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Evaluation of Posterior Segment Manifestations Amomg Children Complaining Visual Disturbances

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Abstract: *Posterior segment Ocular manifestations among children are diverse and global. At this age, eye lesions are often unnoticed because of the incapacity to express eye discomfort and lack of availability of experienced posterior segment eye specialists. The purpose of this study is to describe posterior segment ocular manifestations among children and hence associated factors in the Department of Ophthalmology at the JLN Hospital, Ajmer (Rajasthan) which is a tertiary eye care centre in central Rajasthan, India. This is prospective study conducted between 1st September 2015 and 30th march 2017. A complete ophthalmic examination was done to all children attending the Retina clinic, as part of their routine medical visit.*

Key Words: *Fundus changes in children, Congenital retinal disorders, Berlin's edema*

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The eyes of children basically does not differ much from that of adults. The principles of tests, clinical approach and interpretation is almost same in all ages with little modification

Refractive error changes- Myopia is the commonest cause of gradual painless diminished vision in paediatric age group. It is strong heredity[1]. Congenital myopia is rare in which child is born with eye longer than 24 mm. It is usually unilateral.

Simple myopia is commonest type of refractive error among myopics. It is autosomal dominant in which fundus finding shows temporal crescent and lattice degeneration. Child is on risk of occurrence of retinal detachment

Pathological myopia seen in children present as early and late changes in fundus as myopic crescent, peripapillary crescent, pale and large optic disc, Foster Fuch's spot, lacquer cracks, lattice, snail track or cystoids degeneration, posterior staphyloma, posterior vitreous detachment, vitreous opacities, liquefaction of vitreous etc.

Hypermetropia is commonest refractive error in children under 5 years of age. Overall fundus size is small with small optic disc called pseudo neuritis, and background appear as short silk appearance[2].

Uveitis – About 5% of all uveitis is seen in paediatric age group. Anterior uveitis may present with diffuse retinal oedema , cystoid macular oedema or optic neuritis on fundoscopy. Intermediate uveitis present with CME and snow banking[3]. In posterior uveitis colour of area involved is greyish or yellowish. Vitreous floaters, vitreous flare, PVD, retinitis, vasculitis, papillitis, exudative retinal detachment may be present.

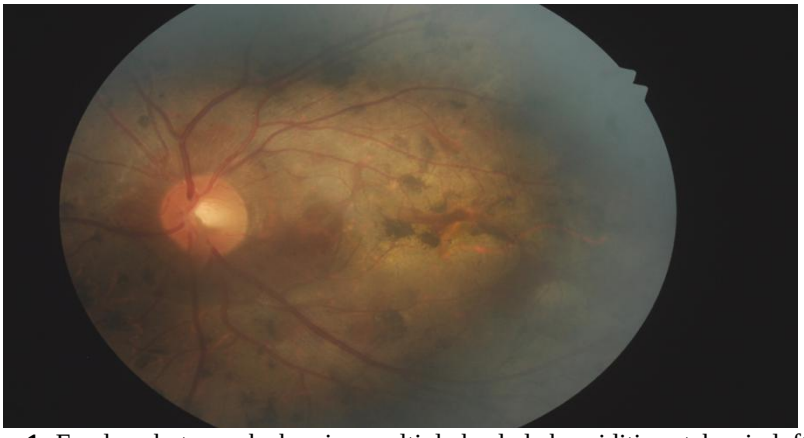


Figure 1: Fundus photograph showing multiple healed choroiditis patches in left eye

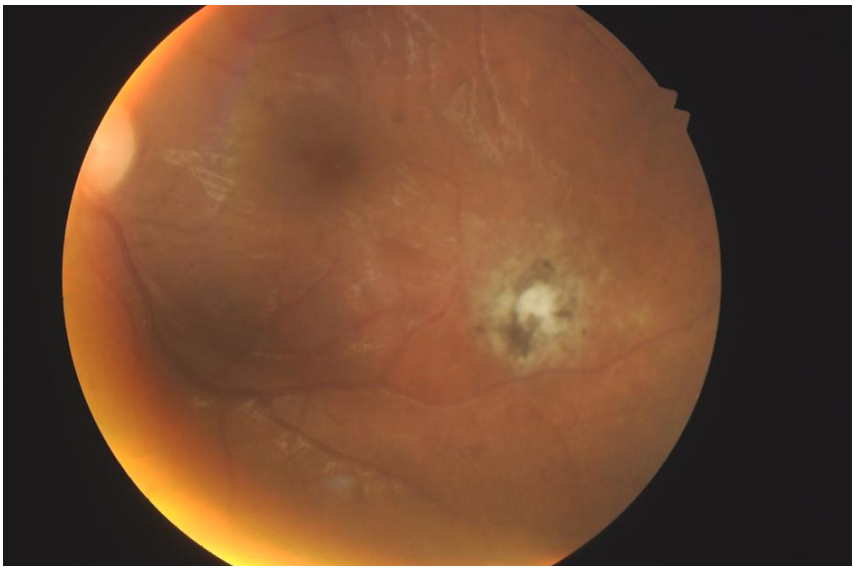


Figure 2: Fundus Photo of other patient showing a single healed choroiditis patch at posterior pole near macula.

Endophthalmitis is a severe form of pan uveitis with involvement of vitreous, retina and optic nerve. The inflammation is always intra ocular. Commonest source of infection is penetrating injury either accidental or surgical. It can acute or chronic; it may be infective or sterile.

Coloboma of choroid - Can be typically situated at site of foetal fissure. Coloboma of macula may be small or larger than optic disc. It is punched out horizontal oval area with clumps of pigments on periphery and a white floor of sclera[4].

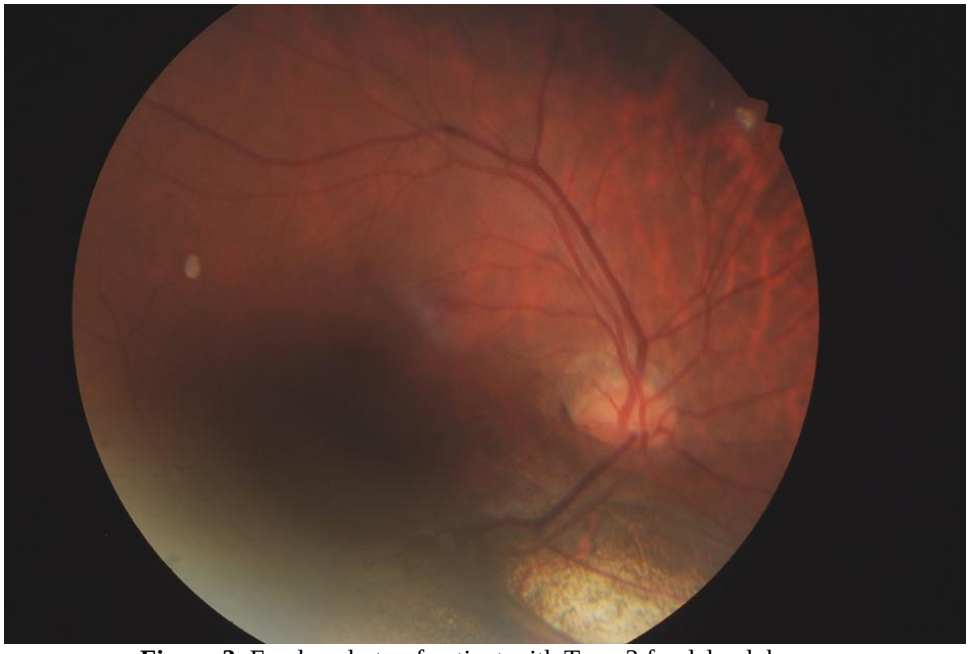


Figure 3: Fundus photo of patient with Type 3 fundal coloboma

Ida Manns classification(1937) of Fundal Coloboma

1-Above the optic disc
2-Superior border of optic disc
3-Seperated from the optic disc by normal narrow area of retina
4-Inferior crescent below the disc
5- Isolated gap in the line of fissure
6-Area of pigmentary disturbance
7-Extreme peripheral colobom

Glaucoma- Prevalence of childhood glaucoma is far less than adult. It could be congenital, developmental and secondary. In these cases it could be angle closure or wide angle glaucoma[5,6]. One way to classify glaucoma is based on the age of onset. Congenital glaucoma is present at birth. Infantile glaucoma develops between the ages of 1-24 months. Glaucoma with onset after age 3 years is juvenile glaucoma. Another way to classify glaucoma is to describe the structural abnormality or systemic condition which has caused the glaucoma.
Most cases of pediatric glaucoma have no specific identifiable cause and are considered primary glaucoma. When glaucoma is caused by, or associated with a specific condition or disease, it is called secondary glaucoma. Examples of conditions which can be associated with childhood glaucoma include Axenfeld-Reiger Syndome, aniridia, Sturge-Weber Syndrome, neurofibromatosis, chronic steroid use, trauma, or previous eye surgery such as childhood cataract removal. Not all patients with these conditions will develop glaucoma, but their incidence of glaucoma is much higher than average and they should be monitored regularly.

