PREVALENCE OF CEREBRAL VISUAL IMPAIRMENT AND OCULAR VISUAL IMPAIRMENT IN CHILDREN AGED 6 TO 18 MONTHS ATTENDING HIGH RISK BABY CLINIC IN A TERTIARY CARE HOSPITAL: A CROSS-SECTIONAL STUDY

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PREVALENCE OF CEREBRAL VISUAL IMPAIRMENT AND OCULAR VISUAL IMPAIRMENT IN CHILDREN AGED 6 TO 18 MONTHS ATTENDING HIGH RISK BABY CLINIC IN A TERTIARY CARE HOSPITAL : A CROSS -SECTIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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ABBREVIATIONS

CVI	Cerebral Visual Impairment
OVI	Ocular Visual Impairment
KIMS	Kerala Institute of Medical Sciences
TAC	Teller Acuity Cards
MRI	Magnetic Resonance Imaging
HIE	Hypoxic-Ischemic Encephalopathy
ROP	Retinopathy Of Prematurity
CSF	Cerebrospinal Fluid
OKN	Optokinetic Nystagmus
VEP	Visually Evoked Potential
PVL	Periventricular Leukomalacia
SPECT	Single-Photon Emission Computerized Tomography
fMRI	functional MRI
PET	Positron Emission Tomography
CP	Cerebral Palsy
HRB	High Risk Baby
NICU	Neonatal Intensive Care Unit
NST	Nonstress Test
BPP	Biophysical Profile
DC	Dichorionic
MC	Monochorionic
BPD	Bronchopulmonary Dysplasia
PDA	Patent Ductus Arteriosus
AED	Acute Encephalopathy with reduced subcortical Diffusion
IVH	Intraventricular Hemorrhage
IQ	Intelligence Quotient
CMV	Cytomegalovirus
MC	Chi square test with Monte Carlo simulation.

ABSTRACT

Introduction: Cerebral Visual Impairment (CVI) and Ocular Visual Impairment (OVI) are significant causes of childhood visual impairment, impacting neurodevelopment and quality of life. With advancements in neonatal care, the prevalence of CVI has risen, surpassing other causes of childhood blindness. This study investigates the prevalence of CVI and OVI among high-risk infants in a tertiary care setting in India.

Objective of the Study:

- The primary objective was to determine the prevalence of CVI and OVI in children aged 6 to 18 months attending High-Risk Baby (HRB) Clinic in KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre.
- The secondary objective was to analyze associated risk factors and severity of CVI.

Methodology: This is a cross-sectional hospital-based study conducted at Departments of Ophthalmology and Pediatric, KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi during the period of April 2023 to March 2024. Total 133 children between 6-18 months of age attending high risk baby clinic diagnosed to be at moderate or high risk using the Kerala Institute of Medical Sciences (KIMS) stratification model were included in the study. Detailed history was taken regarding the perinatal events and the child's behavior. Comprehensive ophthalmic and neurodevelopmental assessments were performed, including Teller Acuity Cards (TAC) for visual acuity and Magnetic Resonance Imaging (MRI) for neuroimaging. CVI diagnosis was based on Roman-Lantzy's assessment criteria. Data was statistically analyzed.

Results: The study found that 18 (13.53%) of children had CVI, 11 (8.27%) had OVI, and 10 (7.52%) had both conditions coexisting. Developmental cataract was the most common

cause of OVI (5, 45.45%). Strabismus (8, 80%) was the most common OVI noted in children with coexisting CVI with exotropia being the most frequent (7, 70%). A significant association was observed between neonatal convulsions and CVI ($p = 0.0299$). Hypoglycemia did not show a statistically significant association with CVI despite showing similar trends as other positively correlated factors ($p = 0.5392^{MC}$). Consanguinity was strongly linked to OVI ($p = 0.009$). Global developmental delay was considerably higher in CVI (12, 66.67%) as well as CVI with coexisting OVI (10, 100%) ($p < 0.001$) but not noted in any of the OVI children. Caregivers of CVI children were concerned about the child's vision more commonly (23, 82.14%) than non-CVI (p-value < 0.001). Behavioral issues were also significantly more prevalent in CVI (p-value < 0.001). Visual reflex abnormality was more common in CVI children (3, 0.95%, p-value = 0.0350), whereas the most common impairment observed during screening was unresolved visual complexity (11, 39.29%, p-value $< 0.001^{MC}$). Most severe risk category children were diagnosed with severe grade of CVI i.e., phase one. No association was found between the CVI phases and postnatal risk factors or TAC vision scores. MRI findings indicated gliosis and periventricular leukomalacia in preterm CVI cases, while hypoxic-ischemic encephalopathy (HIE) was prevalent in term infants. Both CVI and OVI led to significantly impaired vision.

Conclusion: The study underscores the necessity of early screening and intervention in high-risk infants. Neonatal convulsions, perinatal hypoxia were major risk factors for CVI, while consanguinity was linked to OVI. The findings emphasize the importance of multidisciplinary approaches for timely diagnosis and rehabilitation to reduce childhood visual impairment.

Keywords: Cerebral visual impairment, Ocular visual impairment, High risk.

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INTRODUCTION

"The eye sees only what the mind is prepared to comprehend." These were the words of Robertson Davies in his book Tempest-Tost published in 1952.[1] This quote highlights the intricate relationship between cerebral and ocular visual impairment, emphasizing how visual perception is not just about the eyes but also about how the brain processes what is seen.

Our central nervous system gradually develops its visual abilities from infancy. The human brain's occipital region is dedicated to processing and deciphering visual information.[2]

According to clinical definitions, "Cerebral Visual Impairment (CVI) is characterized as a profound and verifiable visual dysfunction that cannot be ascribed to conditions of anterior visual pathway or any possible co-occurring ocular impairment and is associated with damage to brain structures and retro chiasmatic visual pathways" [3]

Diagnosing CVI is often a challenge due to variable visual characteristics and associated neurological morbidities. Visual impairments can vary from profound visual impairment (low functioning CVI) to normal or relatively normal visual acuity accompanied by significant cognitive visual dysfunction (high functioning CVI). It is essential to acknowledge that CVI and ocular visual impairment (OVI) may coexist.[4]

Variable visual attention and inattention (especially in new or complicated surroundings), using touch to augment vision, averting gaze while reaching for objects, close observing with no refractive errors, preferring paying attention to moving objects over static ones, attraction to coloured objects, light gazing, and photophobia are among the behaviours profiled in CVI. Additionally, challenges in

higher-order visuospatial processing have been noted, resulting in significant functional limits that influence a child's education, development, and mobility.[5]

Premature babies with low birth weight and gestational age are demonstrably at heightened risk for visual impairments, including CVI. [6] Regardless, both preterm as well as term born infants are at risk of developing CVI or OVI even though the etiology of the disease varies in both groups. Perinatal or postnatal hypoxia-ischemia is the most prevalent cause of CVI in children. Other insults such as infections, epilepsy, trauma, medications or poisons, and certain neurological illnesses are also important etiological factors.[7]

Owing to the improved newborn care and technological advancements, CVI has surpassed cataract, retinopathy of prematurity (ROP), and glaucoma, as primary cause of children's visual impairment in India as a direct result of increased incidence of infant survival during pregnancy and the perinatal period. A 2019 study performed at South Indian tertiary care centre on 428 children under age of three found that 50.5% of urban children had CVI. Therefore, routine CVI screening is urgently needed.[8]

In developed nations, frequency of CVI in children under age of sixteen ranges from 10 to 22 cases per 10,000 births, while in developing nations, it is approximately 10 cases per 10,000 births.[9] Even though the prevalence is low in comparison to adult visual impairment or blindness, childhood blindness causes a disproportionately high number of future blind years, necessitating immediate attention to this issue.

"Ocular Visual Impairment" (OVI) that causes childhood blindness is often caused by a number of conditions, encompassing cataract, corneal opacity (including vitamin A deficiency), congenital globe anomalies (microphthalmos/anophthalmos), glaucoma, retinal dystrophies, optic atrophy, and amblyopia due to high refractive error, based

on Indian studies.[8] Strabismus and Nystagmus are commonly noted to be associated with paediatric cataract. [10]

Living with visual impairment deeply impacts a child's development in a multitude of ways like delay in achieving milestones, increased susceptibility to trauma and hospitalizations. Such children are forced to experience deprivation with harsh impacts on their emotional and social well-being and less likelihood for future work, all of which ultimately raise the financial burden on society.[11] Prompt care and early detection of impairment assist these children improve their quality of life as well as vision.

There is limited research available on the prevalence of CVI and OVI in Indian scenario. Awareness and studies are still lacking in this field in developing countries. This study was carried out to contribute to raising awareness of CVI and OVI and to bridge the information gap. It also assists in early detection and rehabilitation of at-risk children and ultimately aids in decreasing the burden of childhood blindness.

AIMS AND OBJECTIVES

- To study the prevalence of Cerebral Visual Impairment and Ocular Visual Impairment in children aged 6 to 18 months attending High Risk Baby Clinic in a tertiary care hospital.

- To correlate the various risk factors and severity of Cerebral Visual Impairment.

REVIEW OF LITERATURE

Neurodevelopment- Embryology of the brain:

One of the first systems to develop and the last to be completed after birth is the neural system. The expanded cranial portion of neural tube gives rise to brain. Three dilatations, or principal brain vesicles craniocaudally, are visible in the expanded cephalic portion around the fourth week of pregnancy which include:

i. Prosencephalon
ii. Mesencephalon
iii. Rhombencephalon

Five secondary brain vesicles are produced by the differentiation of the three primary brain vesicles:

1. *Prosencephalon, which becomes the forebrain:* Later on, this gives rise to cerebral hemispheres, containing hypothalamus, thalamus, and epithalamus beneath them. Sensory integration, sensorimotor transformation, and consciousness are all controlled by this area of the brain.

2. *Mesencephalon, which becomes the midbrain:* In contrast to spinal cord and other brain vesicles, this area of brain experiences less structural change.

3. *Rhombencephalon, which becomes the hindbrain:* The following part is further separated into three segments:

1. Metencephalon: The cerebellum's dorsal development (integrating sensory data to optimize output)

2. Caudal myelencephalon: Similar to the spinal cord anatomy, Medulla's central canal is closed.

3. Rostral myelencephalon: Open part of medulla; choroid plexus produces cerebrospinal fluid (CSF), which seeps into subarachnoid space.[12]

Beginning with neurogenesis, the phases of brain development progress to neuronal migration, maturation, synaptogenesis, pruning, and myelin production.[13]

The Visual Processing Hub- Visual cortex and Visual association areas:

The primary visual cortex (V1), often referred to as Brodmann's area 17, is in charge of visual stimulus awareness and is located within the walls of the deep calcarine sulcus in occipital lobe. Secondary visual areas 18 (V2) and 19 (V3), which involve the occipital lobe and posterior portion of parietal lobe, are located next to primary visual cortex. It is involved in perception of colour, depth, motion, and other visual elements as well as the interpretation and identification of objects.[14]

Table 1: Primary cerebral visual processing areas [15]

PRIMARY VISUAL PROCESSING AREAS	FUNCTION
V1 (plexiform lamina)	Transmits information to the ventral and dorsal stream pathways
V2 (external granular lamina)	Aids in attentional modulation and object recognition
V3 (pyramidal lamina)	Processing of global motion
V4 (internal granular lamina)	Directly involved in form recognition and colour information
V5 (ganglionic lamina)	Processes visual motion
V6 (multiform lamina)	Spatially directed reaching movements

Dual Stream Visual Cognition Framework- Dorsal and Ventral stream:

i. Dorsal stream:

The "Where" or "How" Pathway (for action guidance and spatial awareness). It leads to the **parietal** lobe.

ii. Ventral stream:

The "What" Pathway (for object recognition and identification). It leads to the **temporal** lobe.[16]

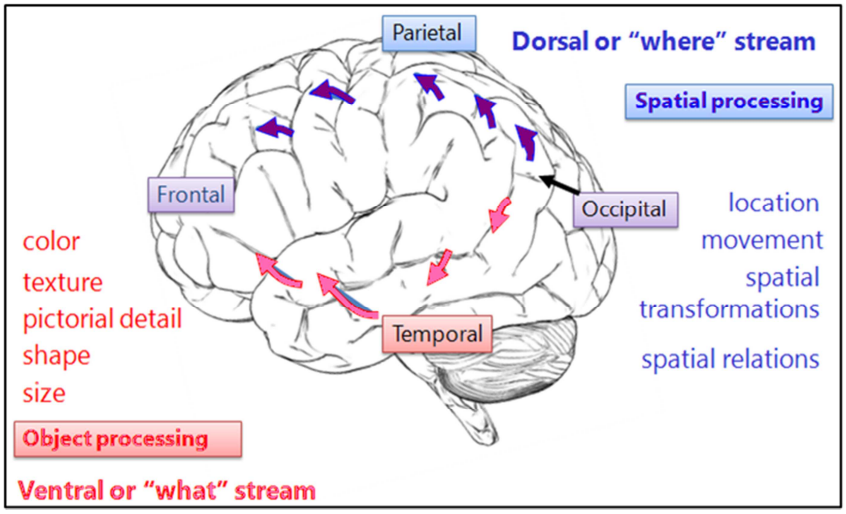

Fig 1: Dorsal and ventral Stream

Source- https://visionhelp.wordpress.com/wp-content/uploads/2012/08/ventral-dorsal-stream.png

