 1 year, or > 13 mm at any age
5. Progressive myopia, myopic shift, or an increase in ocular dimensions out of keeping with normal growth.
6. Reproducible visual field defect consistent with glaucomatous optic neuropathy with no other observable reason for the defect.

GLAUCOMA SUSPECT

No IOP related damage; at least 1 criteria is required for the diagnosis:

1. IOP > 21 mm Hg on 2 separate occasions.
2. Suspicious optic disc appearance for glaucoma, i.e., increased cup disc ratio for size of optic disc.
3. Suspicious visual field for glaucoma.
4. Increased corneal diameter or axial length in the setting of normal IOP.

Primary congenital glaucoma is the most common form of childhood glaucoma. However, secondary glaucoma also forms a significant proportion of childhood glaucoma. Various studies have given variable prevalence of secondary childhood glaucoma ranging from 20-52%[5-7].

In this write-up, we shall be discussing about secondary acquired glaucoma which includes glaucoma secondary to uveitis, trauma, steroid induced, tumours (benign/malignant, ocular/orbital), retinopathy of prematurity (ROP), prior ocular surgery other than cataract surgery. Trauma, uveitis and steroids are the most common ones amongst all these causes.

TRAUMATIC GLAUCOMA

Trauma is a very important cause of secondary glaucoma in children. School going children are most commonly affected due to unsupervised activities among active peers. Mode of trauma can be blunt trauma with toys, household appliances, ball games, gullidanda, sports activities; or penetrating eye injuries with sharp objects, bow and arrow; firecracker related injuries or chemical burns.

Seven rings of trauma include:

1. Sphincter tear
2. Iridodialysis
3. Angle recession
4. Separation of ciliary body attachment to scleral spur-cyclodialysis.
5. Trabecular meshwork (TM) tear.
6. Zonular dialysis resulting in subluxation of the crystalline lens.
7. Retinal dialysis

Post-traumatic glaucoma can be

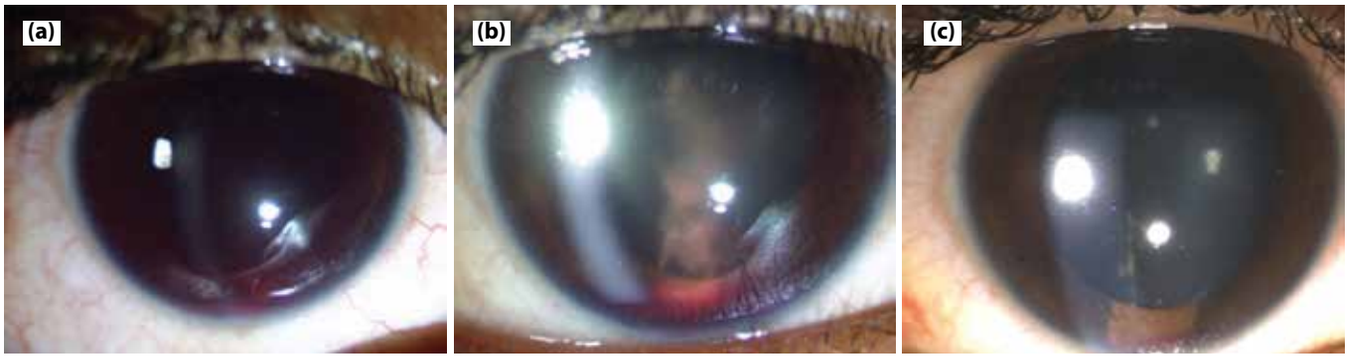

Figure 2: A: *Diffuse hyphaema in a 8 year old child following blunt trauma.* ***B:*** *After 2 days of head end elevation and conservative treatment, hyphaema was resolving and fibrin in pupillary area.* ***C:*** *Near complete resolution of hyphaema and disappearance of fibrin on 5 th day*

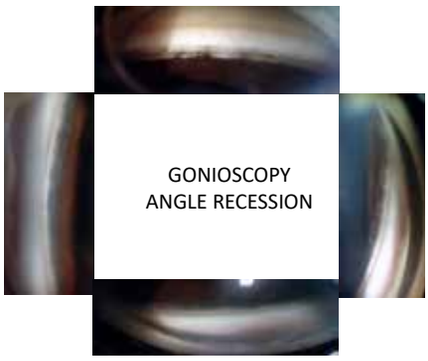

Figure 3: *Angle recession in >270 degrees visible as widened ciliary body band with synechiae and pigmentation in inferior angle.*

- Early onset due to hyphaema (Figure 2), lens related due to cataractous lens/lens subluxation/dislocation leading to angle closure or pupillary block or anterior capsular rupture leading to lens particle glaucoma.
- Late onset due to angle recession (Figure 3), peripheral anterior synechiae, ghost cell glaucoma or post surgical after surgical intervention for traumatic cataract or retinal detachment.

In a case of hyphaema, raised intraocular pressure (IOP) occurs due to inflammation and trabeculitis or blockage of trabecular meshwork with red blood cells/ inflammatory cells.

A large blood clot can cause raised IOP due to pupillary block.

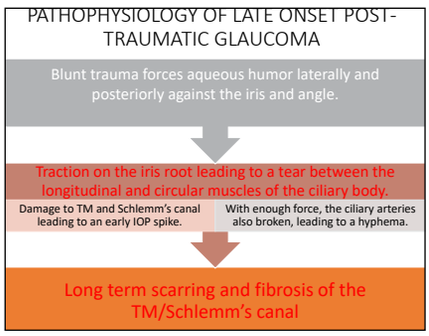

Figure 4: *Flow-chart depicting the pathophysiology of post-traumatic glaucoma.*

Approximately one-third of all hyphema patients exhibit increased intraocular pressure in early period which increases to 2/3rd in cases of rebleed[8,9]. In every case of blunt trauma, gonioscopy and indirect ophthalmoscopy with peripheral scleral depression is very important to rule out angle recession or retinal dialysis so as to prognosticate the disease and treat at the earliest. Scleral depression and dynamic gonioscopy should be avoided for about 4 weeks[10].