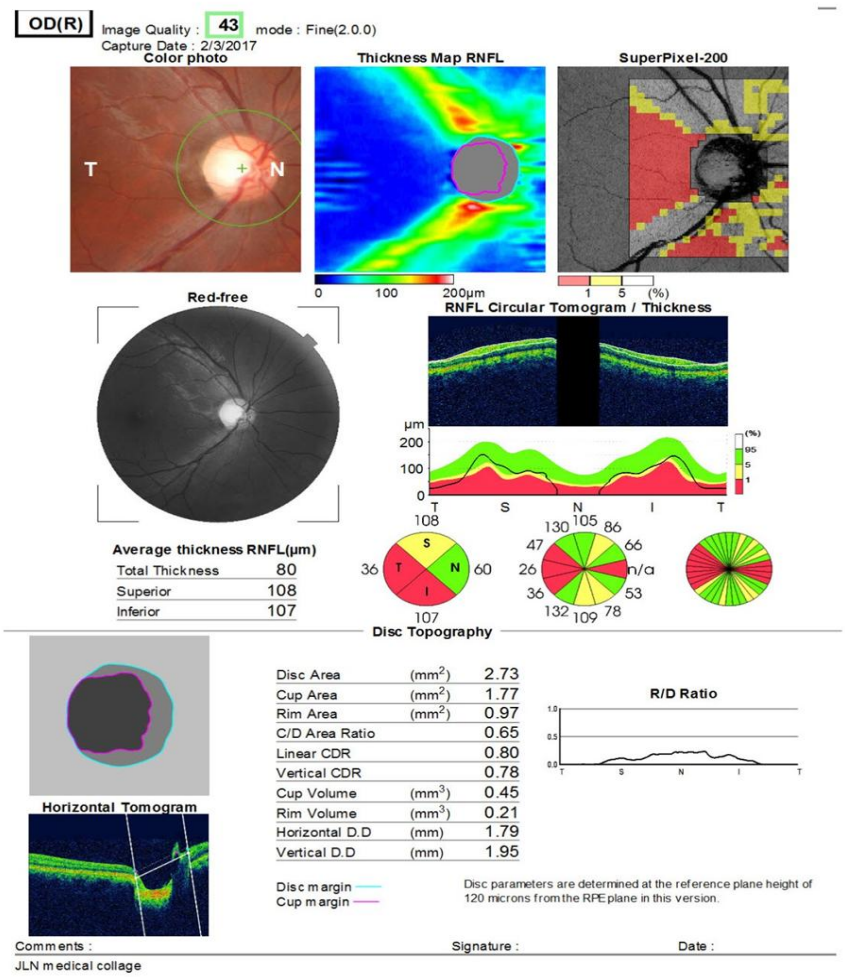


Figure 4: OCT picture for 3D optic disc assessment of patient with glaucoma showing marked thinning of neuroretinal rim in inferior and temporal quadrant and borderline changes in superior quadrant

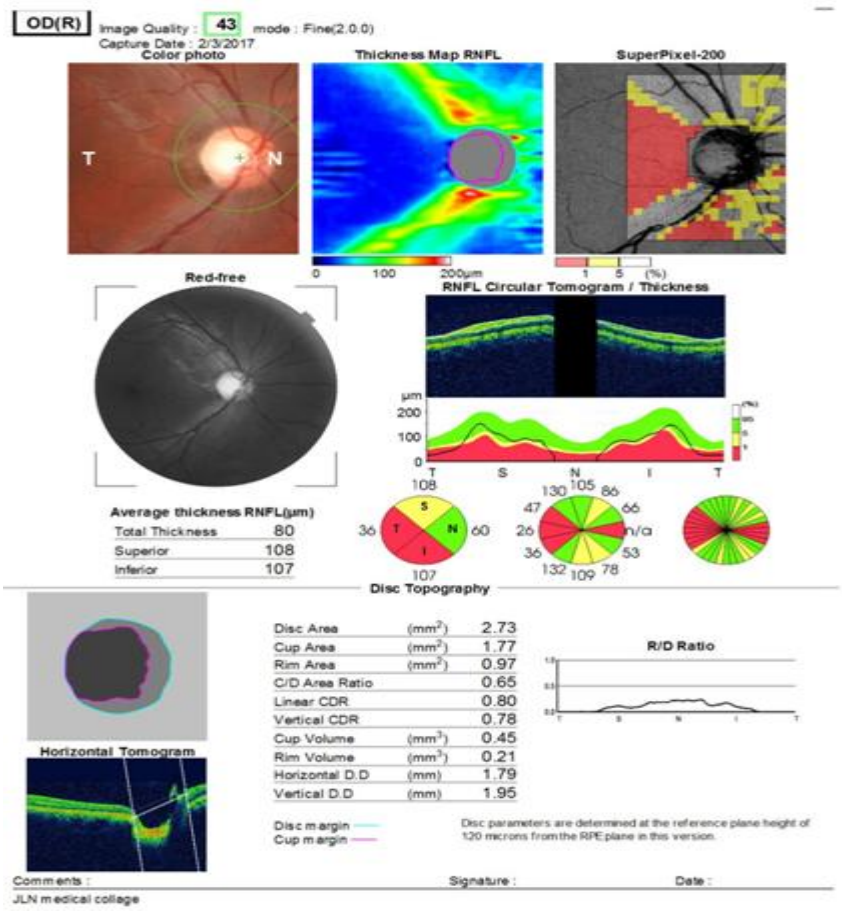


Figure 5: OCT picture of macula showing Retinal Nerve Fibre Layer Thinning

Albinism- On fundus examination retinal and choroidal vessels stand out prominent against sclera, macula is hypoplastic and optic nerve head cannot be differentiated[7].
Retinal gliosis- Proliferation of glial tissue in excess of normal limit[8].
Retinal folds- Caused due to proliferation of inner layer of optic vesicle. Common site being inferior temporal retina[9].
Retinal cyst- Retinal dysplaia or microphthalmia may be associated with retinal cyst. They are precursor of retinal dialysis or retinoschisis[10].
Congenital RD- It takes place when two layers of optic cup fail to come in intimate proximity due to unequal rate of growth of two layers. It is generally seen in periphery. May be caused by intrauterine inflammation[11].
Congenital retinoschisis- Split in sensory retina at the level of outer plexiform layer. Late stage retinoschisis is termed as juvenile or idiopathic retinoschisis[12].
Congenital retinal degeneration and dystrophies. Almost all degeneration/dystrophies are congenital in nature, bilateral and almost symmetrical.
Congenital functional defects of vision due to anomalies of retina are-
Congenital night blindness
Congenital day blindness
Congenital colour blindness[13]
Coats disease (primary /congenital retinal telangiectasia)- Also known as Lebers **military** aneurysm. Peripheral lesion are more common in temporal retina . both arteries and veins are involved. In 80% it is unilateral. In retinal vessels there is bunches of beading, kinking and loop formation. Vessel are tortuous may show aneurysm. Neovascularisation may occur. In retina there is greenish white or yellowish white areas of exudates with shining spots of cholesterol crystals. There may occur exudation of sub retinal space and retinal detachment and vitreous haemorrhage[14].
Retinal angiomatosis (Von hippel Lindau Disease) – The angioma formed is hamartoma(phocomatosis). Lesion develops from retinal capillaries and endothelium of retinal vessels. It is autosomal dominant trait. There could be macular scar, retinal detachment, retinal and vitreous haemorrhage, secondary glaucoma and complicated cataract[15].