CVI can result from either pathway malfunction, however dorsal stream impairment is more prevalent in children with CVI. Depending on how the site and severity of brain injury vary, children with CVI often exhibit a wide range and combination of visual dysfunctions, like decreased visual acuity and anomalies in visual field. [17]

Table 2: Visual milestones:[18]

Age	Visual Milestones
29 weeks gestation	Pupillary reactions to light
30 weeks gestation to birth	• Dislikes bright light (closes lids in response) • Turns to subdued light
Birth to 1week	Fixation present • Follow horizontally moving targets-OKN and vestibular eye movements well developed. • Visual acuity on acuity cards (6/120)
4 weeks to 8 weeks	Fixation well developed • Follows objects moving vertically • Fusion develops • Watches mother's face intently for prolonged duration • Watches toys held in front of face • Doll's head eye movements present
3 months	Observes movements of own hands • Reaching out to objects of interest • Prefers photographs, mirror faces to patterns.
4 months	Foveal differentiation completed. Accommodation developed.
5 months	Blink in response to a visual threat (Menace reflex)
6 months	• Grasps objects and explores with fingers • VEP acuity adult level (6/6), Acuity cards: 6/30 • Stereopsis in PLT well developed • Fusional convergence well developed
9 months	Visual object differentiation, small object pickup
18 months	Visual acuity (Acuity cards: 6/6)
3 years	Vision 6/9- 6/6 on Tumbling E/ HOTV (Recognition) • Contrast sensitivity adult level.
5-7 years	Well-developed stereopsis (adult level).
10 years	Critical period for monocular deprivation ends (synapse formation completed)

Source: Sharma PI. The preliminary examination and assessment of visual acuity. Strabismus Simplified. 2nd ed. New Delhi: CBS Publishers & Distributors;2013. P .52-57.

Obstacles encountered when evaluating children:

Any method for evaluating an infant's visual state must consider two crucial factors because of the infant's visual system's immaturity and the dynamic nature of visual development during the first few months after birth:

1) The first step is to compare the visual assessment results with normative data from infants of the same age evaluated using the same technique. This is done to avoid misdiagnosis by comparing results to data from adults, older children, or newborns examined using a different approach.[19]

2) The second step is to remember that the visual status in later life is not always predicted by the outcomes of visual exams performed during infancy. If the visual system does not go through the significant amount of development that typically takes place between infancy and adulthood, an infant whose eyesight seems normal at birth may eventually exhibit visual impairment. In similar fashion, newborns who exhibit visual impairments earlier may go on to exhibit normal visual responses weeks or months later.[19]

Overview of Cerebral Visual Impairment:

The brain's intricate process of vision includes the eyes, visual pathway, and higher cerebral region. Visual perception involves a significant portion of the brain. The definition of CVI has been specified by numerous writers and is continually changing. Visual impairment brought on by disease of the retro chiasmal and visual association pathways is a hallmark of CVI. It happens without evidence of vision loss that is more than would be predicted given the severity of ocular pathology, or damage to the anterior afferent visual pathways or ocular structures.[20]

Cortical visual impairment is another term for cerebral visual impairment, or CVI. Currently, the term cerebral is preferred over cortical since cortex is rarely involved in isolation. As improved perinatal care facilities and preterm infant survival rates have increased, one of main reasons of visual impairment nowadays is CVI in affluent nations and is also becoming more widespread globally.[9]

In developed nations, the frequency of children's visual impairment under age of sixteen ranges from 10 to 22 instances per 10,000 births, but in developing countries, it is approximately 10 cases per 10,000 births.[21]

In 2009, Dutton explained how the dorsal and ventral pathways are impacted by CVI. Compared to ventral stream dysfunction, dorsal stream dysfunction is more prevalent in CVI. Optic ataxia, simultanagnosia, gaze apraxia, the inability to use many sensory inputs simultaneously, and frequently a lower visual field deficit are all symptoms of dorsal stream dysfunction. Similarly, injury to the ventral stream impairs visual recognition and navigation.[22]

Risk factors of CVI

The most frequent cause of CVI in term or preterm newborns is perinatal or postnatal hypoxic ischemic encephalopathy. A higher possibility of developing CVI is linked to preterm birth, which increases the risk of intraventricular haemorrhage or periventricular leukomalacia. Other common causes documented are: [23]

Table 3: Risk factors of CVI

PRE NATAL	PERINATAL	POSTNATAL
1. Maternal drug use	1. Periventricular leukomalacia (PVL)	1. Head injury (Trauma)
2. Intrauterine infections		2. Stroke
3. Twin pregnancy	2. Hypoxic- ischemic encephalopathy (HIE)	3. Seizures
4. CNS developmental defect		4. Meningitis
	3. Cerebral haemorrhage	5. Hydrocephalus

Pathophysiology of CVI:

Hypoxia

Hypoxic-ischemic brain injury is the most common cause of CVI. However, the pattern of damage brought on by the hypoxic insult varies significantly between term and preterm children and is mostly determined by the age at which the insult occurs.[24]

Term infants-

Since they are watershed regions of the cerebral cortex, the regions most frequently impacted are those between the circulation of the anterior and middle cerebral arteries and the middle and posterior cerebral arteries. These watershed zones experience hypoperfusion due to a loss of vascular flow autoregulation brought on by hypoxia, which causes infarction of the frontal and parieto-occipital regions. The occipital visual areas, temporal and parietal cortices, and the striate cortex are all commonly impacted.[24]

Preterm infants-

These children seldom have hypoxic-ischemia-induced parasagittal infarctions. Deep white matter involvement in the periventricular region is more frequent, particularly when the lesion happens earlier—between 24 and 34 weeks of pregnancy. The adult vasculature eventually replaces the temporary, vulnerable watershed zone in the periventricular white matter. From the pial surface, long penetrating vessels that are primarily derived from the middle cerebral arteries end in the deep periventricular white matter. The final 16 weeks of pregnancy are when this periventricular vasculature actively develops. In the third trimester, there are more short penetrators and anastomoses between the long and short penetrators, which results in fewer border and susceptible end zones. This area's capillaries are vulnerable to hypoxic-ischemia-induced bleeding.[24]

Meningitis

In the past, it was believed that meningitis and hydrocephalus were the most frequent causes of CVI but recently, they account for 11.8% to 15% of the cases.[25] Haemophilus influenzae is the most frequent cause of CVI because it can cause more

damage to the occipital lobe. Other causal agents that have been linked to ocular and cerebral vision issues include pneumococci, meningococci, and herpes simplex virus. Visual impairment usually appears quite delayed in infection's progression, and there are frequently other neurologic sequelae that accompany it. Arterial occlusion, venous sinus thrombosis, hypoxic-ischemic damage, Thrombophlebitis, as well as hydrocephalus are some of the various ways that infection can harm the brain.[26]

Hydrocephalus

It can impact posterior visual pathways passing close to lateral ventricles and impair vision by inducing optic atrophy through a variety of processes. It is common to have both anterior and posterior visual involvement. A common method through which hydrocephalus induces CVI is chronic distention of the posterior cortex, even if ventricular dilatation might obstruct the posterior cerebral arteries. It is commonly recognized that CVI can result from shunt dysfunction; however, ironically, CVI can also occasionally be brought on by shunting, which quickly corrects elevated intracranial pressure. Furthermore, secondary visual atrophy brought on by excessive intracranial pressure from untreated hydrocephalus can exacerbate any related CVI.[27]

Trauma

An additional cause of CVI (around 4% cases, according to 2 researches) is head trauma. Damage might be temporary or irreversible. Shaken baby syndrome is a constituent of most common causes of post-traumatic CVI. Children may also have temporary blindness following minor trauma, which can be followed by headache, disorientation, fatigue, nausea, and convulsions. [28]

Epilepsy

Increased metabolic demands or postictal hypoxia can also cause CVI. Frequent myoclonic seizures are a hallmark of infantile spasms, which can significantly impair an infant's vision. Even while visual function can be restored with the right care, some people may suffer from long-term vision impairment and significant developmental delays. Children with numerous impairments who have CVI frequently have occipital lobe epileptiform discharges. In order to prevent possible CVI, it is crucial to use sedative medications for epilepsy with prudence. This calls for appropriate medication selection and frequent drug level monitoring in children with epilepsy.[29]

Metabolic disorders

Haemodialysis-induced hypoglycaemia can result in acute CVI. In addition to neurodegenerative disorders including Tay-Sachs, Leigh's disease, neuronal ceroid lipofuscinosis, and X-linked adrenoleukodystrophy, it is seen in conditions like maple syrup urine disease. There may also be coexisting retinal or optic nerve disorders. [30]

Hypoglycaemia

Although the precise mechanism of hypoglycemia-induced brain injury is uncertain, hypoglycemia can cause both cerebral ischemia and brain edema. [31] The depletion of energy-rich phosphorylated molecules, including adenosine triphosphate, which is linked to decreased glucose levels and metabolic processes that result in inadequate energy sources, is one potential explanation for brain injury.[32] Given that the occipital brain has been shown to consume the most glucose of any part of the body, this could be particularly important in the visual cortex. According to studies conducted on lab animals, hypoglycemia also raises intracellular electrolytes, which

results in edema and water retention. Increased intracranial pressure and brain injury may result from this electrolyte imbalance. [33]

Maternal intake of drugs and alcohol

CVI can be caused by maternal consumption of drugs and alcohol, while cortical blindness can occur in adults. Certain drugs such as cyclosporin-A, cisplatin, anticonvulsants, methotrexate and carbon monoxide poisoning cause CVI.[34]

Structural brain anomalies

Other documented CVI cases are associated with congenital brain malformations like lissencephaly, holoprosencephaly, schizencephaly, polymicrogyria, pachygyria and porencephaly.[31]

Genetics

A study by Bosch Dg et al. demonstrated for the first time a genetic correlation with CVI and demonstrated functional deletion of PGAP1 in a child with CVI. Trisomy 18, Trisomy 21, Miller-Dieker syndrome, 1p36 deletion syndrome, Phelan-McDermid syndrome, and 17p13.3 deletion syndrome have all been linked to CVI.[35]

Characteristic Visual Behaviour in Children with CVI:

Light gazing, visual field restriction, poor visual attention, colour preference, photophobia, difficulty differentiating or interpreting complex visual patterns, atypical visual reflexive responses, poor depth perception, poor visual novelty, looking away when reaching, attention to moving objects, and variability in contrast are some of the distinctive visual features of children having CVI. These features might be impacted by child's emotional or physical exhaustion because of poor sensory integration or a lack of familiarity with the visual stimuli. Faces, facial expressions, shapes, sizes,

letters, numbers, and directions will all be hard for these kids to recognize. Due to field defects, simultanagnosia, or attention loss, they might not be able to see all the items in the visual image. Moving closer when looking at objects for magnification and to avoid crowding, having trouble focusing on fast-moving objects, and making unusual eye contact. Touch is the primary method of object identification, and a CVI child's visual traits are distinctive and one-of-a-kind.[36]

Ocular manifestations in CVI diagnosed children:

A study done in San Francisco in 1998 by Richard Huo et al on 170 CVI patients reported that esotropia was found in 18.8% cases while exotropia occurred in 18.2%. “Ocular motor apraxia” was noted in 15.3% while nystagmus was observed in 11.2% children. “Optic nerve atrophy” has been found in 16.5% of cases. Significant refractive errors were detected in 8.2% of patients. Concurrent retinal diseases were noted in 2.9% of CVI patients.[37]

Another study done in Nashville in 2007 by Khetpal et al evaluated 98 children with CVI amongst which 40% had exotropia, 19% had esotropia, and significant high refractive error in 21.4 %. Mild optic atrophy occurred in over 40% and nystagmus in 21.4%.[38]

According to Sowmya Raveendra Murthy's 2020 demographic profile research of 85 CVI children at a tertiary eye hospital in South India, the most common conditions were strabismus (exotropia > esotropia) in 40 (47%), nystagmus in 34 (40%), and refractive error in 42 (49.4%) of children. A fundus examination showed that one child had optic nerve head hypoplasia, 28 had disc pallor (33%) and 47 of the 85 children (55.2%) had normal fundi.[39]

Imaging in CVI

Because of its remarkable soft-tissue resolution, “magnetic resonance imaging” (MRI), a non-invasive imaging method, is a very useful diagnostic tool for brain abnormalities. MRI is the preferred test for such children due to the radiation exposure from CT scans during childhood and the fact that ultrasound cannot be performed after the age of one year.