IOP elevation in angle recession usually presents with two peaks of onset, first around 3 months post injury and second after an interval of 10 years[11]. Angle recession is defined as separation of longitudinal and circular ciliary muscles. On gonioscopy, it is visible as wide irregular ciliary body band (Figure 3) and it should always be confirmed by comparing with the fellow normal eye. Although angle recession can be seen with blunt trauma without hyphaema, but the incidence increases in cases of hyphaema (about 60-100%). About 6-7% of the patients with angle recession go on to develop glaucoma[12,13]. Angle recession in itself is not responsible for glaucoma, but it is an indirect measure of the severity of trauma which must have caused damage to the trabecular meshwork (TM) (Figure 4). Late onset glaucoma in cases of angle recession also suggests patient predisposition to open angle glaucoma as IOP rise and glaucoma onset has been noted in 50% of the contralateral eyes years after the IOP rise in traumatic eye. It has been hypothesized that angle recession might be accelerating the process of manifestation of glaucoma in traumatic eye of these individuals[14].

In cases of penetrating trauma, IOP is usually low, but it can be high in cases with flat anterior chamber and peripheral anterior synechiae due to self-sealed corneal laceration or anterior capsule rupture leading to lens particle glaucoma.

- Following factors have been found to be the predictors for post-traumatic glaucoma:
  1. Presence of increased angle pigmentation
  2. Elevated baseline IOP
  3. Hyphema

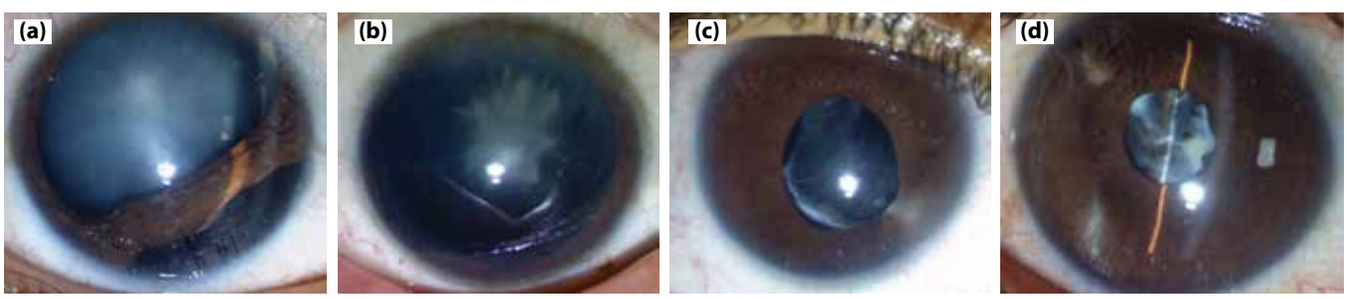

Figure 5: *Different presentations of trauma.* ***A:*** *Iridodialysis and cataract.* ***B:*** *Rosette cataract.* ***C:*** *Absorbed cataract with advanced glaucomatous cupping in a patient with old history of trauma.* ***D:*** *Siderotic cataract with raised IOP, corneal scar and iris hole with foreign body in vitreous cavity and optic nerve head cupping on ultrasonography in a patent with old trauma which was neglected.*

