PREVALENCE AND PATTERN OF CEREBRAL VISUAL IMPAIRMENT AMONGST CHILDREN ATTENDING KENYATTA NATIONAL HOSPITAL EYE CLINIC

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DECLARATION

This dissertation is wholly original with no submissions for degrees at any other institution.

Signed _____ Date 2/05/2023

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APPROVAL

As university supervisors, we have given our permission for the submission of this dissertation.

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DEDICATIONS

This book is dedicated to Jimmy and Amira who have been patient with me all through.

To God, it’s only through His mercy and grace that I have made it this far.

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LIST OF ABBREVIATIONS

ADHD	Attention deficit hyperactive disorder
BCVA	Best corrected visual acuity
C/S	Caesarean section
CP	Cerebral palsy
CVI	Cerebral visual impairment
HIE	Hypoxic ischemic encephalopathy
IUGR	Intrauterine growth retardation
KNH	Kenyatta National Hospital
MR	Mental retardation
NBU	Newborn Unit
NICU	Neonatal intensive care unit
OVI	Ocular vision impairment
PICU	Pediatric intensive care unit
PROM	Premature rupture of membranes
RE	Refractive errors
ROP	Retinopathy of prematurity
UNICEF	United Nations Children's Fund
VA	Visual acuity
WHO	World Health Organization

ABSTRACT

Background: In the absence of injury to the anterior afferent visual pathways or the ocular structures, cerebral visual impairment (CVI) is characterized by vision loss caused by damage to the retro-geniculate pathway or by vision loss that is more than anticipated given the severity of ocular pathology**.** Ocular pathologies such as refractive errors may coexist but insufficient to cause severe vision impairment. Common causes of CVI include birth asphyxia, meningitis and hydrocephalus. Studies on CVI in Sub Sahara Africa are limited as it is listed under other causes of visual impairment or blindness and not as an individual entity. Several patients had been diagnosed with cerebral visual impairment at Kenyatta National Hospital eye clinic however, no audit of the condition had been done.

Objective: To describe the prevalence and pattern of cerebral visual impairment in children attending Kenyatta National Hospital eye clinic.

Design: It was a retrospective, hospital-based study.

Study location: The study was done at Kenyatta National hospital eye clinic.

Methods: The study included all children with CVI seen at Kenyatta National hospital eye clinic from 1st January 2019 to 28th February 2021. A standardized questionnaire was used to obtain patient data. The variables of interest included known causes of cerebral visual impairment, age at diagnosis, age at presentation, associated non-ocular co-morbidities, ocular examination findings and refractive status. Prevalence of CVI was calculated from this study population.

Data was analyzed using SPSS Version 24 software. Descriptive statistical analysis and inferential analysis was used.

Results were presented in text, tables, graphs and charts.

Results: A total of 674 children were reviewed and 53 children met the study inclusion criteria. The prevalence of CVI was 7.9%.The male to female ratio was 1.4:1with the mean age at presentation of 25.9 months. The main presenting complaint was poor vision (100%). The main known cause of CVI was meningitis/encephalitis (41.4%) followed by birth asphyxia (31%). Half of the children were not fixing or following light. The main ocular examination finding was optic atrophy (39.6%). The main refractive error was simple hyperopia (41.7%). The commonest

non-ocular co-morbidity was delayed milestones/motor impairment (61.5%). Some children had more than one non-ocular co-morbidity.

Conclusion: All children presented with poor vision. The commonest known cause was meningitis/encephalitis. More than half of the patients had severe visual impairment. The main ocular finding was optic atrophy. Simple hyperopia was the commonest refractive error. The commonest non-ocular co-morbidity was developmental delay/motor impairment.

1.0: BACKGROUND INFORMATION

1.1 INTRODUCTION

A child is a person under the age of sixteen[1]. Blindness is defined as corrected visual acuity in the better eye of less than 3/60, while severe visual impairment is defined as corrected visual acuity in the better eye of less than 6/60, according to the World Health Organization (WHO) as shown in Table 1[1].