Imaging is one of the diagnostic options for children with CVI, per a study by Boonstra FN et al. An inventory of the damage to the brain regions involved in visual processing might be created using MRI.[40]

According to a recent task force on newborn encephalopathy, Acute brain injury detected by brain MRI that is ascribed to hypoxic-ischemia is now regarded as key characteristic of CVI. The degree of visual impairment can be predicted based on the clinical severity of HIE at birth. Three major categories can be used to classify the MRI lesion pattern: diffuse cerebral atrophy, periventricular leukomalacia, and multi-cystic encephalopathy. Minor MRI damage is associated with a better prognosis for children, children with diffuse cerebral atrophy, encephalic cysts, and periventricular leukomalacia are far less likely to experience major improvements in visual function.[30]

Researchers have attempted to image these patients using functional neuroimaging techniques like- single-photon emission computerized tomography (SPECT), functional MRI (fMRI), and positron emission tomography (PET); because there is inconsistent evidence linking visual impairment to structural changes seen in neuroimaging in children with CVI.

Management of CVI

Preventing preterm birth and HIE may also help avoid CVI, even though there is currently no evidence-based treatment for it. The ophthalmologist, neurologist, and rehabilitation providers must work together in unison to manage children with CVI. Most children with CVI show some degree of visual recovery, while the precise process is unknown and the improvement usually occurs gradually over months. According to Lambert et al.'s summary of several theories put forth to explain visual improvement, the initial insult causing CVI may not result in cellular death but rather simply disrupt neurons' regular protein synthesis, delaying myelination, dendritic formation, and synaptogenesis.[41] It has recently been hypothesized that delayed visual maturation is the cause of sight improvement in CVI patients. Treatment is necessary for any coexisting disorders of the eye, such as refractive error, strabismus and underlying amblyopia.[42] Since every kid with CVI is likely to have distinct visual and motor impairments, a completely tailored approach is required.

Comprehensive Assessment of Ocular Visual Impairment:

Poor vision in children may be caused by structural abnormalities affecting any part of the eye, from anterior to the posterior. Corneal opacification, congenital cataract, primary aphakia, chorioretinal coloboma, optic nerve hypoplasia, and foveal hypoplasia are a few examples, but there are many more.[43]

A child with low vision may exhibit warning signs that parents and/or a paediatrician should be aware of, including decreased sensitivity to bright lights, delayed or absent eye contact, a slowed development of an intentional social smile, a lack of awareness of the infant's own hands, a lack of goal-directed hand movements, and an inability to focus on familiar objects like faces and toys.[44]

The several underlying causes of developmental disabilities, including acquired damage and prenatal and perinatal conditions, contribute for the greater prevalence of visual problems in these children. Therefore, visual problems are prevalent in children born prematurely: those with brain injury that has resulted in learning impairments and/or cerebral palsy (CP), and those with congenital cerebral anomalies or other genetic diseases that may predispose them to ocular anomalies.[45]

One of the factors contributing to visual impairment in preterm newborns is retinopathy of prematurity (ROP), which has been reported to happen in 1% to 3% of cases.[46]

Additionally, preterm births have been reported to predispose four times as much to refractive errors (29.6%) as term births (7.8%).[47]

Although hypermetropia is the most common refractive error observed in children, preterm children regardless of prior ROP status are more likely to develop myopia. In full-term children, esotropia is three times more common than exotropia, but in premature babies, both abnormalities are equally common.[46]

One of the most prevalent genetic disorders in children is trisomy 21, which puts affected children at risk for several additional eye disorders, such as cataracts, congenital or acquired keratoconus, nasolacrimal duct problems strabismus, blepharitis, reduced accommodation, and refractive errors.[48]

Paediatric cataracts are a major cause of blindness in children. Cataract accounts for 7.4% to 15.3% of childhood blindness in underdeveloped nations such as India.[49] In North India, rubella causes 21% of congenital cataracts, compared to 15% in South India.[50] Amblyopia is the main cause of vision loss in congenital or infantile cataracts. The blurriness of the retinal image in one or both eyes at this critical

moment causes irreversible amblyopia. Childhood cataracts can be treated, therefore early detection and treatment can avert blind years.

JUSTIFICATION:

The incidence of CVI and OVI has been on the rise especially in developing countries like India due to better perinatal outcomes owing to advancements in neonatal care. Hence, there is a need to be armed with all the knowledge and skills available to tackle this new and upcoming struggle. On literature search, it was found that awareness and studies are lacking in this field hence, this study is needed for a better understanding of the disease burden in our population as well as for aiding the treatment of the diagnosed children in order to ensure a better quality of life and opportunities for them in the future.

MATERIALS AND METHODS

Study Design:

A hospital-based one-year cross-sectional study.

Study Period:

April 2023 to March 2024.

Study Population:

Children between the age of six to 18 months attending High Risk Baby Clinic of "KLE's Dr. Prabhakar Kore Hospital" from "April 2023 to March 2024" and graded as being at moderate or severe risk by KIMS risk stratification model were evaluated and enrolled for the study.

Sample Size:

The following formula is employed to determine sample size

$$n = \frac{z^2_{\left(1-\frac{\alpha}{2}\right)} pq}{(20\%\ of\ p)^2}$$

n = sample size required,

p = prevalence of CVI and OVI = 44% according to a previous study done,

q = (100 – p) = 100 – 44 = 56

Sample size is calculated at 95% confidence interval and 20% tolerable error. It was derived with reference to the parent article: Pehere NK, Narasaiah A, Dutton GN. Cerebral visual impairment is a major cause of profound visual impairment in

children aged less than 3 years: A study from tertiary eye care center in South India. Indian J Ophthalmol 2019;67:1544-7.

Minimum sample size required was 128.

Null hypothesis:

There is no significant prevalence of CVI and OVI in children aged 6 to 18 months who are diagnosed to be at moderate or severe risk according to perinatal history and evaluation at birth.

Sample selection criteria:

Inclusion criteria:

- Children aged between six to 18 months who are attending High Risk Baby Clinic in "KLEs Dr Prabhakar Kore Hospital, Belagavi", a tertiary care hospital who are graded as Moderate to Severe risk according to KIMS risk stratification model.
- Children for whom informed consent has been obtained from caregivers.

Exclusion criteria:

- Mild risk graded babies according to KIMS risk stratification model attending High Risk baby Clinic are excluded.

Methodology:

A cross-sectional study of 133 children between six and 18 months of age graded to be at moderate or severe risk according to the Kerala Institute of Medical Sciences (KIMS) risk stratification model who were attending the High-Risk Baby clinic of KLE's Dr Prabhakar Hospital was conducted. The Institutional Ethics Committee granted ethical clearance before trial started. Informed Formal written consent in

English /Kannada /Marathi was taken from all parents of children enrolled in the study.

Using a proforma, a thorough history was obtained that covered demographic information, ocular complaints, distinctive visual behavior, delivery method, prenatal history, birth weight, perinatal, gestational age, and postnatal history, milestones, as well as parental consanguinity.

The proforma also consisted of **five behavioral screening questions** to evaluate probable CVI. The behaviors documented were as follows:

1. Parental concern regarding their child's vision.
2. The child's capability to smile at the parent.
3. The child's capability in making eye contact with parent
4. The child's preference to stare at light sources.
5. The child's preference to tilt their head while gazing at an object.

Following this, the direct observation of response to three screening tests for CVI were also documented in the proforma which included:

1. Light Gazing
2. Visual reflexes
3. Visual Complexity

The children were classified to be at moderate or severe risk according to the KIMS (Kerala Institute of Medical Sciences) risk stratification model followed in the NICU of KLE's Dr. Prabhakar Kore Hospital.[51]

Table 4: Risk Stratification Chart (KIMS Model)

	Mild	Moderate	Severe
Gestation (in weeks)	33-34	30-32	<30
Birth weight (g)	>1500	1250-1500	<1250
Fetal growth restriction	>10th centile	3rd to 10th centile	<3rd centile
Antenatal risk	Medical/ Obstetric complications as columns to the right	Abnormal NST/BPP, Maternal fever, DC twins, preterm labour	Eclampsia (seizures), MC twins, triplets or higher order, cord prolapse, chorioamnionitis, abruptio placentae, Absent/reversal of umbilical artery dopplers
Antenatal steroids	Completed	Partial	Not completed
Magnesium sulphate (<32 weeks)	Given		
Need for resuscitation at birth	No resuscitation required/ initial steps/ PPV		Chest compression/ medications
Ventilation	Non-invasive/ short ventilation	Pneumothorax, longer than 7 days of ventilation	BPD
Shock	Nil	Saline bolus	Inotropes/Hemodynamic significant PDA closure
Hypoglycaemia	No	Asymptomatic	Symptomatic
Encephalopathy		Seizures	Discharge on AED/ Encephalopathy>24Hrs

NEC			Stage 2 or more
Neonatal Jaundice			Exchange transfusion/ Encephalopathy
Neurosonogram			Grade 3 IVH/ parenchymal bleed PVL 2 or more

Source- Jain, Niyati. (2012). Risk stratification of neonates at-risk of neuro developmental disability. Indian Journal of Practical Pediatrics. 14. 385-390.

Visual acuity assessment:

Compared to adults, evaluating children's visual acuity takes far more time and requires patience.

Visual acuity assessment methods:

1. Fixation:

Typically, the fixation should be central, stable, and sustained (CSM).

2. Menace Reflex:

Blinking reflexively in reaction to a fast-moving item or visual threat is known as the "menace reflex." By five months of age, the reflex begins to manifest.

3. Brukner's reflex:

The prompt detection of refractive errors can be aided by Brukner's reflex.

4. Preferential Looking test:

When presented with an option between a plain and patterned surface, the infant favors the patterned one.

5. Teller's acuity cards:

One side of the screen has a uniform surface, while the other side alternates at random with black and white stripes. The observer records the baby's head and eye movements in response to the patterned stimulus while the baby is facing the screen.

In this study, visual acuity has been documented with forced choice preferential looking test i.e, “Teller's Acuity Card test”. Across a wide age spectrum and in both clinical and laboratory settings, it offers a quick and precise way to evaluate the visual acuity of both healthy nonverbal newborns/ children and those with neurological or visual impairments. [52]

It is typically agreed upon that a resolution acuity of 30 cycles per degree is equal to a standard Snellen acuity of 20/20. The range of test distances for various age groups is 9.5 cm to 84 cm. The Age Norm Charts established the precise values for various test distances, and they served as a guide to determine the appropriate distance for age. [53]

CONVERSIONS FROM CYCLES/CM TO SNELLEN EQUIVALENTS*@

CYCLES/CM	TEST DISTANCE+		TEST DISTANCE+		
	9.5cm	19cm	38cm	55cm	84cm
38.0	20/57	20/40	20/23	20/16	20/11
26.0	20/84	20/59	20/33	20/24	20/15
19.0	20/110	20/81	20/45	20/32	20/21
13.0	20/170	20/120	20/66	20/47	20/31
9.80	20/220	20/160	20/89	20/63	20/41
6.50	20/340	20/240	20/130	20/94	20/63
4.80	20/460	20/320	20/180	20/130	20/84
3.20	20/680	20/490	20/270	20/190	20/130
2.40	20/910	20/650	20/360	20/260	20/170
1.60	20/1400	20/970	20/540	20/380	20/250
1.30	20/1700	20/1200	20/670	20/470	20/310
0.86	20/2500	20/1800	20/1000	20/710	20/470
0.64	20/3300	20/2400	20/1400	20/960	20/630
0.43	20/4800	20/3500	20/2000	20/1400	20/940
0.32	20/6400	20/4700	20/2700	20/1900	20/1300
0.23	----------	--------- ----------		---------- ----------	

Fig 2: Snellen Equivalents of Teller Visual Acuity

Source-https://childrenseye.org/wiki/lib/exe/fetch.php?media=tellers_cycles-cm_to_snellen.png

The visual acuity tested with Teller’s acuity cards has been then classified, depending on cycles per cm as follows: [54]

Very low vision - < 1.6 cy/cm

Low vision – 1.6cy/cm – 9.6 cy/cm

Near normal – 9.6 -26.0 cy/cm

Ocular Visual Impairment (OVI) assessment:

To rule out any ocular diseases, each participant had a comprehensive ocular examination that included an anterior segment examination as well as dilated fundoscopy. The visual axes was assessed using Hirschberg's test. To rule out strabismus, the cover-uncover test was used. If found to be present, quantification was done with the help of Krimsky test. We also evaluated binocular and uniocular movements. Presence or absence of nystagmus was documented. In cases diagnosed with congenital glaucoma, intraocular pressure was recorded with the help of rebound as well as Schiotz tonometer. Corneal diameter and axial length measurements were also recorded wherever deemed appropriate. In cases diagnosed with congenital ptosis, absence of lid crease and grading were noted. All participants underwent cycloplegic refraction according to American Academy of Ophthalmology guidelines. At a distance of 66 cm, objective refraction was performed using streak retinoscopy (Heine Optotechnik, Germany). When necessary, glasses were prescribed after the cycloplegics' refractive error working distance values were adjusted. After a month, accommodation was corrected by carrying out dynamic retinoscopy.