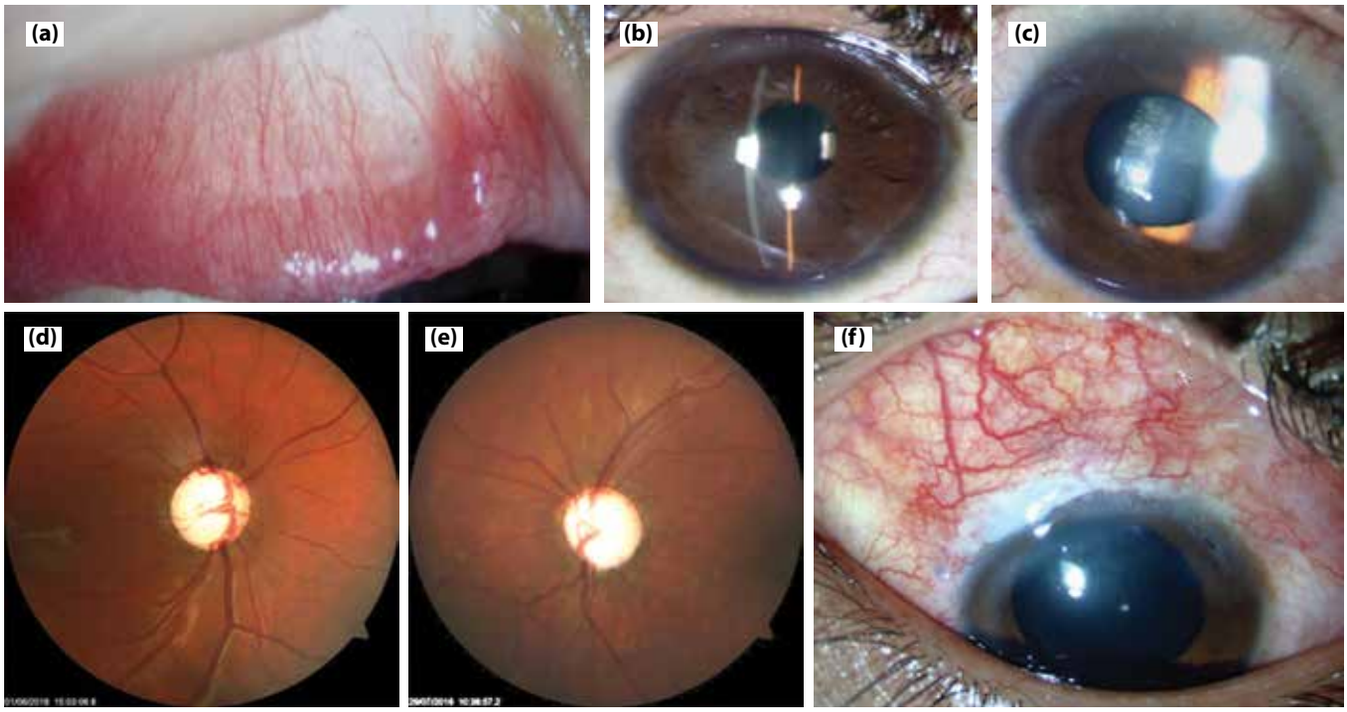

Figure 6: *Steroid induced glaucoma in a 14 year old patient suffering from vernal keratoconjunctivitis.* ***A:*** *Papillae in superior palpebral conjunctiva.* ***B:*** *Pseudogerontoxon and pseudophakia in right eye.* ***C:*** *Pseudogerontoxon and steroid induced posterior subcapsular cataract in left eye.* ***D and E:*** *Advanced glaucomatous cupping in both eyes.* ***F:*** *Left eye required trabeculectomy, Right eye IOP is controlled with medical treatment.*

4. Lens displacement
5. Angle recession more than 180 degrees[15]

Figure 5 shows the variable presentations of traumatic glaucoma with traumatic cataract.

MANAGEMENT

Treatment for hyphaema includes conservative management in majority of the cases (Figure 2):

- Propped up posture in a hyphaema patient helps by following mechanisms:

a. Allows circulating RBCs to settle inferiorly
b. Faster drainage of hyphaema
c. Limits corneal endothelial blood staining.
d. Early evaluation of posterior segment
e. Faster improvement in vision

- Topical steroids and cycloplegics- to reduce anterior chamber inflammation and to minimize the discomfort related to traumatic iritis
- To avoid aspirin/ non-steroidal anti-inflammatory drugs
- Antifibrinolytic agents can be given in high risk cases to reduce rebleed. Aminocaproic acid- 50mg/kg every 4 hrs (max 30g/day) for 5 days. Side effects can be nausea, vomiting, postural hypotension, rarely lethargy, skin rash.

Surgical management is required in about 5% of the hyphema cases[16] and the indications for surgery include:

1. IOP >50 mm Hg for 5 days, >35 mm Hg for 7 days, >60 mm Hg for 24-48 hours
2. Total hyphema not resolving by day 5
3. Grade 3 hyphemas (>50% of anterior chamber) with IOP>25 mm Hg for 5 days.
4. Appearance of corneal blood staining.
5. IOP >25-30 mm Hg for >24 hours in patients with sickle-cell trait[16]

If possible, evacuation of blood should be delayed until the fourth day because at this time, the clot is somewhat retracted and less adherent to the surrounding tissues. Techniques include paracentesis and anterior chamber washout and in cases of clot, clot expression using viscoelastic through an adequate sized incision or by a vitrectomy cutter.

STEROIDS

These are a group of anti-inflammatory drugs, used to treat many ocular and systemic inflammatory conditions, but unmonitored use can lead to cataract and glaucoma formation. Ocular hypertensive response to steroids depends upon the mode of use, potency of steroid, individual variability (depending on low, intermediate or high responders) etc. Patients with primary open angle glaucoma, high myopia, very young age (<10 years), pigment dispersion syndrome, traumatic angle recession are more prone for high steroid responsiveness and the hypothesis is that with already compromised trabecular meshwork, outflow in these cases is further attenuated because of steroid induced trabecular meshwork swelling and damage[17-21]. Steroid hypertensive response is more common with topical use as drops or ointments applied directly to the eye or around the eyelids, periocular injections like posterior subtenon kenacort, intravitreal steroid therapy, inhalational route etc.[22,23] Oral use is less likely to cause steroid responsiveness. Steroid responsiveness usually occurs within first few weeks of use, however, it may occur even after years of use, so it's very important to detect the steroid responsiveness in a particular patient in first 2 weeks after steroid use and then at regular intervals. In cases with only IOP rise and without disc damage or early damage, only discontinuation of steroids can bring down the IOP to normal, however, with long-term use and advanced disc damage, medical and/or surgical treatment might be required because of irreversible trabecular meshwork damage (Figure 6,7). In children, the most common cases to present with steroid responsiveness are vernal keratoconjunctivitis, nephrotic syndrome, chronic uveitis (Juvenile