Coloboma retinal – Commonest congenital anomaly of macula are macular coloboma. As per appearance it has been classified into –
With normal retinal vessels
With abnormal retinal vessels
Second possible classification –
With pigment
Without pigment[16]

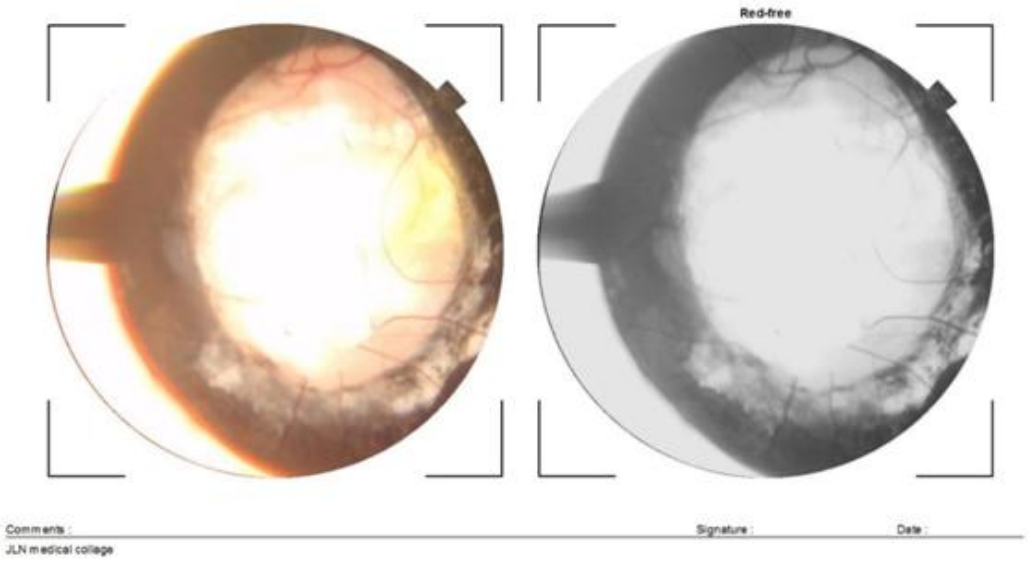


Figure 6: Picture showing retinal coloboma involving optic disc

Retinal haemorrhage – causes are many like traumatic, inflammation, retinopathies, blood disorder etc
Types of retinal haemorrhages
1.Intra retinal -a. Superficial
b. deep
2. Pre retinal – a. Sub hyaloids haemorrhage
b. vitreous haemorrhage
3. Sub retinal haemorrhage – a. Choroidal hematoma.
b.subretinal neovascularisation[17].

Retinal aneurysm – look similar to bunch of deep haemorrhages. Best seen in fundus fluoroscein angiography[18]
Retinitis – inflammation of retina is devided into two types
Primary where retina and retinal vessels are primary site
Secondary, when inflammation spread to choroid and optic nerve.
It could be viral (TORCH, CMV, herpes zoster), Bacterial, and parasitic (toxoplasmosis)[19]

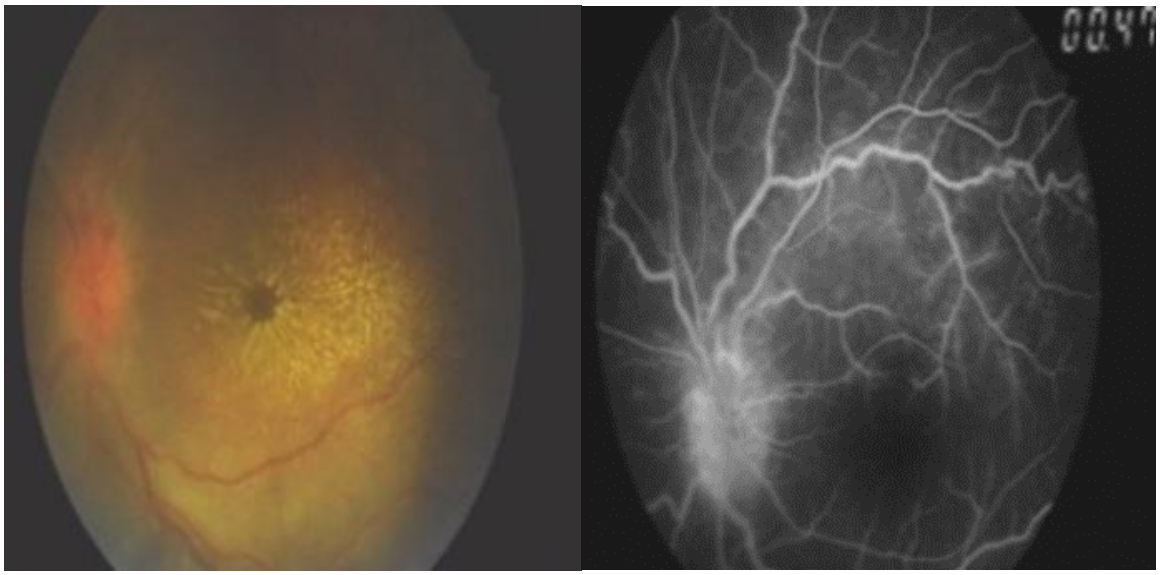
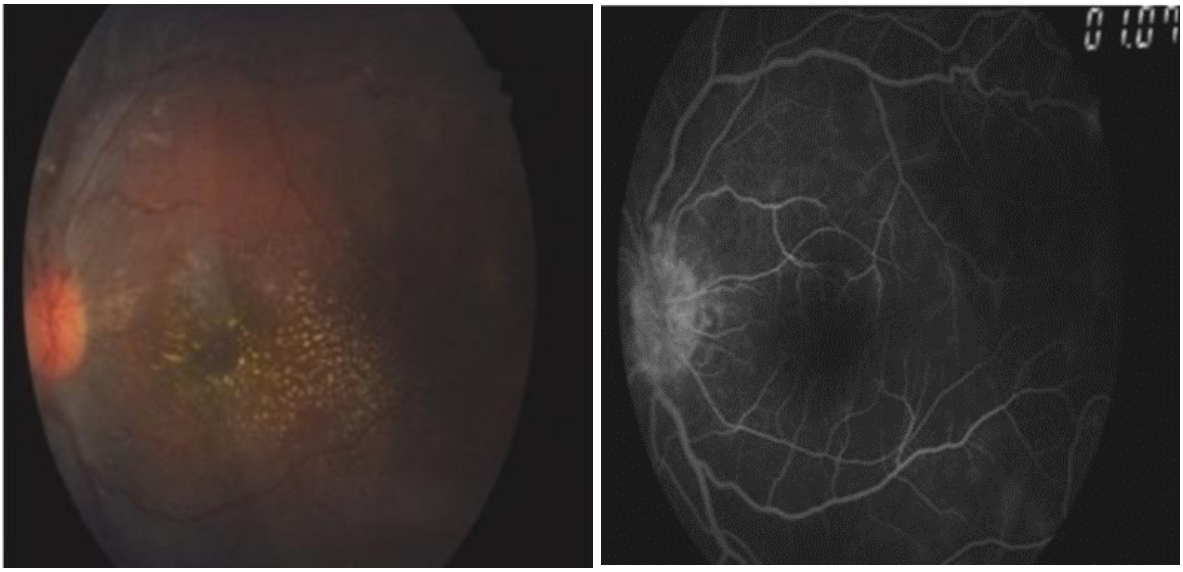


Figure 7: Pre-treatment Fundus Photography and Fluorescein angiography picture of patient of neuroretinitis in a case of Lyme disease

Figure 8: Post treatment pictures of same patient after 4 weeks of treatment



Retinitis pigmentosa – commonest of all hereditary dystrophies. Mostly hereditary, however sporadic may also present. Triad of signs are- Bony specule type pigments, attenuation of vessels, and waxy pale disc. It is characterised by night blindness, cystoids macular edema (CME), atrophic maculopathy, cellophane maculopathy , posterior subcapsular cataract, consecutive optic atrophy, and chronic simple glaucoma[20].

Vitreous floaters- are commonest symptom of disease of vitreous. They cause loss in transparency of the vitreous, may be in the form of scattered dots, strings, or sheets. They are generally mobile[21].

Vitreous degeneration – the liquefaction of vitreous is known as synschisis. In children it is seen in high myopia and trauma with or without retained foreign body. It is also seen following vitreous haemorrhage, endophthalmitis and severe posterior uveitis[22].

Vitreous detachment - for a vitreous to detach its consistency must change towards fluid rather than semi solid gel. Common causes in children are – Myopia, chronic uveitis, trauma, vitreous haemorrhage,vitreous disturbance during intra ocular surgery.