Cerebral Visual Impairment (CVI) assessment:

The following ten visual behavioral responses were assessed routinely, based on the assessment method drawn from **Christine Roman- Lantzy**:

1) Colour preference:

In order to ascertain whether a child showed a stronger interest in particular colours, various coloured illuminated plastic balls and mylars were used.

2) Need for movement:

Most CVI children react by turning toward moving objects or by displaying specific emotions like smiling, shaking their heads, or turning around. They favor slower-moving items since they will have difficulties seeing fast-moving ones. The child's preferred area of view was covered with a mylar or ball of vibrant colour. Both the moving and stationary versions of the object were displayed.

3) Visual latency:

It is used to describe how long it takes a child to turn their head and look at a target to visually perceive it. Children with CVI frequently have prolonged visual delay, though this varies from child to child.

4) Visual field preferences:

The majority of these kids will show preferences for the right or left visual field, and infrequently the central one, depending on the part of the brain that is injured. Colored balls or other appealing toys were used to measure visual fields using the confrontation method.

5) Non-purposeful gaze and Light-gazing:

The term "non-purposeful gaze" describes a child's inability to concentrate on a specified target or to stay focused when there isn't one. The parent or guardian is questioned about whether their child looks at the light or if they have a preferred side that they utilize more often to view the object. To ascertain whether there is a discernible preference for one side, the examiner watches the child as they interact with the lights in the room.

6) Decreased distance viewing:

Based on the child's preferences, the examiner selects a toy and holds it in front of the child during the evaluation. After that, examiner positions themselves across room from child. It is recommended that parents refrain from encouraging their children to gaze at toy. Examiner gradually moves near to child after starting off from afar. The distance at which the child looks at the item is noted to ascertain their visual response.

7) Atypical visual reflexes:

Numerous children with CVI exhibit delayed or missing blink reflexes and threat responses. While a child's response to fear may be seen periodically, a delayed blink reflex may initially emerge and progressively become more consistent as the child progresses through the CVI phases. The presence of both responses is persistent in the advanced stages of CVI.

8) Novelty:

Children with CVI show a preference for items that are familiar. As a result, they are shown the same object again throughout the examination, and then shown an unfamiliar object with comparable characteristics. The child's visual reaction to the unknown object is observed.

9) Complexity:

Complex visual patterns, such as crowded spaces, multi-colored objects, and the simultaneous use of several senses, will be challenging for a child with CVI to distinguish or understand. To escape sensory complexity, they often choose to sleep or show signs of restlessness in crowded environments.

10) Visual-motor:

Children with CVI may have trouble seeing and reaching a target at the same time. But as the CVI phase progresses, these children begin to show their ability to reach for things of their favourite colour and familiarity on a plain background. [55]

The most widely used scale for evaluating visual functioning in The most widely used scale for evaluating visual functioning in children having CVI based on CVI characteristics is CVI Range, created by Roman-Lantzy. There are two primary components to CVI Range score system. The "**Across-CVI Characteristics technique"** which is the first section, provides important information about the child's visual abilities across various visual functioning levels.

"The five levels of visual functioning are:

CVI Range 1-2: Functions with the minimal visual response;

CVI Range 3-4: Functions with more consistent visual response;

CVI Range 5-6: Uses vision for functional tasks;

CVI Range 7-8: Demonstrates visual curiosity; and,

CVI Range 9-10: Spontaneously uses vision for most functional activities." [56]

The Within-CVI characteristics approach is the next part of the CVI Range examination. Each attribute is given a score in this section to determine how much it influences the child's visual functioning. The specific characteristics are scored on a scale of 0 to 1.

"**0** = not resolved/ always a factor affecting visual functioning;

0.25 = resolving;

0.5 = resolving; sometimes a factor affecting visual functioning;

0.75 = resolving; and,

1 = resolved; not a factor affecting visual functioning."[56]

Finally, the total range of visual functioning is determined by comparing the results from the Within and the Across CVI Characteristics Method, and a score between 1 and 10 is assigned. To ascertain degree of severity, CVI children are divided into 3 phases on basis of this score. Depending on CVI phase, several intervention techniques are advised.[56]

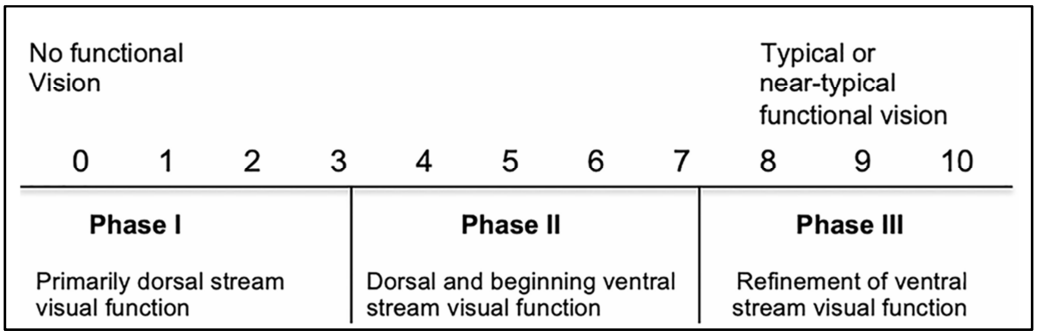

Fig 3: Scoring line for CVI Range

Source- Chang M, Roman-Lantzy C, O'Neil SH, et al. Validity and reliability of CVI Range assessment for Clinical Research (CVI Range- CR): a longitudinal cohort study. BMJ Open Ophthalmology 2022;7:e001144. doi:10.1136/ bmjophth-2022-001144

The children clinically diagnosed with CVI were referred for neuroimaging consisting of an MRI brain. The subjects underwent MR imaging of the brain and orbit. The studies were done in a 3 Tesla MRI machine.

DATA ANALYSIS:

Microsoft Excel and statistical software R version 4.4.0 have been utilized for data analysis. Frequency tables provide information on categorical variables. The continuous variables are shown as Mean ± SD / Median (Min, Max). The Shapiro-Wilk test and QQ plot are employed to verify if the variable is normal. The chi square test is employed to examine the relationship between groups and categorical variables. The QQ plot and Shapiro-Wilk test have been employed for confirming that the variable is normal. Parametric tests are employed when the data is normally distributed. Non-parametric testing are employed otherwise. Whitney, Mann The U test compares variable distributions across groups. A P-value of 0.05 or less denotes statistical significance.

RESULTS

From April 2023 to March 2024, this investigation was performed in a North Karnataka tertiary care teaching hospital. It was a cross-sectional study design. Analysis was conducted for data of 133 children attending High Risk Baby (HRB) clinic. Evaluation was done for all variables in two risk groups according to risk stratification chart of KIMS model as: Moderate and Severe. Results were presented as combined group as well as separately.

Table 5: Distribution according to age and gender

Variables	Sub Category	Number of subjects (n, %)
Age (months)	Mean ± SD	10.34 ± 3.76
Gender	Female	40 (30.08%)
	Male	93 (69.92%)

Table 1 reveals that the mean age of the total children evaluated was 10.34 ± 3.76 months, with a median age of 10 months (Range: 6–18 months). Majority of the children screened were boys.

Table 6: Distribution according to delivery details

Variables	Sub Category	Total population (n, %)	CVI (n, %)	OVI (n, %)
Birth weight (gm)	Mean ± SD	1885.98 ± 694.96	2320.18 ± 749.44	2145.00 ± 593.33
Mode of delivery	Caesarean section	80 (60.15%)	15 (53.57%)	9 (42.86%)
	Normal vaginal delivery	53 (39.85%)	13 (46.43%)	12 (57.14%)
Term/Preterm	Preterm	96 (72.18%)	12 (42.86%)	7 (33.33%)
	Term	37 (27.82%)	16 (57.14%)	14 (66.67%)
Consanguinity	Present	26 (19.55%)	9 (32.14%)	9 (42.86%)

Table 2 reveals that most participants (60.15%) were delivered via caesarean section with a significant proportion (72.18%) being born preterm. The mean gestation period was 32.96 ± 3.39 weeks, with a median of 32 weeks (range: 26–40 weeks).

In the cohort diagnosed with CVI, the mean gestational age was found to be 35.18 ± 3.02 weeks with median of 37 weeks. Majority (57.14%) were term pregnancies and this was statistically significant. Mean age of the children diagnosed with CVI was 11.54 ± 4.26 months with a median age of 11months.

In the OVI diagnosed children, the mean gestational age recorded came out to be 35.33 ± 3.61weeks with median of 37 weeks. Consanguinity was more common among those with OVI (42.86%) than rest of the population and this difference was statistically significant (p-value = 0.0090). Mean age of the children diagnosed with OVI was 11.57 ± 4.59 months with a median age of 11 months.

Table 7: Distribution according to risk status of KIMS stratification model.

Variables	Sub Category	Total population (n, %)	CVI (n, %)	OVI (n, %)
Risk Status	Moderate	101 (75.94%)	19 (18.81%)	15 (14.85%)
	Severe	32 (24.06%)	9 (28.12%)	6 (18.75%)

Table 3 reveals that according to the KIMS risk stratification model, majority of the total screened (75.94%) children were falling under moderate risk. In contrast to 33.66% of children in the moderate risk category, nearly 46.87% of children in the severe risk category subsequently received a diagnosis of either CVI or OVI.

Table 8: Distribution according to risk factors surrounding perinatal events

Risk Factors	Total population (n, %)	CVI (n, %)	OVI (n, %)	CVI with OVI (n, %)	p-value
O2 Therapy	121 (90.98%)	13 (72.22%)	11 (100%)	9 (90%)	0.1164^{MC}
Neonatal Jaundice	89 (66.92%)	7 (38.89%)	5 (45.45%)	6 (60%)	0.6477^{MC}
Convulsions	22 (16.54%)	7 (38.89%)	0	5 (50%)	$\mathbf{0.0299^{MC}*}$
Hypoglycemia	14 (10.53%)	4 (22.22%)	1 (9.09%)	3 (30%)	0.5392^{MC}
Chorioamniotism	2 (1.5%)	0	0	0	1^{MC}
Neonatal Sepsis	5 (3.76%)	1 (5.56%)	0	1 (10%)	0.479^{MC}

Abbreviation: MC – Chi square test with Monte Carlo simulation.

Table 4 reveals that majority of the total screened children (90.98%) required oxygen therapy and 66.92% were diagnosed with neonatal jaundice. Both these categories showed significant associations across all cohorts.

Convulsions were noted to be significantly associated with only CVI (38.89%) and CVI with coexisting OVI (50%) groups (p-value= $0.0299^{MC)}$. Documented hypoglycaemia also showed similar trends in both groups but was not statistically significant. On the contrary, chorioamniotism and neonatal sepsis showed no significance in any group.

Table 9: Distribution according to delayed milestones.

Variables	Sub Category	Total population (n, %)	CVI (n, %)	OVI (n, %)	CVI and OVI (n, %)
Milestones	Delayed	30 (22.56%)	12 (66.67%)	0	10 (100%)
	Normal	103 (77.44%)	6 (33.33%)	11 (100%)	0

Table 5 reveals that majority of the children screened (77.44%) had normal milestones. Developmental delays were significantly higher in CVI diagnosed (66.67%) as well as children with coexisting OVI (100%). None of the OVI cohort children were found to have delayed developmental milestones.

Table 10: Distribution of children according to diagnosis (CVI/ OVI).

Diagnosis	Total children (n,%)
CVI	18 (13.53%)
OVI	11 (8.27%)
CVI with OVI	10 (7.52%)
No CVI or OVI	94 (70.68%)

Graph 1: Distribution according to diagnosis.