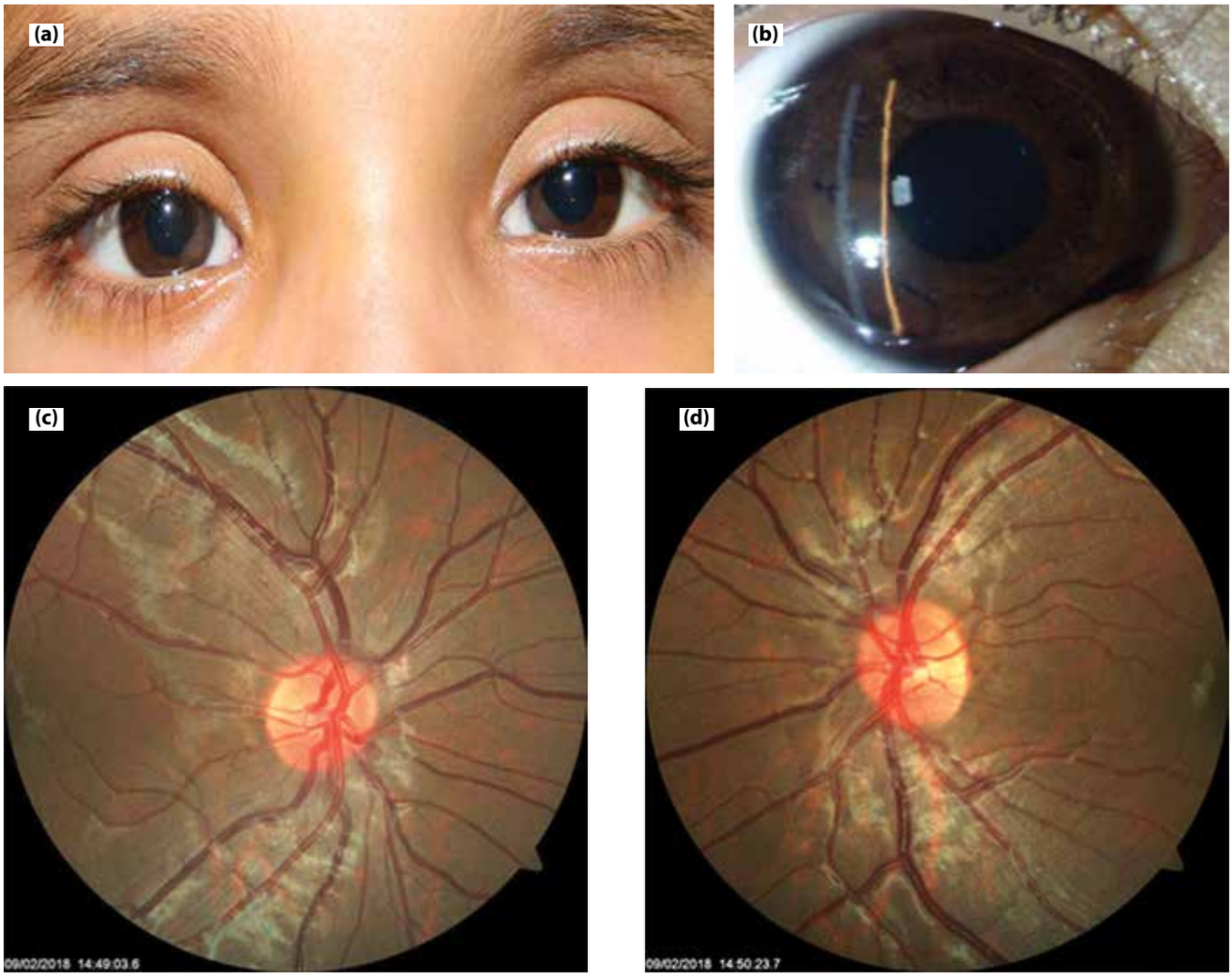

Figure 7: *Steroid induced ocular hypertension in a 7 year old girl due to steroid use after bilateral squint surgery.* ***A:*** *Residual esotropia.* ***B:*** *Normal anterior segment with clear crystalline lens.* ***C and D:*** *Healthy optic discs in both eyes. Patient had high IOP of 40 and 46 mm Hg in right and left eyes respectively and on discontinutaion of steroids only, IOP returned back to 16 and 18 mm Hg within 2 weeks.*

idiopathic arthritis). In such cases, it's very important to monitor these patients regularly and to replace high potency steroids like betamethasone, prednisolone, and dexamethasone with nonadrenal steroids like rimexolone, loteprednol etabonate, fluorometholone and one should consider steroid sparing agents to prevent cataract formation or irreversible glaucomatous disc damage. In a study, 24% of secondary glaucoma cases were due to steroid induced out of which 44% cases were managed by stopping steroids only, 36% were controlled by medical therapy and 19.4% required surgical intervention[24].

UVEITIS

Prevalence of glaucoma in uveitis patients is variable ranging from 5-25%[25-27]. Juvenile idiopathic arthritis (with ANA positivity) has been found to be the most common cause[28]. Severity of glaucoma in uveitis depends upon the type of uveitis, response to treatment, duration of the disease, steroid therapy. The mechanism of increased IOP is complex in uveitis patients. It can be because of chronic trabeculitis leading to sclerosis of trabecular meshwork canals or it can be steroid induced. Acute exacerbation of uveitis can present with low IOP because of ciliary body shutdown and with treatment, as ciliary body function starts, IOP can rise because of compromised trabecular meshwork with longstanding uveitis or steroid responsiveness[29]. It can be an open angle or a closed angle glaucoma. Angle closure glaucoma can occur because of peripheral anterior synechiae or seclusio pupillae leading to iris bombe formation.

Management of uveitic glaucoma is difficult because of the various mechanisms involved in its pathogenesis.

Prostaglandin analogues are usually contraindicated due to increase of inflammation with their use. Iris bombe formation due to seclusio pupillae requires urgent laser iridotomy (Figure 8A&B). In such cases, laser iridotomies need to be more (≥2) and larger in size as they have propensity to close because of inflammation and it's difficult sometimes to make big iridotomy, as there are high chances of bleed with inflamed tissues. Refractory glaucoma requires surgical management. Trabeculectomy with mitomycin-C (MMC) is more likely to fail in such cases because of inflammation, thus glaucoma drainage devices might be considered as a primary procedure (Figure 8C&D)[30]. Goniotomy has also been tried in such cases with studies reporting good outcome in upto 70% of the cases and poor prognostic factors for failure have been found to be older age at surgery, longer duration of glaucoma, greater clock hours of peripheral anterior synechiae and aphakia[31].