According to configuration of detachment it has been divided into

Posterior vitreous detachment (It is more common) and Anterior vitreous detachment

Posterior vitreous detachment can be: Small peripheral, Infundibular or conical detachment – when attachment round the disc and base but separated from internal limiting layer of retina all around and Globular detachment – when attachment round the disc has been severed but the basal attachment is maintained.[23]

Retinoblastoma – it is most common intraocular malignancy of early childhood. It is uniformly fatal if not treated in time. Commonest mode of presentation in first two age groups is white reflex in pupillary area leucocoria. Second mode of presentation is strabismus which is commonly esotropia. Child may be brought with red and watery eyes, by the time child develops photophobia eye has passed into stage of secondary glaucoma. Proptosis is late presentation[24-27]

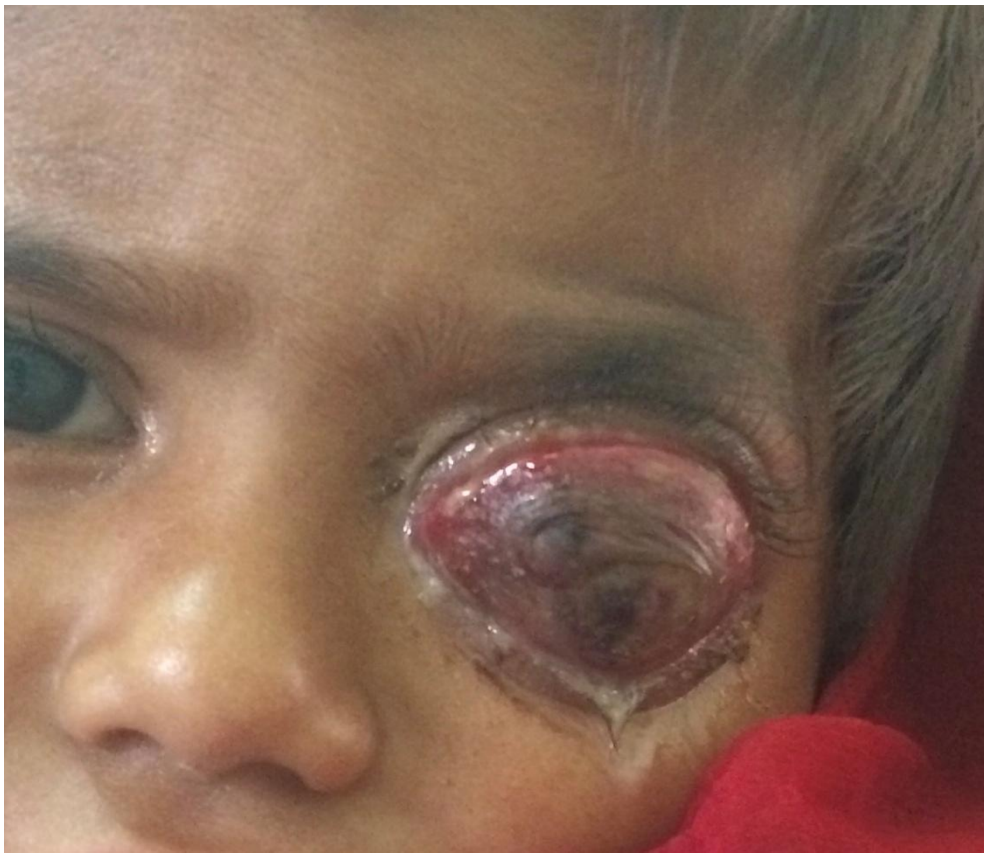


Figure 9: Clinical picture of Retinoblastoma presenting as proptosis

ROP – ROP occurs when abnormal blood vessels grow and spread throughout the retina, the tissue that lines the back of the eye. These abnormal blood vessels are fragile and can leak, scarring the retina and pulling it out of position. This causes a retinal detachment. Retinal detachment is the main cause of visual impairment and blindness in ROP.

Persistent hyperplastic primary vitreous.- this is congenital anomaly of primary vitreous and hyaloid artery. It is due to failure of regression of primary vitreous. This is non hereditary generally uniocular. These eyes are microphthalmic and the child is full term. It is of two types – more common anterior type and rare form of posterior type[29].

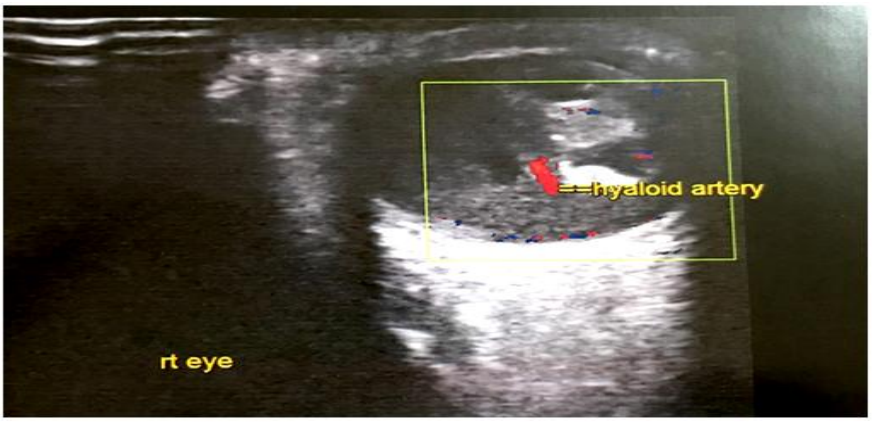


Figure 10: B Scan showing Hyaloid artery in a case of persistant hyperplastic primary vitreous

Myelinated nerve fibres – myelination of anterior visual pathway begins in the lateral geniculate body and stops short at the lamina. Due to some unknown causes in infants myelination extends well beyond the disc. It appears as white, fluffy, irregular patches extending from disc towards retinal periphery[30]

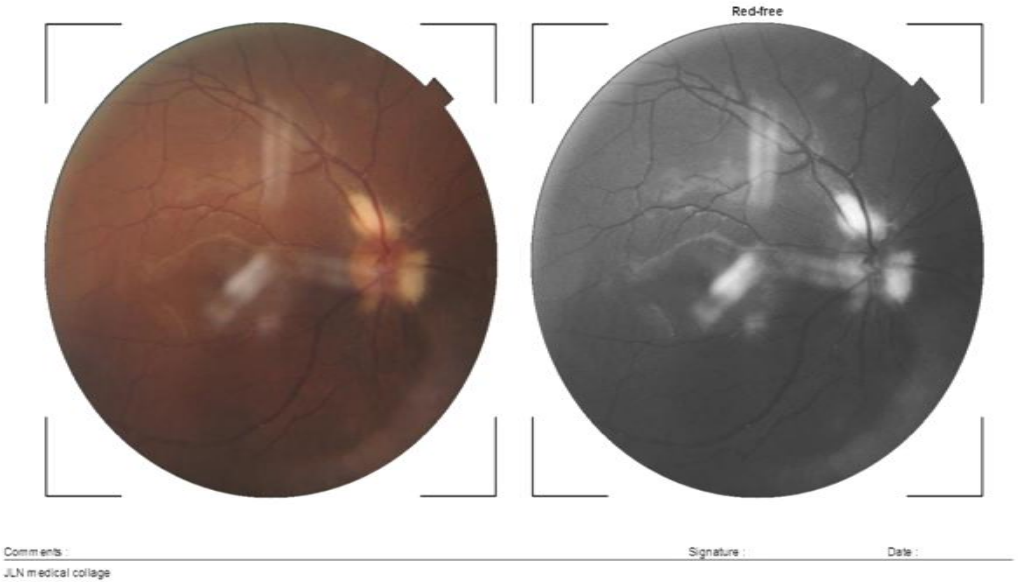


Figure 11: Fundus picture of patient with myelinated nerve fibres around disc.

Congenital coloboma of disc – it may be localised to disc itself or may be associated with uveo retinal coloboma. Cause of both form is faulty closure of embryonal fissure hence situated in lower part of disc.
Papilloedema – it is not a disease but a sign that reflects more serious primary systemic condition mostly intra cranial causing bilateral passive oedema of optic disc. Ophthalmic signs can be divided into following groups
Early papilloedema
Established papilloedema
Late papilloedema
Optic atrophy[31]

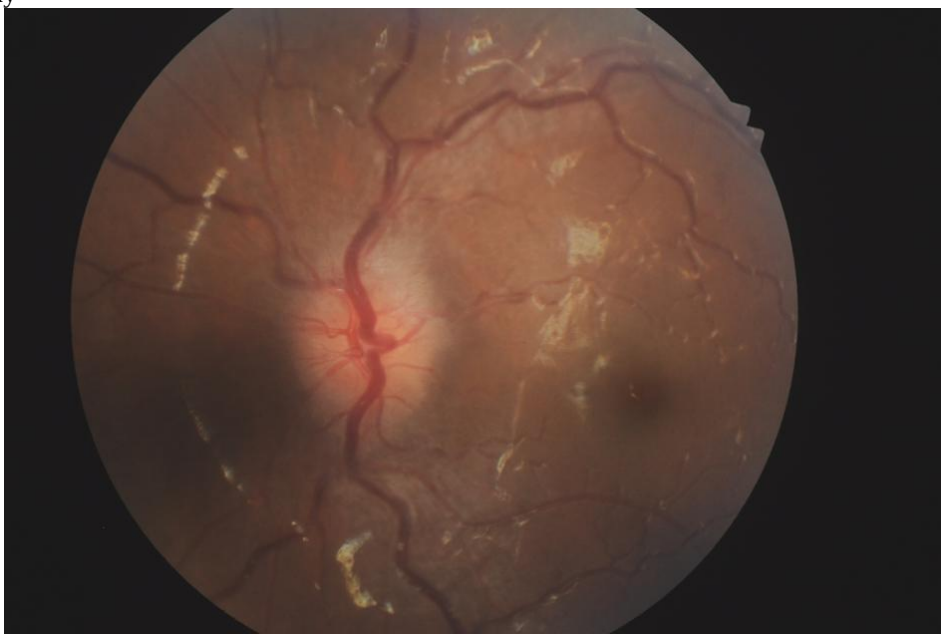


Figure 12: Fundus photo showing disc oedema

Papillitis – it is generally unilateral disc swelling causing rapid fall of vision. Variable degree of disc swelling not more than 3DD. May be normal initially, blurring of dic margins, few flame shaped haemorrhages, vitreous haze is seen over disc.