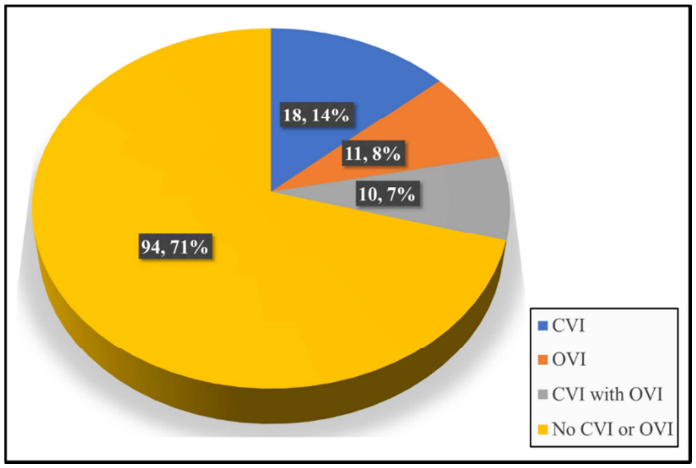

Table 6 and figure 1 reveal that among the participants, majority (70.68%) did not have either condition.

Table 11: Presentations in OVI children.

Total diagnosed = 11 (8.27%)		
Variable		**Number of subjects (n, %)**
Developmental cataract		5 (45.45%)
Congenital Glaucoma		2 (18.18%)
Retinopathy of Prematurity		2 (18.18%)
Strabismus	Esotropia	1 (9.09%)
Congenital Ptosis		1 (9.09%)

Table 7 reveals that among the children screened, majority were diagnosed with developmental cataract (45.45%). The next most common diagnosis were congenital Glaucoma and retinopathy of prematurity at 18.18%.

Table 12: Distribution of children diagnosed with both CVI and OVI.

Total diagnosed = 10 children (7.52%)		
Variable		**Number of subjects (n, %)**
Strabismus	Exotropia	7 (70%)
	Esotropia	1 (10%)
Congenital Nystagmus		1 (10%)
Congenital cataract		1 (10%)

Table 8 reveals that most of the children diagnosed as CVI with coexisting OVI had exotropia (70%). While most of these children (70%) had Phase one CVI, 30% had Phase two CVI at diagnosis.

Grap 2: Mean plot of TAC vision over OVI.

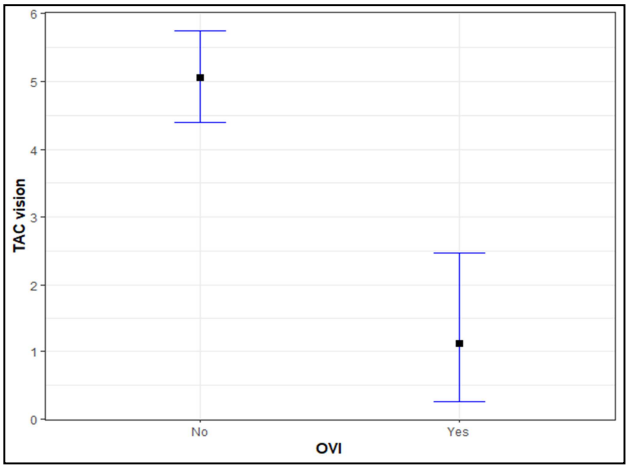

Figure 2 depicts that the OVI group also had significantly poorer vision like CVI, with a lower mean TAC vision score of 1.11 ± 2.84 cycles/cm and median of 0 cycles/cm. (p-value < 0.001). Three (14.29%) children were not able to fixate and follow light or objects.

Graph 3: Mean plot of TAC vision over CVI.

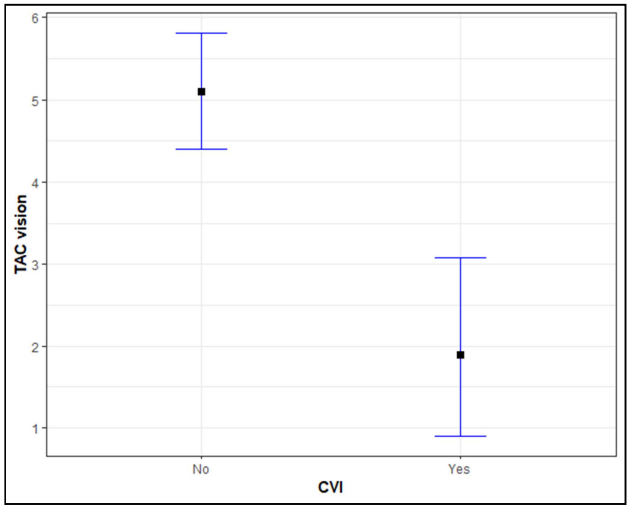

Figure 3 depicts TAC vision scores were significantly lower in CVI subjects, indicating poorer visual acuity (p-value < 0.001). The mean vision was 1.89 ± 3.00 cycles/cm with a median vision of 0.54 cycles/cm. Five (17.86%) children were unable to fixate and follow light or objects which was statistically significant (p-value < 0.001^{MC}*).

Table 13: CVI behavioural screening questions.

Variables	Total population (n, %)	CVI (n, %)
Caregiver concerned	31 (23.31%)	23 (82.14%)
Child makes eye contact	118 (88.72%)	16 (57.14%)
Child smiles at caregiver	120 (90.23%)	17 (60.71%)
Child stares at light sources	69 (51.88%)	25 (89.29%)
Child tilts head while looking	22 (16.54%)	8 (7.62%)

Table 9 reveals that among the total children screened most caregivers (76.69%) did not report any concerns about the child's vision, while 23.31% suspected visual impairment. On the contrary, caregivers of CVI diagnosed children were concerned about the child's vision more commonly (82.14%) than in non-CVI children (7.62%, p-value < 0.001). Behavioural issues such as lack of eye contact (42.86%), smiling (39.29%), and responses to light sources (89.29%) were significantly more prevalent in CVI (p-value < 0.001). Head tilting (50%) and light gazing (17.86%) were also more frequent in CVI subjects compared to non-CVI population (p-value < 0.001).

Table 14: Direct observation of three screening signs in CVI.

Variables	No. of children (n, %)	p-value
No response to bright light	5 (17.86%)	**0.0260^{MC}***
Absent visual reflex	3 (10.71%)	**0.0350^{MC}***
Unresolved visual complexity	11 (39.29%)	**< 0.001^{MC}***

Abbreviation: MC – Chi square test with Monte Carlo simulation.

Table 10 reveals that visual reflex abnormalities were more common in CVI children (10.71%) compared to non-CVI subjects (0.95%, p-value = 0.0350), and the most common impairment observed during screening of CVI children was the behaviour of visual complexity (39.29%) which was statistically significant.

Table 15: Distribution according to CVI phase.

Total diagnosed CVI (n, %) 28 (21.05%)			
CVI Phase	**Number of children (n, %)**	**Moderate risk (n, %)**	**Severe risk (n, %)**
CVI PHASE I	17 (12.78%)	11 (64.71%)	6 (35.29%)
CVI PHASE II	10 (7.52%)	7 (70%)	3 (30%)
CVI PHASE III	1 (0.75%)	1 (100%)	0

After screening and thorough examination, the children were distributed according CVI phases. Table 11 reveals that 12.78% were in phase one CVI. Majority of the children in all phases belonged to moderate risk group. Phase one CVI was identified in most of the children who were determined to be at high risk.

Table 16: Association of CVI phases with postnatal history.

Variables	**CVI phase**			**p-value**
	Phase 1 (n, %)	**Phase 2 (n, %)**	**Phase 3 (n, %)**	
O2 Therapy	13 (76.47%)	8 (80%)	1 (100%)	0.9999^{MC}
Neonatal Jaundice	9 (52.94%)	4 (40%)	0	0.8341^{MC}
Convulsions	8 (47.06%)	4 (40%)	0	0.9999^{MC}
Documented Hypoglycemia	2 (11.76%)	4 (40%)	1 (100%)	0.0620^{MC}
Chorioamniotism	0	0 (0%)	0 (0%)	1^{MC}

Abbreviation: MC – Chi square test with Monte Carlo simulation.

Table 12 reveals that none of the associations between postnatal history and CVI phases were statistically significant (p-values >0.05).

Table 17: Comparison of TAC Vision Scores across CVI Phases based on risk status.

Risk Status	TAC vision (cycles/cm)	CVI phase			p-value
		Phase 1	Phase 2	Phase 3	
Moderate	Mean ± SD Median (Min, Max)	0.8 ± 0.96 0.32 (0, 2.4)	3.9 ± 4.2 2.6 (0, 9.8)	3.8	0.0795^{K}
Severe	Mean ± SD Median (Min, Max)	2.01 ± 3.87 0.32 (0 ,9.8)	0.32 ± 0.32 0.32 (0, 0.64)	-	0.7865^{MW}

Abbreviation: K – Kruskal Wallis test, MW – Mann Whitney U test.

Table 13 reveals that TAC vision scores showed no statistically significant differences across CVI phases within the analyzed risk categories (p-values > 0.05).

Table 18: MRI results in CVI based on gestational age

PRETERM (12 children)		**TERM (16 children)**	
MRI Finding	**Number of children (n, %)**	**MRI Finding**	**Number of children (n, %)**
Gliosis/ Cerebral Atrophy	6 (50%)	Hypoxic Ischemic Encephalopathy (HIE)	13 (81.25%)
Periventricular Leukomalacia	5 (41.67%)	Periventricular Leukomalacia	1 (6.25%)
Genetic	1 (8.33%)	Mass lesion	1 (6.25%)
		No significant pathology	1 (6.25%)

Table 14 summarizes the brain abnormalities of children diagnosed with CVI. Gliosis was the most frequent alteration in preterm children (50%) but hypoxic ischemic encephalopathy (HIE) was more prevalent in term children (81.25%).

DISCUSSION

The present study titled: Prevalence of Cerebral Visual Impairment and Ocular Visual Impairment in children aged 6 to 18 months attending High risk Baby Clinic in a tertiary care hospital: A cross-sectional study, was carried out from April 2023 to March 2024.

The purpose of this investigation was to evaluate associated risk factors and visual acuity among children attending the High-Risk Baby (HRB) clinic. The findings provide crucial insights into the prevalence, risk factors, and early indicators of cerebral visual impairment (CVI) and ocular visual impairment (OVI) among infants and young children.

This study comprised of 133 children, with a mean age of 10.34 ± 3.76 months. In our study, the predominance of male infants (69.92%) and the high incidence of preterm births (72.18%) are noteworthy. The mean birth weight was 1885.98 ± 694.96 grams. The present study consisted of 19 (67.86%) boys and 9 (32.14%) girls having CVI. There have been no reports of sex predilection in CVI in any pre-existing literature hence, this gender inequality is attributed to societal norms. Eleven months was the median age at presentation.

The most active and crucial period for ocular development in a foetus is between 6 months of pregnancy till term.[57] Even though majority of the children screened were preterm babies (72.18%), the diagnosis of CVI (57.14%) and OVI (66.67%) were seen more commonly in term babies. Birth weight of most of the diagnosed children was more than 1.5 kg. Majority of the OVI children (57.14%) were born via normal vaginal delivery whereas in CVI (53.57%) Caesarean section was more common. The incidence of CVI or OVI and gestational age did not directly correlate.

These results imply that the incidence of CVI is not substantially different between term and preterm infants and is not influenced by birth weight in the study. On the other hand, CVI is becoming the most frequent cause of childhood vision impairment. Children born too soon have a serious risk of brain injury, which can result in conditions like cerebral palsy and low intelligence quotient IQ.