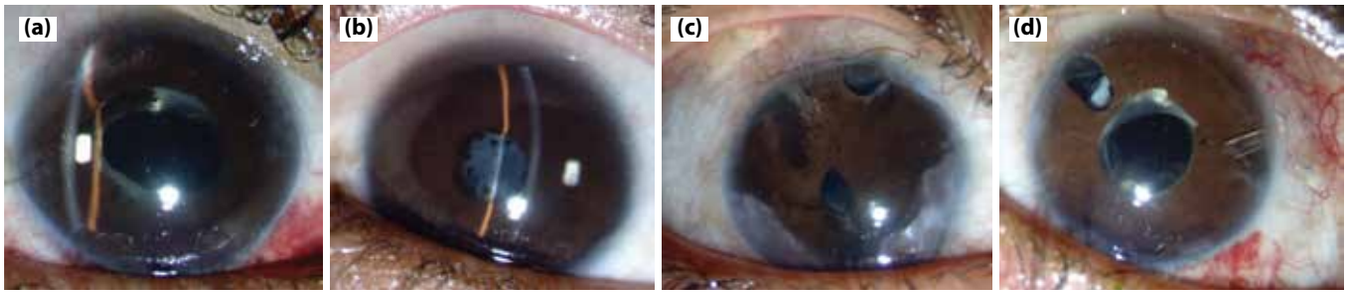

Figure 8: A and B: *A young patient of 16 year age with a known case of Juvenile idiopathic arthritis (JIA) having iris bombe in the right eye after cataract surgery (A), other eye with posterior synechiae and complicated cataract (B). Right eye IOP was 28 mm Hg, iris bombe was treated by laser iridotomy with control of IOP on one topical drug for short-term use.* ***C: and D:*** *Another uveitis patient 16 year old suffering from JIA with refractory glaucoma which was managed by glaucoma drainage devices in both eyes.*

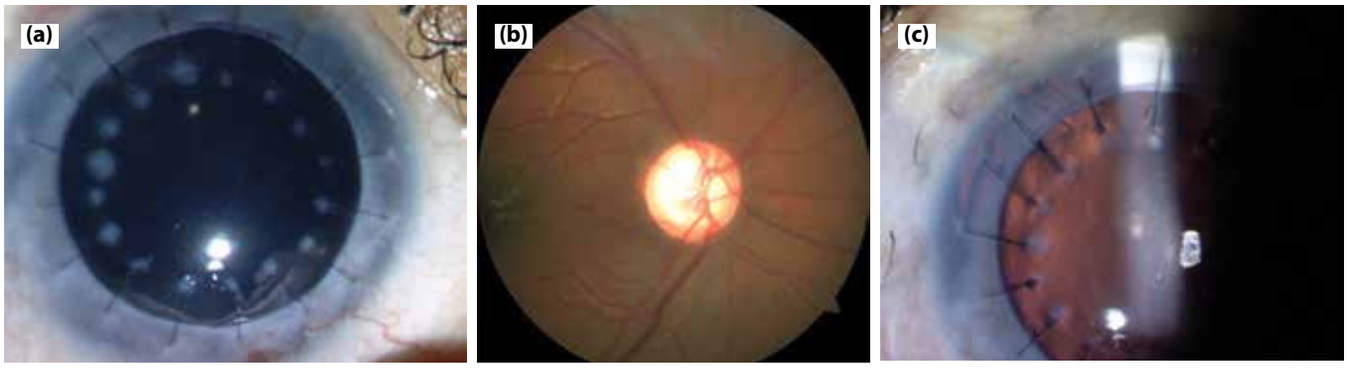

Figure 9: *A 8 year old patient with history of corneal laceration repair presented with raised IOP which was controlled on medical treatment, underwent optical penetrating keratoplasty for corneal opacity.* ***A:*** *Clear corneal graft.* ***B:*** *Advanced glaucomatous optic disc cupping.* ***C:*** *Patient required surgical management for post-keratoplasty uncontrolled IOP (Glaucoma drainage device in anterior chamber visible on retroillumination).*

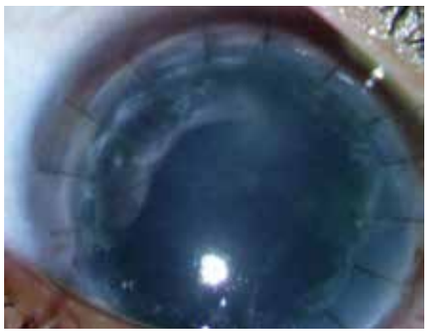

Figure 10: *This patient, 10 year old male child underwent therapeutic penetrating keratoplasty for perforated corneal ulcer. Anterior chamber was shallow with peripheral flat AC and peripheral anterior synechiae with high IOP in range of 50 mm Hg. IOP was controlled on medical treatment first followed by diode laser cyclophotocoagulation in view of decompenstaed graft with visual acuity limited to perception of light inaccurate projection of rays and poor visual potential.*

POST-SURGERY OTHER THAN CATARACT SURGERY

Penetrating keratoplasty: Pediatric penetrating keratoplasty is required for corneal opacity secondary to congenital cause (sclerocornea, corneal opacity in congenital glaucoma, Peters anomaly, aniridia etc), acquired traumatic (post-corneal laceration repair, corneal blood staining) (Figure 9) or non-traumatic corneal opacities (infectious keratitis, keratoconus etc). Peters anomaly and aniridia are more likely to have associated glaucoma and the IOP control gets disturbed after keratoplasty because of surgical trauma and inflammation as well as long-term steroid use after keratoplasty and keratoplasty success rates are also lesser in these cases[32]. Various other factors responsible for post-keratoplasty glaucoma can be: angle closure because of pupillary block by an intact hyaloid phase or peripheral anterior synechiae formation (more common with perforated corneal ulcer cases), (Figure 8) distortion of angle anatomy due to tight sutures or trabecular meshwork collapse in aphakic cases due to loss of ciliary body and lens support[33].