Optic neuritis – it is non specific term that includes all the conditions, which have loss of central vision, dyschromatopsia and central feild defect, which can be acute and chronic due to involvement of optic nerve from papilla to beginning of the chiasma. Fundus finding are –
Blurring of disc margins and hyperaemia
Late changes are disc pallor
Fully developed neuritis is always associated with the cells in vitreous infront of the disc[32].

Optic atrophy – it is the end result of many diseases where optic nerve is visually a scar with loss of function, occurs due to degeneration of axons in the anterior visual pathway. Anatomically it is divided into ascending and descending type.
On the basis of ophthalmoscopic appearance-
Primary optic atrophy
Secondary optic atrophy – post neuritic or post papilledematous
Consecutive optic atrophy
Cavernous optic atrophy
Glaucomatous optic atrophy
Temporal pallor
Bow tie pallor[33].

Ocular trauma – Non penetrating or blunt trauma, orbital trauma and systemic trauma may cause a variety of posterior segment abnormalities. Blunt trauma may cause damage to retina(commotio retinae), retinal pigment epithelium edema, choroid(choroidal rupture) and optic nerve(optic nerve evulsion)alone or in combination. Traumatic macular hole and retinal detachment or dialysis may also occur. Systemic trauma may result in diffuse retinopathy (purtscher's retinopathy, shaken baby syndrome) or localised retinal abnormalities (whiplash retinopathy, fat embolism syndrome). Alterations in intravascular (Valsalva retinopathy) or intracranial pressure (terson's Syndrome) due to variety of causes may result in preretinal or vitreous hemorrhage and associated visual loss.

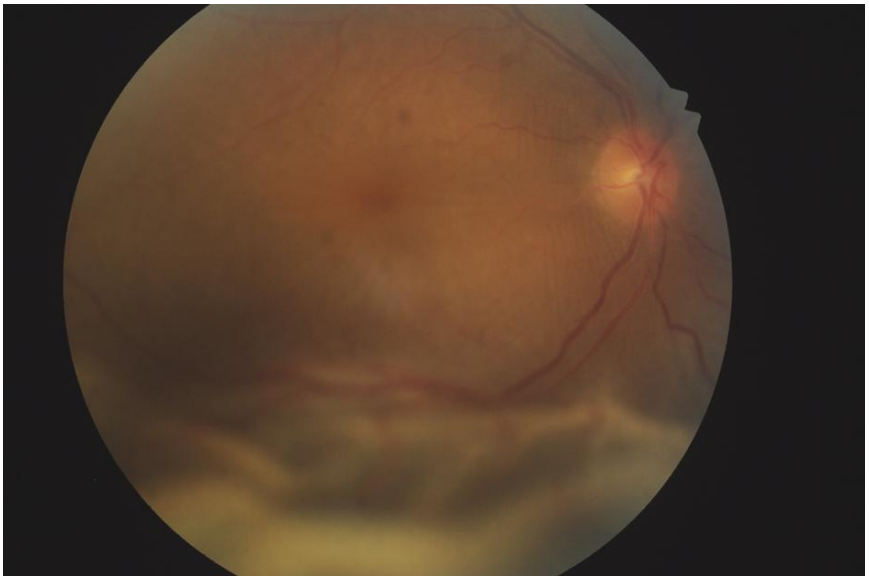


Figure 13: Fundus photo of a patient with trauma showing bullous Reinal Detachment

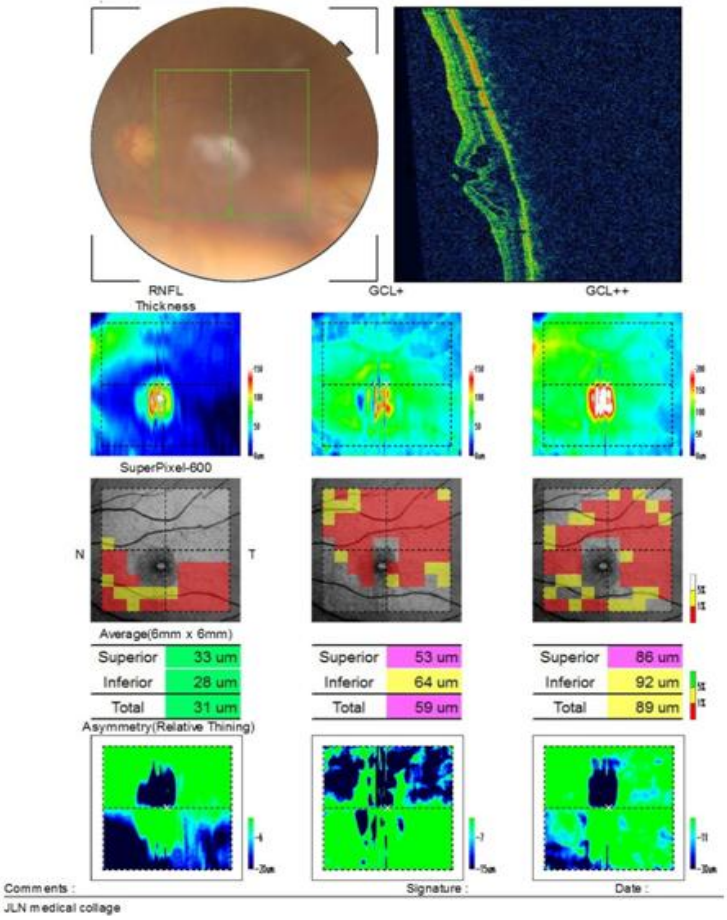


Figure 14: OCT picture of patient with blunt trauma showing Macular Hole

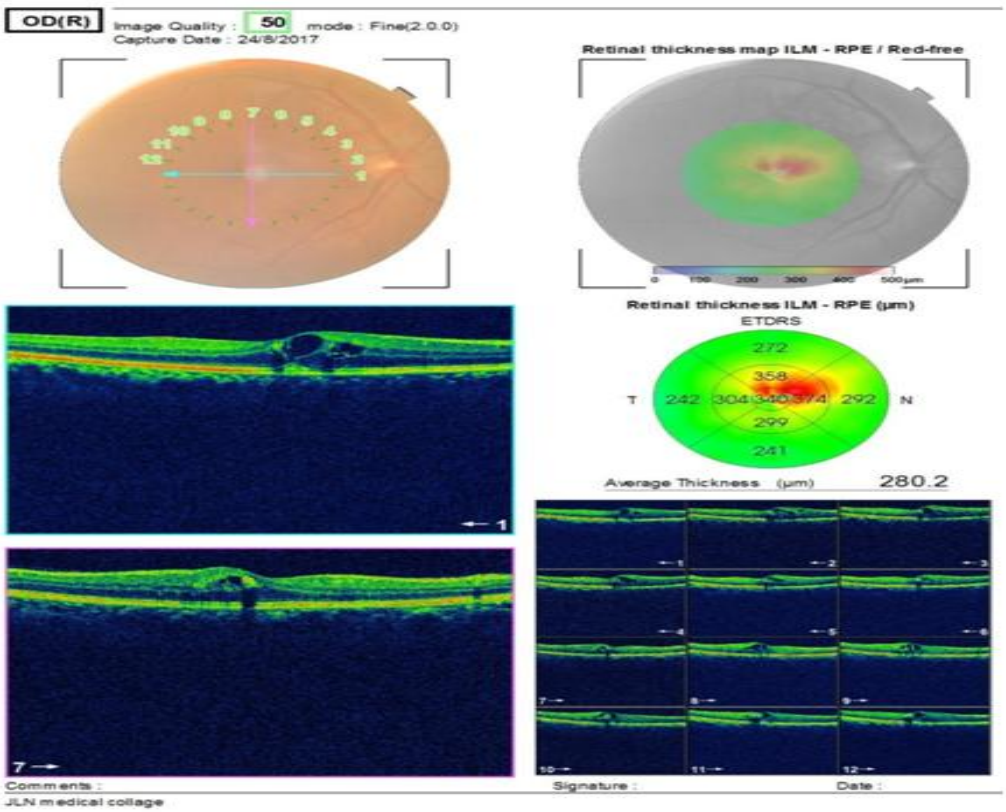


Figure 15: OCT picture of a patient with blunt trauma showing Berlin's edema

I. Material & Methods

The present clinical study was conducted in the Upgraded Department of Ophthalmology, JLN Medical College & Hospital, AJMER (Rajasthan), India which is a tertiary eye care centre in central Rajasthan, India.