A higher prevalence of consanguinity was noted among children with OVI (42.86%) than rest of the population and this difference was statistically significant ($p = 0.0090$) thus, underscoring genetic factors in ocular abnormalities. A minority of the CVI children also showed a history of consanguinity among parents (32.14%). This is in trend with the fact that consanguineous marriages have been linked to a higher predilection for ocular genetic disorders. [58]

Using the KIMS risk stratification model, majority, i.e., 75.94% of the children screened were classified as moderate-risk, while 24.06% fell under the severe-risk category. Amongst these, in contrast to 33.66% of children in the moderate risk category, nearly 46.87% of children in the severe risk category subsequently received a diagnosis of either CVI or OVI. This correlates with the fact that children deemed to be at severe risk for visual impairment after birth were diagnosed with either of the ocular conditions.[59]

The visual processing pathways, which make up 80% of the brain, are severely damaged by hypoxic insult from seizures. Convulsions were most common cause of perinatal insult in CVI phase I identified children (52%), whereas jaundice was the most common cause in phase III, according to a second study conducted in 2023 at our center among children attending Child Development Centers (CDC) between ages of six months and twelve years. According to the study, children who experienced convulsions in first few months after birth acquired a severe type of CVI (phase I),

which was unrelated to age or prematurity. The severity of CVI was not found to be influenced by a history of oxygen therapy or jaundice.[60]

A two-year study conducted in Raipur in 2022 assessed 405 patients between the ages of 3 months and 16 years. After determining the probable causes of CVI, the researchers found that 142 (35.1%) of the patients had hypoxic ischemic encephalopathy, 127 (31.3%) had seizures linked to brain damage, 44 (10.9%) had neonatal hypoglycaemia, 39 (9.6%) had structural neurological malformations, and 13 (3.2%) had infection.[61]

On evaluating the perinatal history in our screened population, it was found that a significant proportion of the children had received oxygen therapy (90.98%) and had neonatal jaundice (66.92%), suggesting perinatal complications as significant contributors to developmental challenges. Notably, 16.54% of these children experienced neonatal convulsions, which was significantly associated with neurological disorders, including CVI (38.89%) and CVI with coexisting OVI (50%) (p-value= 0.0299^{MC}**)**. Hypoxic-ischemic brain injury, often resulting from such neonatal complications, is a common cause of CVI. [62] The other factor analysed was documented hypoglycaemia (CVI= 22.22%, CVI with OVI= 30%, p = 0.5392^{MC}) but despite showing a similar trend, it was not found to be statistically significant. Similarly, history of jaundice was prevalent amongst all groups but was not found to have a significant correlation in our study. A possible explanation for this could be a smaller number of children in this group in comparison to convulsions on evaluation.

In our study, CVI was diagnosed in 18 children (13.53%), OVI in 11 (8.27%), CVI with co-existing OVI in 10 children (7.52%). The majority (70.68%) did not have either condition. Majority of the children screened (77.44%) had normal milestones. Among our patients, global developmental delay was fairly prevalent. It was

considerably higher in CVI diagnosed (66.67%) as well as CVI children with coexisting OVI (100%) ($p < 0.001$). None of the OVI cohort children were found to have delayed developmental milestones. This indicated a strong association between CVI and neurodevelopmental impairments which is in trend with pre-existing studies.

A study done in UK over a period of 2 years evaluating 558 children found a high incidence of ocular abnormalities with majority (12.5%) being diagnosed with strabismus, 2.9% suffered from sequelae of retinopathy of prematurity and other significant refractive errors (12.7%). It is observed that reports of strabismus are usually more in children born prematurely, regardless of the presence of ROP. [63]

A retrospective study done in Karnataka (2019) congenital paediatric cataract was diagnosed in 71.26% of the cases. Among these 12.90% were hereditary, 9.67% were associated with syndromes.[64]

In 2023, Kavitha V et al. conducted a cross-sectional study in Shimoga that evaluated 94 children aged one to eighteen. The study found that 88.29% of children with developmental delays had ocular abnormalities, with the most prevalent being refractive error (74.47%). Low birthweight and consanguineous marriage were frequently documented risk factors while epilepsy was the most prevalent systemic relationship.[65]

Over the course of five years in 2018, a study conducted in Vijayawada by Pehere NK et al. assessed 428 severely visually impaired children who were younger than three years old. Ocular visual impairment (OVI) alone was 56%, congenital cataract 13.1%, retinopathy of prematurity I 2.6%, optic atrophy 4.5%, congenital nystagmus 4.4%, congenital globe anomalies 5.2%, and high refractive errors 2.8%. Cerebral visual impairment (CVI) was 33% of the total.[66]

While evaluating for Ocular visual impairment in the present study, 11 (8.27%) of the total participants were found to have OVI with no coexisting CVI. Developmental cataract (45.45%) was the most common ocular abnormality diagnosed. Congenital glaucoma affected 2 children (18.18%), and retinopathy of prematurity affected the same number of patients. One child (9.09%) was diagnosed with esotropia, and another with congenital ptosis. This correlates with other studies done in the past.

CVI can be secondary to a great number of neurological disorder and can often coexist with ocular visual loss.[67] Among the 28 children diagnosed with CVI, ten (7.52%) children had coexisting ocular visual impairment as well. Strabismus was noted in eight of these cases with one having esotropia and seven having exotropia. Other anterior segment abnormality included one child with pendular, moderate amplitude nystagmus. It was also noted that one of the children had bilateral congenital cataract with microphthalmos secondary to CMV infection and microcornea. On evaluation of the posterior segment, five (17.86%) children were found to have abnormalities with four having temporal disc pallor. These findings are consistent with pre-existing studies.

The diagnosis of CVI is begins with direct observation and history of CVI specific behaviours from the caregivers and other family members who have frequent contact with the child.[68] The specific screening questions and three screening signs are adapted from the guidelines proposed by Dutton. [69]

In the study, evaluation for CVI was done with a five-point questionnaire and according to the responses it was noted that 82.14% of caregivers of CVI-affected children expressed concerns about their child's vision, compared to 7.62% of non-CVI caregivers ($p < 0.001$), emphasizing the role of caregiver perception in early identification. Behavioural screening responses to the other questions showed that

CVI children had significantly reduced eye contact (57.14%, $p < 0.001$) and smiling at caregivers (60.71%, $p < 0.001$). Direct observation of response to screening tests revealed that 17.86% of CVI children had an altered response to light sources, a key indicator of visual impairment. Moreover, 3 (10.71%) children had no reaction to visual reflex test and 11 (39.29%) had no visual complexity. This highlights how crucial it is to keep an eye out for warning signs and symptoms and follow the three observation signs for screening of neonates and infants for timely intervention.

Among the 28 children diagnosed with CVI in the present study, A majority (17, 61%) of the children fell under Phase I. Delay in hospital presentation and diagnosis may be the cause of the higher percentage of children in phase I. Majority of the children in all phases belonged to moderate risk group. Phase one CVI was identified in most of the children who were determined to be at high risk according to the standardized KIMS stratification model followed in the neonatal intensive care unit of our institute. This was consistent with the fact that prematurity and high-risk perinatal events predispose the child to develop visual impairment later in life. None of the associations between postnatal history and different phases of CVI were statistically significant (p-values >0.05).

"A study carried out by Sumalini et al. in, Telangana (2023), analysed 73 children in the 7-month to 7-year age range and revealed that grating visual acuity varied with age and was significantly different across the three phases of CVI. They also observed that the functional vision score significantly decreased by 2.8 points for every 1.0 logMAR increase (i.e., worsening) in grating acuity." [70]

On assessing that TAC vision scores in the present study, we found that it was significantly lower in CVI subjects, indicating poorer visual acuity (p-value < 0.001). The median vision observed was 0.54 cycles/cm. Moreover, five (17.86%) children

were unable to fixate and follow light or objects which was statistically significant (p-value < 0.001^{MC}*). Whereas, across various phases of CVI, it was noted that there were no statistically significant differences across the phases (p-values > 0.05). Children with OVI also exhibited a lower mean TAC vision score (1.11 ± 2.84 cycles/cm) and median of 0 cycles/cm (p-value < 0.001). Three (14.29%) children were not able to fixate and follow light or objects. This reinforces the impact of ocular abnormalities on visual function.

On evaluating the pre-existing literature, It was shown that the observed deficit in visual cortex activation in CVI is probably caused by early developmental brain injury.[71] Because periventricular white matter damage, sometimes referred to as periventricular leukomalacia, or PVL, is commonly associated with neurological impairment, prematurity is a high risk factor for developing CVI.[72] On the contrary, the main cause of CVI in term infants is perinatal hypoxia ischemia, which causes hypoxic ischemic encephalopathy (HIE) in these babies.[73] The brainstem, thalamic, hippocampal, and deep gray matter regions are the most frequently injured structures in HIE.[74]

All the children diagnosed with CVI in our study were followed up with an MRI to ascertain the cause of the disease. Among the brain abnormalities noted, gliosis was the most frequent alteration in preterm children (50%) followed by 41.67% of the children having Periventricular Leukomalacia. On the contrary, hypoxic ischemic encephalopathy (HIE) was more prevalent in term children (81.25%). This highlights the role of perinatal events leading to birth asphyxia as an important risk factor predisposing to CVI. One child (8.33%) in the preterm group was believed to have a genetic predisposition to CVI and one child (8.33%) in the term birth cohort did not show any significant brain pathology on imaging. These results are in agreement with

past research. Since all these children had a positive history of oxygen therapy and MRI findings suggestive of birth asphyxia, it warrants a detailed history of type on ventilation in postnatal period in the study.

STRENGTHS OF OUR STUDY:

In this study, early age of presentation in children diagnosed with Cerebral Visual Impairment reinforced the fact that HRB babies are screened in an ideal window of time. Moreover, patients from both urban and rural backgrounds were included in the study. The control group of children not diagnosed with either CVI or OVI provided an appropriate comparison among various groups.

LIMITATIONS AND RECOMMENDATIONS:

While this study provides valuable insights, it is limited by its cross-sectional design, which precludes longitudinal assessments of visual outcomes. A long-term follow-up study ensuing rehabilitation for functional vision is required to accumulate better data to analyze and predict the overall prognosis of the diagnosed children. Ocular findings such as accommodation, visual field, and contrast sensitivity data were not documented in our samples due to age group limitations which are equally significant and grossly affected in CVI. They also help guide our plan for rehabilitation of the affected children. Further studies can be planned while including these parameters to get a better evaluation of the effect of CVI in the diagnosed children. This study underscores the significant burden of CVI and OVI in high-risk infants, highlighting the need for early screening, timely intervention, and caregiver education. Addressing the risk factors identified in this study can aid in mitigating visual impairments and improving developmental outcomes in this vulnerable population.

CONCLUSION

The study highlights the importance of early screening for both CVI and OVI in high-risk infants as CVI and OVI were prevalent in nearly 30% of high-risk infants, with CVI being slightly more common than OVI.

The significant correlation of neonatal convulsions, birth asphyxia and developmental delays with CVI suggests that targeted interventions for at-risk neonates are necessary. History of postnatal hypoglycemia showed similar trends but was not found to be statistically significant.

Furthermore, the higher prevalence of OVI in children with positive consanguinity suggests the need for genetic counselling and ophthalmic evaluations in families with a history of ocular conditions.

Both CVI and OVI resulted in significantly impaired vision, reinforcing the need for early diagnosis and intervention.

SUMMARY

.The present study, conducted from April 2023 to March 2024, was aimed at investigating the prevalence, risk factors, and early indicators of Cerebral Visual Impairment (CVI) and Ocular Visual Impairment (OVI) among infants aged 6 to 18 months attending the High-Risk Baby (HRB) clinic in a tertiary care hospital in South India. This cross-sectional study included 133 children and focused on the impact of perinatal events on visual impairment. The key findings of the study can be summarized under the following headings:

Risk Stratification after birth

Using the KIMS risk stratification model, majority of the children screened were classified as moderate-risk. On the contrary, a significant proportion of children in the severe risk category subsequently received a diagnosis of either CVI or OVI. This reinforced the fact that children deemed to be at severe risk after birth go on to develop either of the ocular abnormalities later in life.

Prevalence of CVI and OVI

After thorough evaluation, CVI was diagnosed in 13.53%, OVI in 8.27%, and CVI with coexisting OVI in 7.52% children. In conclusion, CVI and OVI were prevalent in nearly 30% of high-risk infants with CVI being more common than OVI. Strabismus was the most often observed ocular abnormality in CVI cases with exotropia being the most common. On the contrary, developmental cataract was the most frequent ocular abnormality in the OVI cohort, followed by congenital glaucoma and retinopathy of prematurity.

Association of visual impairment with perinatal risk factors

A significant proportion of the children had received oxygen therapy and had neonatal jaundice suggesting perinatal complications as significant contributors to developmental challenges. Neonatal oxygen therapy, jaundice, and convulsions were significantly correlated with CVI and CVI-OVI combined cases. Hypoglycemia also did not show a statistically significant association with CVI despite showing similar trends as the positively correlated factors. These factors however, did not show a significant association with CVI phases. In conclusion, CVI was strongly linked to birth asphyxia and convulsions while OVI was more commonly associated with genetic factors. A higher prevalence of consanguinity was noted among children with OVI which was statistically significant thus, underscoring the role of genetic factors in ocular abnormalities

Global developmental delay and Visual impairment

Global developmental delay was strongly associated with CVI and CVI with coexisting OVI. On the contrary, none of the OVI children were found to have delayed developmental milestones. This indicated a strong association between CVI and neurodevelopmental impairments which was in trend with pre-existing studies

Screening and Diagnosis of CVI

Caregivers of CVI-affected children frequently reported visual concerns, reduced eye contact and lack of smiling. Direct screening tests showed altered responses to light, lack of visual reflex response and difficulty perceiving visual complexity. This emphasizing the role of caregiver perception in early identification as well as the need to keep an eye out for warning signs and symptoms.