Management includes prophylactic measures before surgery including good control of IOP in pre-existing glaucoma cases and intraoperative precautions like adequate tightness of sutures, intraoperative synechiolysis and careful wound closure to prevent post-operative shallow anterior chamber. Treatment includes medical treatment first and the options include β-blockers, alpha-agonists systemic carbonic anhydrase inhibitors (for short-term use after surgery). The carbonic anhydrase inhibitors should be used with caution as they can inhibit carbonic anhydrase enzyme in corneal endothelium and disturb the endothelial function. Prostaglandins can increase the inflammation and can cause cystoid macular edema or reactivation of herpetic keratitis, so should be used with caution after acute inflammatory period is over. Miotics should be avoided as they can increase inflammation and there is risk of graft rejection. Topical steroid use should also be monitored and steroid sparing agents like cyclosporine or tacrolimus should be preferred in cases with steroid responsiveness. Surgical treatment options include trabeculectomy with MMC, glaucoma drainage devices and caution is required in such cases to prevent anterior chamber shallowing so as to prevent endothelial cell loss and glaucoma drainage device should be placed as far as possible from the graft. Cyclodestructive procedures might be required for recalcitrant cases with poor visual potential (Figure 10).

After retinal detachment surgery: Intraocular pressure can rise both after scleral buckling or vitrectomy. Scleral buckle leads to secondary angle closure due to obstruction to the drainage of vortex veins leading to swelling and anterior rotation of ciliary body which pushes the lens-iris diaphragm forward. It usually improves spontaneously over several days-weeks and requires short-term use of antiglaucoma medications.

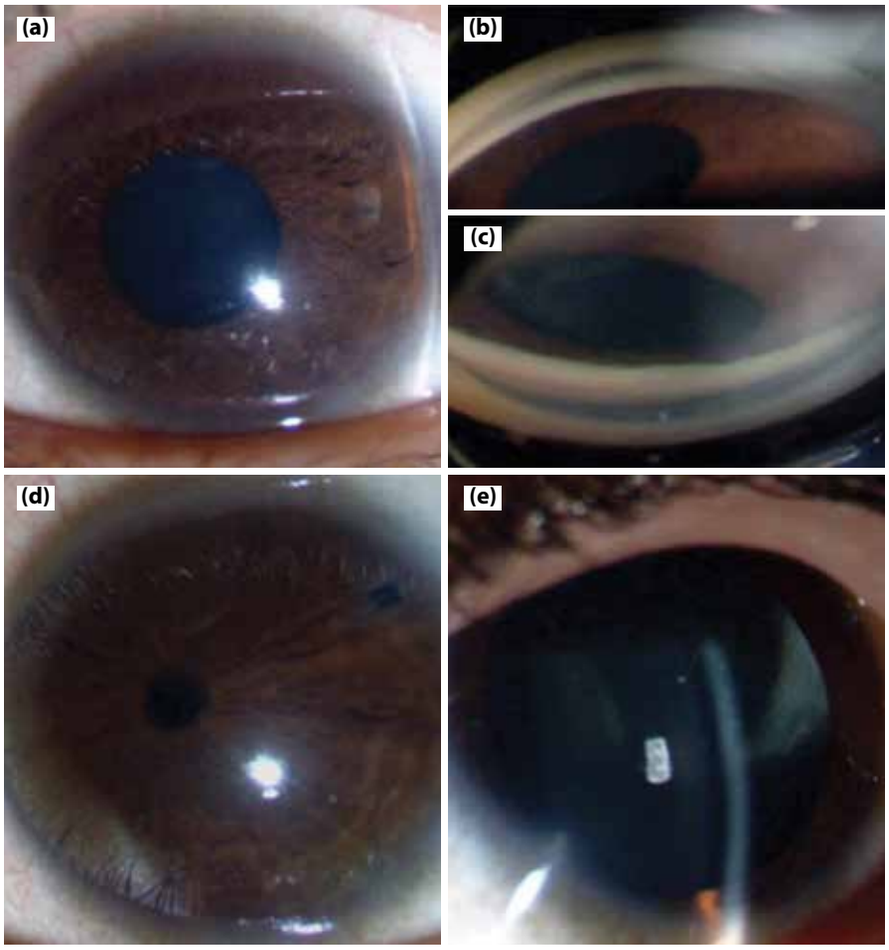

Figure 11: *A 18 year male patient with old history of retinopathy of prematurity and laser treatment for the same, presented with diminution of vision and ocular pain.* ***A:*** *Patient had shallow anterior chamber with IOP of 60 mm Hg.* ***B: and C:*** *Gonioscopy showed closed angles* ***D:*** *After controlling the IOP with medical treatment, laser iridotomy was done with control of IOP with gradual discontinuation of antiglaucoma drugs.* ***E:*** *Dilated examination showing a retrolental glial mass and shallow central AC with thick crystalline lens; lens thickness could not be measured with A-scan.*

RETINOPATHY OF PREMATURITY

Glaucoma after retinopathy of prematurity can occur in about 2% cases of high-risk prethreshold/threshold ROP and is most likely due to angle closure because of retrolental membrane or swollen lens or ciliochoroidal detachment following laser treatment which can lead to forward movement of lens-iris diaphragm[35,36]. In some cases, it can be open angle type because of inflammation post laser treatment or vitrectomy surgery. It can develop months or years after ROP development or treatment or may also occur in adulthood[37]. A case series has described neovascular/non-neovascular angle closure glaucoma because of neovascularization of angle, retrolental mass or swollen lens. These patients have been found to have longer axial length with shallow anterior chamber and swollen lens (Figure 11). Management includes medical treatment, laser iridotomy, lensectomy in cases of intumescent lens, trabeculectomy, glaucoma drainage devices. Cyclodestructive procedures are done in eyes with poor visual potential.