All cases from 1st September 2015 to 30th march 2017 under age of 18 years were included in the study. Cases were deleted from the study when patient's fundus examination was not possible or severely debilitated patients which cannot be shifted for fundus examination.

II. Methodology

After taking informed consent, all the subjects were asked a detailed ocular and systemic history and they underwent a thorough ophthalmic examination.

Preliminary eye examination included were visual acuity, Slit lamp biomicroscopy of anterior segment to rule out any corneal pathology and refraction.

Intraocular pressure was recorded using Goldmann applanation tonometry(GAT)/Schiotz tonometer wherever possible

Fundus examination was done using Direct ophthalmoscope, Indirect ophthalmoscope and +90 D mirror.

Gonioscopy was done to rule out closed angle glaucoma wherever possible.

Visual field examination by Humphery field analyser was done to look for visual field defects in elderly children.

Fluorescein Angiography was carried out using a fundus camera (Kowa 10 Xa, Japan) wherever possible and required. After proper consent and dilation of pupil with tropicamide and phenylephrine, 3cc of 20% Na-fluorescein is injected into the antecubital vein and photographs were taken at both early and late phases of angiography.

OCT(Optical Coherence Tomography) was performed through a dilated pupil by 3D OCT – 1 mestro, Topcon). Patients were explained about the procedure and after proper positioning of patients for each eye, scans were taken.

Follow up: All cases were followed up regularly for 6 months wherever required. 3 follow ups were done at 2 months, 4 months & 6 months after initial examination.

III. Results

Our study was done in JLN Hospital, Ajmer, Rajasthan. A total of 12,528 patients presented in our OPD out of which 2160 were screened for posterior segment pathology and of these cases 151 children had positive finding. Following are the results of our study.

The average age wise distribution of posterior segment findings in children is shown in Table 1.

There were 23 children of age <3 months, 18 between 3-12 months of age, 29 between 1-5. years of age and 81 children were of age more than 5 years.

ROP positive cases presented to us were less than 12 months of age.

Table 1: Age wise distribution of patients

Age Group	No. of pattients
< 3 months	23
3- 12 months	18
1-5 Years	29
>5 year	81

The average sex wise distribution of posterior segment finding in children is depicted in its pie chart.

There were 59 female patients and 92 male patients which shows that in all the categories males outnumbered the females

Table 2 shows correlation of birth history with posterior segment findings. 33 patient were born full term delivery whereas 11 patients were premature according to history given by their parents. The patients who are positive for ROP or had Generalised pallor in their fundus finding were all premature.

Table 2: Correlation of birth history with fundus findings

Birth History	No. of patients
Full term	33
Premature	11

Table 3 shows the association of personal history to their fundus finding.

Table 3: Association of personal history of children to their fundus findings

Personal History	No. of patients
Anaemia	1
HIV	1
Hydrocephalous	3
Hypertension	1
IUGR	11
Jaundice	1
MAS	1
Oxygen therapy	5
Pyrexia	1
Seizures	4
Sepsis	2
Tubercular Meningitis	1

Table 4 shows the details of patients presented with posterior segment findings and gave positive history of trauma

Table 4: Details of type of trauma patients got

	No. of patients
Blunt trauma	21
Hammer Chisel injury	1
Trauma with vegetative matter	1
Trauma with sharp object	3

IV. Discussion

The eyes of children basically does not differ much from that of adults. The principles of tests, clinical approach and interpretation is almost same in all ages with little modification

A total of 12,528 patients under age of 18 years presented in our OPD from 1st September 2015 to 30thmarch 2017, of which 2160 who complained of vision related eye problem were screened for posterior segment pathology in our department and a thorough ocular and systemic examination was done.

It is essential to keep in mind that a child's eye is not a condensed form of an adult eye. So for the purpose of examination, the patients in our study have been divided into following age groups:

<3 months
3 months to 12 months
1year to 5 years of age
>5 years of age

In our study we found that out of 151 patients who got positive findings**,** 81 i.e. 53.6%were above 5 year of age. These findings were supported by study of Yang M et al where prevalence of myopia was higher in age 11-13 years.

Puodziuvene E et al studied that closed globe injury noted to be higher in children aged 13-18 years which also supports our findings.

It is seen that vision problems are common among school age kids. This could be explained on the grounds that they can convey their visual problems or ocular discomfort to their parents better than small kids who are unable to express their discomfort. This group of kids also spend a lot of time in outdoor recreational activities that require good vision. They are more mobile than other age group kids that make them even more susceptible for ocular trauma.

In our study we tried to find out the correlation between the gender and the posterior segment finding in children up to 18 years of age. This study showed slight preponderance of various ocular diseases in males as compared to female children. Though there are some diseases like congenital glaucoma, Retinitis Pigmentosa, Coat's disease which has male preponderance still the results in our study are just a coincidence as there is no study to support this result. It could be possibly because of ignorant nature of females and the fact that males are more mobile so they are more prone to trauma. These findings were supported by Puodziuvene E et al, who studied total 268 cases of paediatric ocular trauma and found that boys were more likely to suffer ocular injuries than girls.

In this study we studied the relation of birth history with the posterior segment eye disorders. Preterm birth can inflict a lot of challenges on the developing ocular system, resulting in visual manifestations of varied significance and pathological scope. The ophthalmic condition associated with preterm birth is retinopathy of

prematurity, which has potential to result in devastating vision loss. Other problems associated with prematurity are myopia, strabismus, astigmatism, amblyopia, and glaucoma.

In our study it was found that twin delivery was also associated with ocular morbidity as it can land to premature delivery. This finding was supported by Vedel C et al, where it was found that twin delivery was a risk factor for induced preterm delivery and infant mortality In case of identical twins chromosomal abnormality will be found in the both the babies.

Instrumental delivery using forceps can cause vitreous haemorrhage, hyphaema, intracranial haemorrhage leading to occulomotor nerve palsy. Retinal haemorrhage is also found in neonate following vacuum extraction. Caesarean delivery can sometimes lead to direct blunt trauma to the eye. Holden R et al studied comparison of ocular trauma in instrumental and normal deliveries and found that instrumental deliveries were associated with eye injuries

The relation of maternal history and eye problems were found in our study. There are many hereditary eye diseases which are transferred to child by mother in utero. The developing embryo may be exposed to maternal infections, such as TORCH infections (ie, Toxoplasmosis, Rubella, Cytomegalovirus, and Herpes simplex, and Syphilis) and lymphocyte choriomenigitis. Ophthalmia Neonatorum have been largely associated with various factors including premature rupture of membranes in preterm mothers, subclinical infections of the lower female genital tract during birth, and nutritional deficiency during pregnancy-prevalent mostly in developing countries.

Afr et al found that etiology of Opthalmia Neonatorum may be gonococcal or nongonococcal, Chlamydia trachomatis being the most important cause in the latter group. The risks of gonococcal and chlamydial ophthalmia in infants born to infected mothers may be up to 30% and 50%, respectively. Gonococcal ophthalmia, if untreated, may progress rapidly to corneal ulceration, perforation, and eventually blindness. Chlamydial ophthalmia is generally milder.

Diabetic pregnancy, despite the improved metabolic control, is still a strong risk factor for alterations in foetal development, particularly in patients with a tendency to brittle glycaemia during the first trimester.

The most common ocular malformation in babies born to diabetic mothers is optic nerve hypoplasia as seen in one of the patient in our study

This is supported by study done by Seo S et al in which superior segmental optic nerve hypoplasia was detected in 16 eyes of 14 subjects, or 0.24% of the 5,612 subjects. All 16 eyes showed a corresponding visual-field defect. In multivariate logistic regression analyses, maternal history of diabetes 95% and paternal history of ischemic heart disease were associated with increased risk of SSOH.

Maternal addiction to tobacco and alcohol is also found to be associated with eye malformation like microophthalmia. Fetal alcohol syndrome (FAS) is the most extreme manifestation of prenatal alcohol exposure.