Visual acuity in CVI and OVI

After assessing the TAC vision scores in the present study, we found that it was significantly lower in both CVI and OVI subjects, indicating poorer visual acuity. A statistically significant proportion of children in either groups were unable to fixate and follow light or objects. This reinforces the impact of ocular abnormalities on visual function.

Imaging in CVI

MRI results of CVI children revealed gliosis and periventricular leukomalacia as the most common finding in preterm children whereas hypoxic-ischemic encephalopathy was more frequent in term infants. This highlights the role of perinatal events leading to birth asphyxia as an important risk factor predisposing to CVI.

To summarize, early screening for CVI and OVI in high-risk infants is crucial. Neonatal convulsions and perinatal hypoxia are key indicators for early intervention. Genetic counselling is recommended for families with consanguineous marriages to reduce OVI risk. Early diagnosis and intervention are essential for improving visual and developmental outcomes in these children. Further research with long-term follow-up is needed to assess visual outcomes and rehabilitation strategies.

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ANNEXURE – I - INFORMED CONSENT FORM

TITLE OF THE STUDY: PREVALENCE OF CEREBRAL VISUAL IMPAIRMENT AND OCULAR VISUAL IMPAIRMENT IN CHILDREN AGED 6 TO 18 MONTHS ATTENDING HIGH RISK BABY CLINIC IN A TERTIARY CARE HOSPITAL: A CROSS-SECTIONAL STUDY

Introduction: Your child/ward is being invited to participate in this study to determine the prevalence of cerebral and ocular visual impairment amongst children attending high risk baby clinic at KLEs Dr. Prabhakar Kore Hospital.

Explanation of procedure: If, you agree to be part of the research study, you will be asked the relevant history and your child/ward will be subjected to relevant clinical examination and investigations.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: Your child/ward will not be eligible for any kind of monetary benefits or free services by virtue of participation in the study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data

collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not be paid / offered any gifts /incentives for participation of your child/ward in this study.

Cost of investigations done during the course of study will be paid by the Participant.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed. The results of the study would be forwarded to the KAHER, Belgaum as part of requirement towards the completion of MS degree, review and publishing.

Questions: In case of any questions with regard to this study, you are free to contact:

If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waiving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **"PREVALENCE OF CEREBRAL VISUAL IMPAIRMENT AND OCULAR VISUAL IMPAIRMENT IN CHILDREN AGED 6 TO 18 MONTHS ATTENDING HIGH RISK BABY CLINIC IN A TERTIARY CARE HOSPITAL: A CROSS-SECTIONAL STUDY"**. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE – II - PROFORMA

PARENT DETAILS: **Date:**

Name Age:

Address: Mobile no:

Education status:

Occupation: Father: Mother:

CHILD DETAILS:

Name:

Age: ☐☐

Gender: Male ☐ Female ☐

Gestation Period: weeks PRETERM/ TERM

Birth weight - _____ grams

Risk Status:

Mode of delivery:

- ☐ Normal vaginal delivery (0)
- ☐ Assisted vaginal delivery (1)
- ☐ Caesarean section (2)
  - o Elective
  - o Emergency

- ☐ Moderate
- ☐ Severe

History of:	Oxygen Therapy	No (0)/ Yes (1)
	Neonatal Jaundice	No (0)/ Yes (1)
	Convulsions	No (0)/ Yes (1)
Documented Hypoglycaemia		No (0)/ Yes (1)
	Chorioamniotism	No (0)/ Yes (1)

Milestones:

Normal (0) Delayed(1)

Family History: Pedigree

SCREENING QUESTIONS:

		Yes	No
1.	Does the caregiver have any concerns about the way child sees?		
2.	Does the child look at the caregiver's face and make eye contact?		
3.	Does the child smile and respond to your smile towards them?		
4.	Does the child stare at light sources?		
5.	Does the child tilt their head to look at something?		

BEHAVIORAL SCREENING:

		Yes	No
1.	Light gazing: The child closing eyes to intense light		
2.	Visual reflex differences: The child blinking on touching the nose bridge		
3.	Visual complexity: The child making eye contact		

OCULAR EXAMINATION:

Extra ocular movements	Normal (0)	Restricted (1)
Visual axes	Parallel (0)	Unparallel (1)

Binocular vision: Orthotropia (0) Esotropia (1) Exotropia (2)

Nystagmus:

Absent (0)	Present (1)

Visual acuity	OD	OS
Fixes/ Follows light		
Vision with Teller's acuity card		

Anterior Segment: Normal (0) Abnormal (1)

Posterior Segment: Normal (0) Abnormal (1)

CVI CHARACTERISTICS EVALUATION:

		Not Resolved		**Resolving**		**Resolved**
1.	Colour	0	0.25	0.50	0.75	1
2.	Movements	0	0.25	0.50	0.75	1
3.	Latency	0	0.25	0.50	0.75	1
4.	Visual fields	0	0.25	0.50	0.75	1
5.	Complexity	0	0.25	0.50	0.75	1

6.	Light gazing	0	0.25	0.50	0.75	1
7.	Distance viewing	0	0.25	0.50	0.75	1
8.	Visual reflexive responses	0	0.25	0.50	0.75	1
9.	Visual novelty	0	0.25	0.50	0.75	1
10.	Visual motor	0	0.25	0.50	0.75	1

CVI PHASE:

Phase I	Phase II	Phase III

MRI:

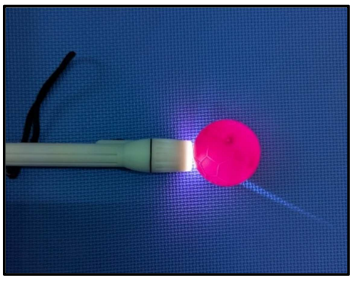
NAME OF INVESTIGATOR:

SIGNATURE:_____

NAME OF GUIDE:

SIGNATURE:_____

ANNEXURE III : PHOTOGRAPHS



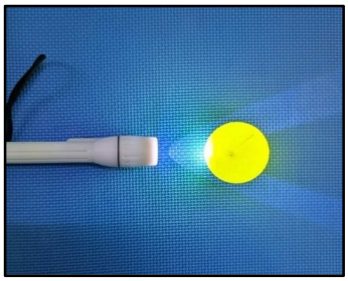


Photo 1: Various coloured illuminated plastic balls for testing color preference

Photo 2: Mylars

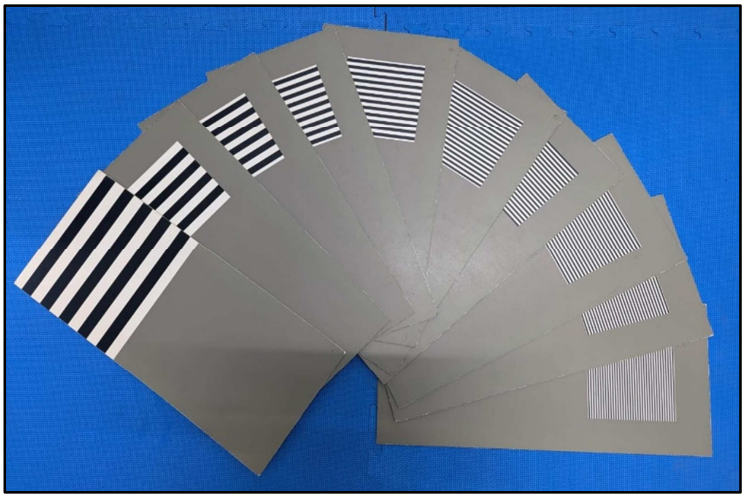


Photo 3: Teller's Acuity Charts

Photo 4: Colour preference and visual acuity examination

ANNEXURE IV: KEY TO MASTER CHART

Prematurity:

Preterm (1) Term (0)

Mode of Delivery:

Normal vaginal delivery (0), Assisted vaginal delivery (1), Caesarean section (2)

Risk status:

Moderate (0) Severe (1)

Consanguinity:

Absent (0) Present (1)

Questions:

Yes (1) No (0)

Fixates and follows:

Yes (0) No (1)

CVI phase

No CVI (0) Phase one (1) Phase two (2) Phase three (3)

ANNEXURE V:

MASTER CHART

S. No.	OP/ IP Number	Consanguinity	Age (in months)	Gender	Gestation Period (in weeks)	Term/ Preterm	Birth Weight (in grams)	Mode of Delivery	Risk Status	O2 Therapy	Neonatal Jaundice	Convulsions	Documented Hypoglycemia	Choriamniotism	Milestones	Any concern	Eye contact	Smiles	Light sources	Tilts head	Light gazing	Visual reflex	Visual Complexity	Extraocular Movements	Visual axes	Binocular Vision	Nystagmus	Fixates and follows	TAC vision (in cycles/cm)	Anterior Segment	Posterior Segment	Total CVI score	CVI Phase
1	7148339	0	11	Male	32	1	1500	2	0	1	0	0	0	0	1	1	0	1	1	0	1	1	1	0	0	0	0	0	9.8	0	0	6.75	2
2	7173718	0	14	Male	37	0	2500	2	0	0	0	1	0	0	1	1	0	0	1	1	1	1	0	0	1	2	0	0	0	1	1 (BE Disc pallor)	2	1
3	7197818	0	12	Male	32	1	2000	0	0	0	0	0	0	0	1	0	1	1	1	1	1	1	1	0	0	0	0	0	1.6	0	0	5.75	2
4	7094010	0	11	Female	40	0	2900	0	1	1	0	1	1	0	1	1	1	1	1	1	1	1	0	0	1	1	0	0	0	0	1	4.75	2
5	7093947	0	5	Female	28	1	980	2	1	1	0	0	0	0	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0.64	0	0	2	1
6	7245771	1	11	Male	35	1	2200	2	1	1	1	0	0	0	1	1	1	1	1	1	0	1	1	0	0	0	0	0	0	0	0	2	1
7	7214712	0	7	Female	34	1	2500	0	1	1	1	0	1	0	1	1	1	1	1	0	1	1	1	0	0	0	1	0	0.32	0	0	3.25	2
8	7111669	1	12	Female	37	0	2550	0	0	1	0	0	0	0	1	1	1	1	1	0	1	1	1	0	0	0	0	0	0.23	0	0	12.5	2
9	7258940	1	18	Female	32	1	1365	2	1	1	1	0	1	0	1	1	0	0	1	0	1	0	0	0	1	1	0	1	0	1	0	3.75	1
10	7173824	0	11	Male	31	1	1500	2	0	1	1	0	0	0	1	1	0	1	1	0	1	1	1	0	0	0	0	0	2.4	0	0	3.5	1
11	7103427	1	17	Male	37	0	2490	0	1	0	1	0	0	0	1	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	1 (Large Disc and cup)	3.75	1
12	7073292	0	5	Male	37	0	3200	0	0	1	1	1	0	0	0	1	1	1	1	1	0	1	1	0	0	0	0	0	1.3	0	0	1.75	1
13	1177794	1	8	Female	37	0	2500	0	0	0	1	1	1	0	1	0	1	1	1	1	1	1	1	0	0	0	0	0	3.3	0	0	4	2
14	6834862	1	18	Male	37	0	4000	2	0	1	0	0	0	0	0	1	0	0	0	1	1	1	0	0	0	0	0	1	0	0	0	2.75	1
15	7264942	1	12	Female	37	0	2700	2	0	1	1	0	0	0	1	1	0	0	1	1	1	1	0	0	1	1	0	0	0.32	1	0	3.25	1
16	10035909	1	11	Male	37	0	2800	2	0	0	0	1	0	0	1	0	1	0	1	1	1	1	1	0	0	0	0	0	2.4	0	0	3	1
17	7314055	0	9	Female	37	0	2000	0	0	1	0	1	0	0	1	1	0	0	1	0	1	1	0	0	0	0	0	0	0.43	0	0	2.5	1
18	7303317	0	7	Male	37	0	3400	2	0	1	1	1	0	0	1	1	0	0	1	1	1	1	1	0	1	2	0	1	0	0	1(temporal pallor)	4	2
19	7323787	1	8	Male	36	0	2300	0	0	1	0	1	0	0	1	1	1	1	1	0	1	1	1	0	0	0	0	0	2.6	0	0	3	2
20	7329674	0	18	Male	40	0	1920	0	0	1	1	0	0	0	1	1	1	1	1	1	1	1	1	0	1	2	0	0	0.32	1	0 (B/L oval disc)	2	1
21	7093447	0	12	Male	35	1	1900	2	1	1	1	1	1	0	1	1	0	0	1	0	1	1	0	0	0	0	0	1	0	0	1 (Large disc, temporal pallor)	2.75	1
22	7413305	0	6	Male	37	0	1350	0	0	1	1	0	0	0	1	1	0	0	1	1	0	1	0	0	1	2	0	1	0	1	0	2.5	1
23	7099020	0	18	Male	37	0	4000	2	0	1	0	0	1	0	0	1	1	1	0	0	1	1	1	0	0	0	0	0	3.8	0	0	6.75	3
24	10052292	0	14	Female	31	1	1500	2	0	0	0	1	0	0	0	1	0	0	1	1	1	1	0	0	0	0	0	0	1.6	0	0	1.75	1
25	7199844	0	18	Male	38	0	2750	0	0	1	0	1	0	0	1	1	0	0	1	1	0	0	0	0	1	2	0	0	0	1	0	0	1
26	6925832	0	15	Male	30	1	2000	0	0	1	1	0	0	0	0	1	1	1	1	0	1	1	1	0	0	0	0	0	9.8	0	0	5.25	2
27	7153922	0	9	Male	33	1	2260	2	1	1	0	1	0	0	1	1	1	1	1	0	1	1	1	0	1	2	0	0	1.6	1	0	1	1
28	7154012	0	6	Male	34	1	1900	2	1	1	0	0	1	0	1	1	1	1	1	0	1	1	1	0	0	0	0	0	0.64	0	0	6.25	2
29	7229889	1	7	Male	37	0	2900	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	0	1	0	0	0
30	6502311	1	18	Male	37	0	1800	0	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	1.6	0	1	0	0
31	10022326	0	7	Male	30	1	1500	0	0	1	0	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	0	1	0	0	0
32	7255378	1	7	Male	29	1	1700	0	0	1	0	0	0	0	0	1	1	1	1	0	1	1	1	0	0	0	0	0	0	0	1(B/L fully vascularized retina Zone III, no Plus Disease with white patches)	S	0
33	7305914	0	6	Male	27	1	1100	0	1	1	1	0	0	0	0	0	1	1	1	1	1	1	1	0	0	0	0	0	0.8	0	1 (ROP +)	0	0