RETINOBLASTOMA

Retinoblastoma can be a cause of secondary buphthalmos and the mechanisms for IOP elevation in cases of retinoblastoma includes neovascularization of iris, anterior

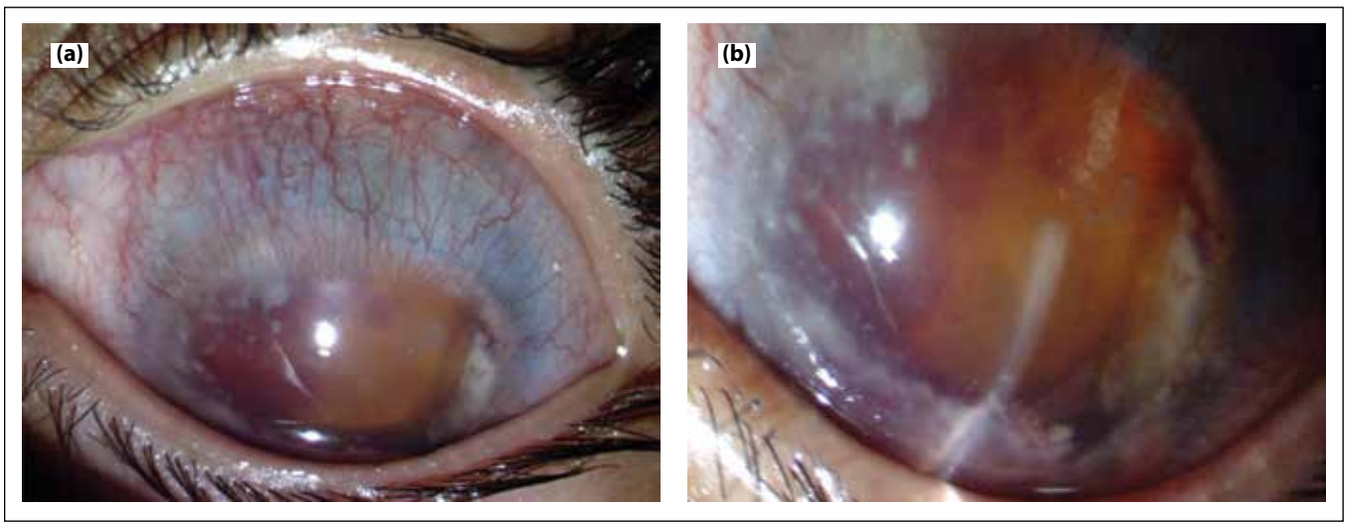

Figure 12: *A 2 year male child of retinoblastoma with secondary buphthalmos and limbal stretching* ***(A)*** *and anterior chamber showing white tumour cells* ***(B)****. CT scan revealed extensive calcification of intraocular mass with optic nerve thickening and spread to the brain.*

After vitrectomy, glaucoma can occur with the use of gas or silicone oil tamponade and the mechanism can be angle closure because of pupillary block by oil/gas bubble or open angle because of gas expansion or overfill by silicone oil or inflammation or steroid use. Angle recession/ trabecular meshwork damage due to previous trauma can also predispose to increased IOP post retinal detachment or macular hole or cataract surgery in traumatic cases[34].

displacement of lens-iris diaphragm or by tumour seeding of trabecular meshwork (Figure 12)[38]. Neovascularization can occur because of angiogenic factors produced by tumour itself or vascular endothelial growth factor produced by

hypoxic retinal cells[39]. Seeding of tumour cells can be seen as pseudohypopyon. Presence of pseudohypopyon suggests extraocular spread of tumour because of drainage through angle of anterior chamber. Patients usually present with pain, photophobia, watering and vomiting in extreme cases. Thorough examination under anaesthesia, ancillary investigations including ultrasonography is must to rule out intraocular mass. CT scan to detect the calcification or MRI orbit and brain are required to detect optic nerve or brain involvement in advanced cases. It's very important to be aware of the association and need of collaborated work between glaucoma specialist and oculoplastic/ retinoblastoma specialists to take care of this potentially life-threatening disease. It's very important to rule out retinoblastoma in every case of congenital glaucoma by ultrasonography before going ahead with the surgery.