Gummel K et al supported this where Children with FAS showed a higher incidence of amblyopia, hyperopia, astigmatism, and anisometropia. In children with FAS, the incidence of blepharophimosis was 34% (8% in controls), epicanthus 14% (2% in controls), telecanthus 32% (compared to 4% in controls), eyelid ptosis 9% (none in controls), and strabismus 26% (10% in controls). Ophthalmoscopy revealed a tilted optic disc in 5 children with FAS (7%) compared with none in controls

Exposure to terotogenic drugs can affect the fetus eyes like carbamazepine can cause retinal folds with undeveloped layers. Newborns exposed to cocaine in utero may show marked vascular disruption in the retina manifested as superficial and deep haemorrhages. Although morphologically similar to retinal haemorrhages related to birth trauma, these haemorrhages take longer to reabsorb

Many people are unaware that eye diseases such as myopia, glaucoma and even retinoblastoma can run in families. Having a family history of these diseases can significantly increase a person's risk for developing them.

Prk HL et al studied that of the 236 disc suspect children, 100 (42.4%) had at least one disc suspect parent. Intraocular pressure (IOP) was higher in disc suspect children with disc suspect parents (16.52±2.66 mmHg) than in disc suspect children without disc suspect parents (14.38±2.30 mmHg, $p = 0.023$). In the group with disc suspect parents, vertical CDR significantly correlated with IOP, average retinal nerve fiber layer (RNFL) thickness, rim area, and cup volume. Larger vertical CDR was associated with the presence of disc suspect parents, larger disc area, thinner rim area, larger average CDR, and larger cup volume.

Pathologic myopia represents a subgroup of myopia and affects up to 3% of the world population. High myopia is defined as refractive error of at least -6.00D *or* an axial length of 26.5mm or more. Primary risk factors for pathologic myopia include greater axial length and age. Additional possible risk factors such as female gender, larger optic disc area and family history of myopia have been suggested by additional studies.

This was supported by Guo y et al, where,during the study period, the mean axial length elongated by 1.15±0.56mm in boys and 1.10±0.63mm in girls. Axial length was significantly ($P<0.001$) longer in boys at baseline, with no difference ($P = 0.50$) between genders in axial elongation at the end of follow up. In multivariate analysis, greater axial elongation was associated with less time spent outdoors, more time spent

indoors with studying and paternal myopia. Larger increases in the axial length/anterior corneal curvature (AL/CC) ratio were associated with less time spent outdoors and maternal myopia.

Routine fundus screening of siblings allows for early detection of Retinoblastoma in otherwise asymptomatic children. Detection of spontaneously regressed RB in parents may act as a surrogate marker for germline RB1 mutation and is helpful in genetic counseling.

This is supported by Kalki S et al where routine ophthalmic examination of families (parents and siblings) of 131 consecutive newly diagnosed RB patients, including 262 parents and 23 siblings, revealed spontaneously regressed RB in at least 1 parent of 10 (8%) patients and active RB in at least 1 sibling of 3 (2%) patients. Of the 10 parents with spontaneously regressed RB, the lesions were unilateral (n = 7) or bilateral (n = 3). The regression patterns (n = 13) were comparable with postirradiation regression patterns Type 1 (n = 3), Type 2 (n = 2), Type 3 (n = 2), and Type 4 (n = 3), and spontaneous phthisis bulbi (n = 3). Fundus screening of siblings revealed active RB in at least 1 sibling of 3 (2%) patients. Of these 3 siblings, 2 had unilateral and 1 had bilateral disease. The mean age at detection of RB was 15 months (median, 6 months; range, 2-36 months). The disease was unilateral in 2 and bilateral in 1 patient. Based on International Classification of Intraocular Retinoblastoma, the tumors (n = 4) were classified as Group A (n = 2) and Group B (n = 2).

We also studied the relation of personal history with eye problems in children with the associated posterior segment disease. We found that patient who were diagnosed with ROP or generalised pallor of fundus had earlier history of intra uterine growth retardation, respiratory distress syndrome, Meconium aspiration syndrome, sepsis and oxygen therapy given to them. Among various risk factors, only prematurity is well established.

In this study the patient with hypertensive emergency showed changes of hypertension in fundus finding. Retinopathy is dangerous due to the fact that it may provoke sharp occlusions of the blood vessels in the retina and the optic nerve which require immediate treatment. Hypertensive retinopathy deals with hemorrhages and exudation within the eye fundus and also edema of the optic nerve.

The risk groups are Children with high arterial blood pressure, Children with the hypertension disease, kidney hypertension, and adrenal disease.

This was supported by Foster BJ et al where out of 35 patients with hypertension, 3 were diagnosed with hypertensive retinopathy as their fundus finding revealed macular exudates, scattered haemorrhages, and optic disc oedema.

In our study the patient with severe anaemia showed Roth's spot in their fundus finding. Anemia causes retinopathy in 28% of patients, especially when there is coexisting thrombocytopenia (38%). Anemia causes retinal hypoxia, which leads to infarction of the nerve fiber layer and clinically manifests as cotton wool spots. Retinal hypoxia also leads to vascular dilatation; increased transmural pressure owing to hypoproteinemia; and microtraumas to the vessel walls, which cause retinal edema and hemorrhages. In many clinical situations, thrombocytopenia is associated with anemia, and that leads to defective coagulation and hemorrhages.

This was supported by Macauley M et al where they described the association of Roth spots and severe anaemia. Correct diagnosis and treatment with intramuscular vitamin B(12) injections resulted in complete resolution of the anaemia and Roth spots.

Patients who were diagnosed or operated for hydrocephalus presented with disc oedema. Disc oedema is seen in any pathology that will raise intra cranial tension.

This was in agreement with Lee HJ et al whose study included forty-six patients. The 19 patients without papilledema had a mean age of 2.7 ± 2.7 years, and the 27 patients with papilledema had a mean age of 8.8 ± 4.2 years ($p < 0.001$). The mean ICP was 19.9 ± 10.0 cm H_2O among those without papilledema and 33.3 ± 9.1 cm H_2O among those with papilledema ($p < 0.001$). The mean duration of signs or symptoms was 3.0 ± 4.6 months in the patients without papilledema and 3.4 ± 3.9 months in those with papilledema ($p = 0.704$). The patients with papilledema were older and presented with higher ICP than those without. The causes of hydrocephalus were tumor (59%), congenital anomaly (19%), hemorrhage (13%), and infection (9%). Papilledema was more common in patients who were older, who had higher ICP, and whose hydrocephalus had been induced by brain tumor. However, since papilledema was absent in 41% of the children with hydrocephalus, papilloedema's absence does not ensure the absence of hydrocephalus, especially in younger patients

We found out in our study that Glaucoma was associated in few children who had previous history of seizures. As seen in Sturge Weber Syndrome patients develop glaucoma during infancy or later in childhood. This was supported by Lawlor M et al where report of two cases of asymmetrical papilloedema in patients with asymmetrical intraocular pressures (IOP). The first patient presented with headaches, transient visual obscurations (TVOs), and elevated IOPs, and was found to have increased intracranial pressure caused by a torcula meningioma. He developed papilloedema after his IOPs were pharmacologically lowered; the papilloedema resolved after the IOP became elevated again after stopping his glaucoma drops, and then again returned as the IOP reduced when the drops were restarted. The second patient with a history of Sturge-Weber

syndrome requiring previous left trabeculectomy, presented with left-sided TVOs, photopsia, and pulsatile tinnitus caused by idiopathic intracranial hypertension. Asymmetrical papilloedema was observed, worse in the eye with the lower IOP following trabeculectomy. These cases suggest that asymmetric IOP may be one factor that can influence the development of asymmetric papilloedema.

Pediatric uveitis is challenge to diagnose and manage, with frequent and potentially severe complications. Most cases were bilateral, recurrent, and idiopathic. Prompt referral to uveitis-specialized centers and an appropriate systemic therapy are mandatory for good visual outcome. Anterior uveitis is commonest, and juvenile idiopathic arthritis and toxoplasmosis are the most frequent etiologies. Diagnosis of pediatric ocular tuberculosis is more difficult than in adults and needs better and well-defined criteria.

In our study we have seen 4 cases of choroiditis. The exact etiology could not be identified even after a thorough examination and investigations

This was supported by Ferrara M et al where, 286 children included, 62.24% were females. Mean age of onset was 8.4 years. The uveitis was mainly anterior (61.9%), recurrent (68.53%), bilateral (81.82%), and noninfectious (96.5%). Idiopathic cases accounted for 51.4%. The most frequent systemic association was juvenile idiopathic arthritis (34.96%). The majority of patients (78.32%) experienced complications.