S. No.	OP/ IP Number	Consanguinity	Age (in months)	Gender	Gestation Period (in weeks)	Term/ Preterm	Birth Weight (in grams)	Mode of Delivery	Risk Status	O2 Therapy	Neonatal Jaundice	Convulsions	Documented Hypoglycemia	Choriamniotism	Milestones	Any concern	Eye contact	Smiles	Light sources	Tilts head	Light gazing	Visual reflex	Visual Complexity	Extraocular Movements	Visual axes	Binocular Vision	Nystagmus	Fixates and follows	TAC vision (in cycles/cm)	Anterior Segment	Posterior Segment	Total CVI score	CVI Phase
34	7278843	1	10	Female	37	0	1900	2	0	1	0	0	0	0	0	1	1	1	1	0	1	1	1	0	0	0	0	0	1.3	1	0	0	0
35	7278848	1	10	Female	37	0	2000	2	0	1	0	0	0	0	0	1	1	1	1	0	1	1	1	0	0	0	0	0	0.84	1	0	0	0
36	7276105	1	17	Male	37	0	2500	0	0	1	0	0	0	0	0	1	0	0	1	1	0	0	0	0	0	0	0	0	0	1	0	0	0
37	10041818	0	18	Male	37	0	2000	2	0	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	1	1	0	0	0	1	0	0	0
38	6965997	1	11	Male	38	0	2000	0	1	1	1	0	1	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	1	0	0	0
39	7210617	0	12	Male	31	1	2000	0	0	1	1	0	0	0	0	1	1	1	0	1	1	1	1	0	0	0	0	0	13	1	0	0	0
40	1181701	1	12	Male	28	1	2000	2	1	1	0	1	0	1	0	0	1	1	0	1	1	1	1	0	0	0	0	0	6.8	0	0	0	0
41	1181712	0	7	Female	32	1	1200	2	1	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
42	1181778	1	6	Female	39	0	2680	0	0	1	0	1	0	0	1	1	0	0	1	0	1	1	0	0	0	0	0	0	1.6	0	0	0	0
43	6740148	0	11	Male	31	1	1700	0	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
44	6942561	0	6	Male	28	1	1400	2	1	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	4.7	0	0	0	0
45	6905775	0	7	Female	38	0	2480	1	1	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	3.1	0	0	0	0
46	6821836	1	9	Male	32	1	1980	2	1	1	1	0	0	0	0	0	1	1	0	1	1	1	1	0	0	0	0	0	3.2	0	0	0	0
47	6831370	0	9	Female	30	1	1000	2	1	0	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	2.4	0	0	0	0
48	6605292	0	14	Male	28	1	1000	0	1	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
49	6394416	0	12	Female	28	1	735	0	1	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	2.4	0	0	0	0
50	6823259	0	12	Male	29	1	1200	0	1	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
51	6787397	0	10	Male	32	1	1400	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
52	6942764	0	11	Male	39	0	2700	0	0	0	1	0	1	0	0	1	1	1	1	1	0	1	0	0	0	0	0	0	3.2	0	0	0	0
53	6950421	0	6	Male	32	1	2850	0	0	1	1	1	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	0.42	0	0	0	0
54	1181502	0	14	Male	27	1	1000	0	0	0	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
55	7153197	0	6	Male	34	1	2000	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
56	7153188	0	6	Female	34	1	1700	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
57	1165590	0	10	Female	32	1	2100	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
58	1165583	0	10	Male	32	1	2100	2	1	1	0	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
59	6550665	0	11	Male	28	1	1900	2	0	0	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	13	0	0	0	0
60	6935693	0	11	Female	31	1	1400	0	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
61	6991875	0	9	Male	28	1	926	2	1	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
62	6965844	0	9	Female	29	1	940	2	1	1	0	0	0	0	0	0	1	1	0	1	1	1	1	0	0	0	0	0	2.4	0	0	0	0
63	6974060	0	10	Male	30	1	1200	2	1	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
64	6794629	0	12	Male	31	1	1550	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
65	6794631	0	12	Female	31	1	1520	2	0	1	1	0	0	0	0	0	0	1	1	0	1	1	1	1	0	0	0	0	1.3	0	0	0	0
66	7109237	1	7	Female	34	1	1500	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
67	7109298	1	7	Male	34	1	2500	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	2.4	0	0	0	0
68	7127777	0	7	Female	33	1	1025	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
69	7128050	0	7	Female	33	1	1025	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
70	7172403	0	6	Male	34	1	1140	2	0	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	2.4	0	0	0	0
71	6809587	0	16	Male	28	1	1000	0	1	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
72	7095286	0	8	Male	30	1	1805	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0

S. No.	OP/ IP Number	Consanguinity	Age (in months)	Gender	Gestation Period (in weeks)	Term/ Preterm	Birth Weight (in grams)	Mode of Delivery	Risk Status	O2 Therapy	Neonatal Jaundice	Convulsions	Documented Hypoglycemia	Choriamniotism	Milestones	Any concern	Eye contact	Smiles	Light sources	Tilts head	Light gazing	Visual reflex	Visual Complexity	Extraocular Movements	Visual axes	Binocular Vision	Nystagmus	Fixates and follows	TAC vision (in cycles/cm)	Anterior Segment	Posterior Segment	Total CVI score	CVI Phase
73	7279853	0	10	Male	32	1	2250	0	0	1	0	0	0	0	1	0	1	1	1	0	1	1	1	0	0	0	0	0	2.4	0	0	0	0
74	6690043	0	18	Male	37	0	3300	2	1	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
75	7090015	0	7	Female	35	1	1346	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	2.4	0	0	0	0
76	7117549	0	6	Male	37	0	1500	2	0	1	1	0	0	0	0	1	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
77	6596435	0	18	Male	33	1	2000	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
78	6932604	0	11	Male	34	1	1800	2	0	1	1	0	1	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
79	6932605	0	11	Male	34	1	1900	2	0	1	1	0	1	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
80	7172378	0	7	Male	32	1	2060	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
81	7216032	0	6	Male	30	1	2030	0	0	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
82	7117441	0	8	Male	38	0	1600	2	0	1	0	1	1	0	1	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
83	1186170	1	11	Male	28	1	909	2	1	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	13	0	0	0	0
84	10042740	0	17	Male	37	0	3350	0	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
85	7153922	0	7	Male	31	1	2260	0	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
86	7172768	0	6	Male	32	1	1020	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
87	6814584	0	16	Male	32	1	1680	0	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	13	0	0	0	0
88	7046367	0	10	Male	32	1	1590	0	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
89	7093887	0	9	Male	32	1	1860	0	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
90	6983611	0	12	Male	30	1	1520	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
91	6983610	0	12	Male	30	1	1740	2	0	1	1	0	1	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
92	7190615	0	6	Male	32	1	1700	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
93	7109791	1	8	Male	32	1	1100	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
94	7190601	0	6	Female	31	1	1220	2	0	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
95	6935108	0	13	Female	31	1	1900	0	0	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
96	6756647	0	17	Male	30	1	1300	2	0	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	19	0	0	0	0
97	6722679	0	17	Female	37	0	1409	2	0	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	13	0	0	0	0
98	7021351	0	7	Male	26	1	750	0	1	1	1	1	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
99	6929320	0	13	Female	36	1	1700	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
100	6852442	0	12	Female	30	1	1340	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
101	6993994	1	10	Male	31	1	1800	2	1	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
102	7172768	1	6	Male	28	1	1100	2	1	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
103	7118712	0	9	Male	32	1	1500	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
104	6445417	1	18	Male	32	1	2200	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
105	7118714	0	9	Female	32	1	1500	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	2.4	0	0	0	0
106	6935685	0	12	Female	30	1	1600	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
107	7002996	0	10	Female	33	1	3000	0	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
108	7172756	0	6	Male	33	1	2080	0	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
109	1181412	0	13	Male	31	1	1340	2	0	1	0	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	2.4	0	0	0	0
110	1181411	0	13	Male	31	1	1350	2	0	1	1	1	1	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
111	7147532	0	15	Male	32	1	1380	2	0	0	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0

S. No.	OP/ IP Number	Consanguinity	Age (in months)	Gender	Gestation Period (in weeks)	Term/ Preterm	Birth Weight (in grams)	Mode of Delivery	Risk Status	O2 Therapy	Neonatal Jaundice	Convulsions	Documented Hypoglycemia	Choriamniotism	Milestones	Any concern	Eye contact	Smiles	Light sources	Tilts head	Light gazing	Visual reflex	Visual Complexity	Extraocular Movements	Visual axes	Binocular Vision	Nystagmus	Fixates and follows	TAC vision (in cycles/cm)	Anterior Segment	Posterior Segment	Total CVI score	CVI Phase
112	7147598	0	7	Male	37	0	4000	2	0	1	0	1	0	0	1	0	1	1	1	0	0	1	1	0	0	0	0	0	3.2	0	0	0	0
113	7148110	0	7	Male	32	1	1500	2	0	1	0	0	0	0	0	0	1	1	1	1	1	1	1	0	0	0	0	0	6.5	0	0	0	0
114	7097355	0	11	Male	37	0	3000	0	0	0	1	0	0	0	1	0	1	1	1	0	0	1	1	0	0	0	0	0	9.8	0	0	0	0
115	7147321	0	12	Female	32	1	1960	0	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
116	7147222	0	7	Male	31	1	1000	2	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
117	7046367	0	7	Male	32	1	1590	0	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	1.3	0	0	0	0
118	7325106	0	6	Male	30	1	1800	0	0	1	0	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
119	7153922	0	6	Female	30	1	2280	0	0	1	1	0	0	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	3.2	0	0	0	0
120	6862697	0	14	Male	37	0	2800	0	0	1	1	1	0	0	1	0	1	1	0	0	1	1	1	0	0	0	0	0	4.8	0	0	0	0
121	7021351	0	7	Male	26	1	750	0	1	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
122	10018930	0	6	Male	39	0	3050	2	0	1	1	1	0	0	1	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
123	7032783	0	10	Male	32	1	1750	0	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
124	6879696	0	14	Female	32	1	2900	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
125	7102289	0	8	Female	30	1	1300	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
126	7102290	0	8	Male	30	1	1250	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
127	7172403	0	6	Male	30	1	1150	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
128	7172378	0	6	Male	30	1	2060	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	1.6	0	0	0	0
129	7216032	0	6	Male	35	1	2030	0	0	1	0	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
130	6948580	0	12	Female	37	0	2300	2	0	1	0	1	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0
131	6935685	0	13	Female	36	1	1600	2	0	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
132	7153922	0	8	Male	33	1	2260	0	0	1	1	0	0	0	1	0	1	1	0	0	1	1	1	0	0	0	0	0	6.5	0	0	0	0
133	6798058	0	16	Female	39	0	2500	0	1	1	1	0	0	0	0	0	1	1	0	0	1	1	1	0	0	0	0	0	9.8	0	0	0	0