REFERENCES

1. Durnian JM, Cheeseman R, Kumar A, Raja V, Newman W, Chandna A. Childhood sight impairment: a 10year picture. Eye (Lond) 2010; 24: 112–7.
2. Bhattacharjee H, Das K, Borah RR, et al. Causes of childhood blindness in the northeastern states of India. Indian J Ophthalmol. 2008; 56: 495–9.
3. Dorairaj SK, Bandrakalli P, Shetty C, RV, Misquith D, Ritch R. Childhood blindness in a rural population of southern India: prevalence and etiology. Ophthalmic Epidemiol. 2008;15:176–82.
4. Beck AD, Chang TCP, Freedman SF. "Definition, Classification, Differential Diagnosis." Childhood Glaucoma: Consensus Series 9. Weinreb RN et al. Amsterdam: Kugler, 2013.
5. Hoguet A, Grajewski A, Hodapp E, Chang TCP. A retrospective survey of childhood glaucoma prevalence according to Childhood Glaucoma Research Network Classification. Indian J Ophthalmol. 2016;64:118-23.
6. Kaur S, Dhiman I, Kaushik S, Raj S, Pandav SS. Outcome of ocular steroid hypertensive response in children. J Glaucoma 2016;25:343-7.
7. Papadopoulas M, Cable N, Rahi J, Khaw PT, the BIG Eye Study Investigators. The British infantile and childhood glaucoma (BIG) Eye Study. Invest Ophthalmol Vis Sci. 2007;48:4100-6.
8. Walton W, Von Hagen S, Grigorian R, Zarbin M. Management of Traumatic Hyphema. Surv Ophthalmology. 2002;4:297-334.
9. Coles WH. Traumatic hyphema: an analysis of 235 cases. South Med J. 1968; 61: 813-6.
10. Sellors PJH, Mooney D. Fundus changes after traumatic hyphaema. Brit J Ophthal. 1973;57:600-7.
11. Tumbocorn JA, Latina MA. Angle recession glaucoma. Int Ophthalmol Clin. 2002;126:921-6.
12. Tonjum AM. Intraocular pressure and facility of outflow late after ocular contusion. Acta Ophthalmol 1968;46:886-908.
13. Blanton FM. Anterior chamber angle recession and secondary glaucoma: a study of the after effects of traumatic hyphemas. Arch Ophthalmol 1964;72:39-44.
14. Tesluk GC, Spaeth GL. The occurrence of primary open angle glaucoma in the fellow eye of patients with unilateral angle-cleavage glaucoma. Ophthalmology 1985;92:904-11.
15. Alper MG. Contusion angle deformity and glaucoma. Arch Ophthalmol. 1963;69:455-67.
16. Macsai M. Surgical management and rehabilitation of anterior segment trauma. In: Krachmer J,Mannis M, Holland E ed. Cornea.2nd ed. Volume 2.Philadelphia,PA: Elsevier Mosby; 2005.
17. Armaly M F. Effect of corticosteroids on intraocular pressure and fluid dynamics. II. The effect of dexamethasone in the glaucomatous eye. Arch Ophthalmol 1963;70:492-9.
18. Biedner B-Z, David R, Grudsky A, et al. Intraocular pressure response to corticosteroids in children. Br J Ophthalmol. 1980;64:430–1.
19. Podos SM, Becker B, Morton WR. High myopia and primary open-angle glaucoma. Am J Ophthalmol 1966;62:1038-43.
20. Becker B, Podos SM. Krukenberg's spindles and primary open angle glaucoma. Arch Ophthalmol 1966;76:635-9.
21. Spaeth GL. Traumatic hyphema, angle recession, dexamethasone hypertension and glaucoma. Arch Ophthalmol 1967;78:714-21.
22. Zugerman C, Saunders D, Levit F. Glaucoma from topically applied steroids. Arch Dermatol 1976;112:1326-41.
23. Cubey RB. Glaucoma following the application of corticosteroid to the skin of the eyelids. Br J Dermatol 1976;95:207-8.
24. Kaur S, Dhiman I, Kaushik S, Raj S, Pandav SS. Outcome of ocular steroid hypertensive response in children. J Glaucoma 2016;25:343-7.
25. Da Mata AP, Foster CS,. Ahmed valve and uveitic glaucoma. Int Ophthalmol Clin. 1999;39:155-67.
26. Papdopoulas M, Cable N, Rahi J, Khaw PT. BIG Eye Study Investigators. The British Infantile and childhood glaucoma (BIG) Eye Study. Invest Ophthalmol Vis Sci. 2007;48:4100-6.
27. Paroli MP, SPenza S, Marino M, Pirraglia MP, Pivetti-Pezzi P. Prognosis of juvenile rheumatoid arthritis-associated uveitis. Eur J Ophthalmol. 2003;13:616-21.
28. Sijssens K, Rothava A, Berendschot TTJM, De Boer JH. Ocular hypertension and secondary glaucoma in children with uveitis. Ophthalmology 2006;113:853-9.
29. Kaur S, Kaushik S, Pandav SS. Pediatric Uveitic Glaucoma. J Curr Glaucoma Pract. 2013;7:115-7.
30. Sung VC, Barton K. Management of inflammatory glaucoma. Curr Opin Ophthalmol. 2004;15:136-40.
31. Ho CL, Walton DS. Goniosurgery for glaucoma secondary to chronic anteriorJ Glaucoma 2004;13:445-9.
32. McClellan K, Lai T, Grigg J, Billson F. Penetrating keratoplasty in children: visual and graft outcome. Br J Ophthalmol. 2003;87:1212-4.
33. Dada T, Aggarwal A, Minudath KB, Vanathi M, Choudhary S, Gupta V, Sihota R, Panda A. Post-penetrating keratoplasty glaucoma. Indian J Ophthalmol. 2008;56:269-77.
34. Gedde SJ. Management of glaucoma after retinal detachment surgery. Curr Opin Ophthalmol. 2002;13:103-9.
35. Bremer DL, Rogers DL, Good WV, Tung B, Hardy RJ, Fellows R. Glaucoma in early treatment for Retinopathy of Prematurity (ETROP) study. J AAPOS. 2012;16:449-52.
36. Modzejewska M, Lachowicz E, Kubasik-Kladna K, Tokarz- Sawinska E, Kiedrowicz R. Glaucoma after laser diode treatment in retinopathy of prematurity- case series. Klin Oczna. 2014;116:39-43.
37. Michael AJ, Pesin SR, Katz LJ, Tasman WS. Management of late-onset angle-closure glaucoma associated with retinopathy of prematurity. Ophthalmology 1991;98:1093-8.
38. Shields CL, Shields JA, ShieldsMB, Augsburger JJ. Prevalnce and mechanism of secondary intraocular pressure elevation in eyes with intraocular tumours. Ophthalmology 1987;94:839-46.
39. Folkman J. Tumour angiogenesis factor. Cancer Res. 1974;34:2109-13.

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