Takkar b et al also supported this, where One hundred and thirty-four children were analyzed. Anterior uveitis (40%) was the commonest pattern followed by intermediate uveitis (25%), panuveitis (18%) and posterior uveitis (17%). Bilateral disease was present in 54%, 15% had infectious uveitis, 10% had granulomatous uveitis and 54% had idiopathic uveitis. Complications were present in half of the patients. Juvenile idiopathic arthritis (22), followed by toxoplasmosis (10) and tuberculosis (5), were the commonest etiology. Intermediate uveitis, non-granulomatous inflammation and older onset of disease had the high odds ratio of having idiopathic disease.

A major portion of children with positive finding presented with the history of trauma. It was found that blunt trauma caused retinal haemorrhage, vitreous haemorrhage, berlin's oedema, macular hole, macular scar and retinal detachment.

The term *commotio retinae* describes the damage to the outer retinal layers caused by shock waves that traverse the eye from the site of impact following blunt trauma. Ophthalmoscopically, a sheen like retinal whitening appears some hours following injury. It is most commonly seen in the posterior pole but may occur peripherally as well. Several mechanisms for the retinal opacification have been proposed, including extracellular oedema, glial swelling, and photoreceptor outer segment disruption. With foveal involvement, a cherry-red spot may appear, because the cells involved in the whitening are not present in the fovea. Commotio retinae in the posterior pole, also called *Berlin oedema*, may decrease visual acuity to as low as 20/200. Fortunately, the prognosis for visual recovery is good, as the condition clears in 3–4 weeks. In some cases, however, visual recovery is limited by associated macular pigment epitheliopathy, choroidal rupture, or macular hole formation.

In our study we found that among patients with history of blunt trauma 8 presented with Berlin's oedema. Choroidal tear, macular hole, macular scar and retinal haemorrhage were also the presentation. 3 patients presented with retinal detachment.

This was in agreement with William DF et al who stated that nonpenetrating or blunt ocular trauma, orbital trauma and systemic trauma may cause a variety of posterior segment abnormalities. Blunt ocular trauma may cause damage to the retina (commotio retinae), retinal pigment epithelium (retinal pigment epithelial edema), choroid (choroidal rupture) and optic nerve (optic nerve evulsion) alone or in combination. Traumatic macular holes and retinal detachment or dialysis may also occur after blunt ocular trauma. Trauma to the orbital tissues adjacent to the globe can cause concussive forces with damage to multiple structures within the eye **(chorioretinitis sclopetaria).** Systemic trauma may result in diffuse retinopathy (Purtscher's retinopathy, shaken baby syndrome) or localized retinal abnormalities (whiplash retinopathy, fat embolism syndrome). Alterations in intravascular (Valsalva retinopathy) or intracranial pressure (Terson's syndrome) due to a variety of causes may result in preretinal or vitreous hemorrhage and associated visual loss. The purpose of this report is to review each of these entities of traumatic posterior segment abnormalities

Post-traumatic endophthalmitis in children is most often caused by injury with organic matter. Corneal abscess and retinal detachment are associated with poor outcome. **E. fecalis** is the most common causative organism. Early vitrectomy results in better outcomes.

In our study we have found that the patient with history of injury with organic matter developed post traumatic endophthalmitis. Thorough ocular and systemic examination was done followed by ultrasonography to confirm the diagnosis. First 3 doses of intravitreal antibiotic i.e ceftazidime and vancomycin were given. Since patient does not responded well to intravitreal injections so pars plana vitrectomy was planned after which the infection subsided and visual acuity improved.

These findings were supported by E Rishi et al where mean age at presentation was 9.2 years (median 8 years, range: 2 months to 18 years). Broomstick and hypodermic needle were most common causes for injuries. Common presenting features were cataract (**n**=51), hypopyon (**n**=45) and retinal detachment (**n**=29). Corneal

abscess (**n**=21; OR: 5, CI: 1.4–18.7) and retinal detachment (**n**=29, OR: 5, CI: 1.6–11.3) were independent risk factors for poor outcome (**P**=0.04 and 0.012, respectively). Gram-positive bacteria were isolated in 54% (**n**=31) of culture-positive cases. Forty-nine (34%) patients had ambulatory vision at final visit. Patients who received treatment within 24 hours were more likely to have better anatomical outcome than those treated at 2–7 days, or >7 days, respectively (**P**=0.001). Patients undergoing early vitrectomy were 27 times more likely to have better outcome (**P**=0.0001).

In this study we reported a case of microphthalmia with white pupillary reflex with esotropia. Ultrasonography was done to evaluate which revealed Persistent hyperplastic primary vitreous.

This was supported by Kumari R et al who found that Persistent Hyperplastic Primary Vitreous (PHPV), now-a-days referred to as Persistent Fetal Vasculature (PFV), is a rare congenital anomaly of the eye that typically presents unilaterally with white pupillary reflex and microphthalmia without systemic associations. Bilateral presentations are rare. A case of 15-year-old male with bilateral PFV without leucokoria who was misdiagnosed as congenital esotropia with amblyopia, treated for refractive errors and referred to higher centre for squint corrections. The diagnosis of PFV was made easily on slit lamp examination with dilated pupil due to media clarity. Findings were confirmed by colour doppler ultrasonography of the eyes.

In this study 2 patients otherwise asymptomatic on their fundus finding showed myelinated nerve fibres around disc.

Gradle HS et al found that Myelinated retinal nerve fiber layers (MRNF) occur in 0.57- 1% of the population and can occur bilaterally in 7.7% of affected patients. MRNF are grey-white well-demarcated patches with frayed borders along the retinal nerve fiber layer, obscuring underlying retinal vessels. Clinically, MRNF appear continuous with the optic nerve, but they are often discontinuous with the optic nerve head as well. Most patients with MRNF are asymptomatic; however, visual function can become affected, resulting in axial myopia, amblyopia, and strabismus in the affected eye. Though rare, familial cases of MRNF have been reported both in isolation and in combination with ocular and systemic syndromes. MRNF are typically present at birth and are static lesions, but a few cases of acquired and progressive lesions in both childhood and adulthood have been described. Disapearance of MRNF had also been reported after surgery and insults to the optic nerve.

Ocular coloboma occurred in 1 in 2077 live births. More than half were diagnosed with an ocular disorder other than coloboma, including strabismus or amblyopia in approximately one-third of patients. In this study we found 7 patients who presented with decreased vision but on fundus finding showed fundal coloboma

Kelly M Nakamura et al studied Thirty-three children with newly diagnosed with ocular coloboma during the 40-year study period for an annual incidence of 2.4 per 100,000 residents < 19 years of age, or a prevalence of 1 in 2077 live births. The median age at diagnosis for the 33 subjects was 3.9 months (range, 2 days to 18.4 years), and 22 (67%) patients had unilateral involvement. Twelve (36%) patients had involvement of the anterior segment only, 13 (39%) of the posterior segment only, and 8 (24%) of both anterior and posterior segments. During a median ophthalmologic follow-up of 9.2 years (range, 13 days to 35.9 years), 19 (58%) had other ocular disorders including amblyopia in 11 (33%) and strabismus in 10 (30%). During a median medical follow-up of 16.8 years, 22 (67%) were diagnosed with a non-ocular disorder, including abnormal development in 12 (36%) and CHARGE syndrome in 4 (12%).

In our study 3 patients of albinism were presented with decreased vision. On fundus finding retinal and choroidal vessels stand out prominent against sclera, macula is hypoplastic and optic nerve head cannot be differentiated.

This was in agreement with Ferdinand Rodriguez et al who studied Two unrelated patients with Oculocutaneous albinism who were referred for genetic analysis. Patients had almost total iris transillumination, clear lenses, foveal hypoplasia with transparent maculae, and albinotic mid peripheries. Both patients had nystagmus, and only one patient had strabismus

Thus it is inferred that, in our study there was association of posterior segment ocular findings to age, birth history, family history, personal history, history of trauma as well as type of trauma. Our findings are supported by previous studies also.

V. Conclusions

It is estimated that 1.5 million children suffer from severe visual impairment and of these, one million children live in Asia. Eye diseases are an important cause of medical consultation.

The relation of age of patient to the diagnosis in our study explained that kids above 5 years of age were in majority, which in turn explains that since small children cannot communicate so their eye problems goes undiagnosed. It is therefore needed that these children should be examined for their posterior segment finding by ophthalmologists so the problem can be early diagnosed and treated.

Eye exams for children are extremely important, because 5 to 10 percent of preschoolers and 25 percent of school-aged children have vision problems. Early identification of a child's vision problem can be

crucial because children often are more responsive to treatment when problems are diagnosed early. Good vision is key to a child's physical development, success in school and overall well-being.

There is a critical need for the development of tailor made programs which encompass the issues of the target population in developing nations. Our hope with this study was to bring to light the efforts that should be undertaken to screen fundus of the children for eye pathology and treat them accordingly to decrease the burden of preventable blindness among children.

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