Table 1: WHO classification of vision

Distance vision classification	Visual acuity
Mild	Worse than 6/12- 6/18
Moderate	Worse than 6/18-6/60
Severe	Worse than 6/60- 3/60
Blindness	Worse than 3/60

In 2001, there were 37 million blind people and 124 million with impaired eyesight (excluding those with uncorrected refractive errors). Amongst the 37 million, 1.4 million were aged 0-14 years. Asia and Africa were home to two-thirds of the 1.4 million children.[2].

More recently, under-5 mortality rates and hence blindness prevalence rates have declined in most countries. Globally, the number of blind children has reduced by 10% to 1.26 million, of whom 0.42 million live in Sub- Saharan Africa. However, there is an increase in the number of blind children in Sub-Saharan Africa by 31%[3].

A blind child is more likely than a child who is not blind to grow up in a poor socioeconomic environment, to have delayed or disordered development, to have more frequent hospital admissions and to die during childhood[4].

A blind child will have a lifetime of blindness, therefore increasing the family's emotional, social, and financial costs. As a result, the number of 'blind years' in children due to all causes of blindness is about equivalent to the number of 'blind years' in adults with cataracts. Many of the

causes of childhood blindness can be avoided or treated. Furthermore, several causes of childhood blindness result in child mortality, implying a strong relationship between the prevention of childhood blindness and child survival. As a result, the World Health Organization's VISION 2020- The Right to Sight program has made preventing blindness a top priority.[1].

The principal causes of childhood blindness differ greatly across regions, with socioeconomic development and primary health care and eye care facilities availability playing a key role. Optic nerve and upper visual pathway lesions are the commonest causes of blindness in developed nations, whereas corneal scarring secondary to measles, vitamin A deficiency, harmful traditional eye medicine use and ophthalmia neonatorum are the most common causes in developing nations[1].

Poor perinatal care and high childhood morbidity in low income areas have led to an increase in prevalence of cerebral visual impairment[5]. Studies on cerebral visual impairment in Sub Sahara Africa are limited as it is listed under other causes of visual impairment and blindness and not as an individual entity.

1.2 CEREBRAL VISUAL IMPAIRMENT (CVI)

This is described as vision loss that is more than anticipated for the level of ocular pathology, or vision loss that results from injury to the retro-geniculate pathway without disruption to the anterior afferent visual pathways or ocular structures [6][7][8].

In the absence of anterior pathway disease and a clinical history or direct sign of neural injury, the diagnosis of children with diminished visual acuity may be straightforward. However, it may be difficult to distinguish visual impairment caused by CVI or other developmental problems in some complex presentations. This has led to the contested proposition that a visual processing deficiency must be established by sufficient difference between cognitive and visual perception quotients [6].

Children with CVI fall into three categories:

- Children with profound vision loss
- Those with cognitive and motor challenges and impaired but functional vision
- Those with impaired but functional vision who work near their expected academic level for age[9].

CVI is associated with neuro-developmental and systemic co-morbidities which cause cognitive, speech and motor delays. These include cerebral palsy, chromosomal disorders, metabolic disorders and brain malformations such as microcephaly, schizencephaly and congenital hydrocephalus[10].

Many milestone delays can be attributed to movement or attentional system impairment, especially when there is a history of intellectual or motor impairment. The role of vision in the noticed delay is rarely discussed[11].

1.3 PREVALENCE

CVI is more common in high-income societies than in low-income societies. It is the leading cause of childhood blindness in high and middle-income countries[4][12]. In comparison to children in the general population, it is a prominent cause of vision loss in children already diagnosed with severe vision impairment i.e. those in schools for the blind[1].

There is no clear data on the global burden of CVI. However, studies have been done in a few regions in the world especially in visually impaired children.

A retrospective study by Huo et al done in a large American pediatric ophthalmology practice over 15 years, reviewed records of 7200 patients of whom 2.4% (170 patients) had CVI. The average age of patient was 3 years[13].

Rajedra et al retrospectively studied causes of childhood vision impairment at an Eritrean hospital and identified 249 (1.1%) with vision impairment of whom 3.6% had CVI following meningitis and other systemic illness[14].

CVI was prominent in 33% of 428 children with severe visual impairment in a retrospective study by Pehere et al at a tertiary eye hospital in South India, with an age range from 4 months to 3 years[15].

In a cross-sectional, hospital based study, Njambi et al (2009) found causes of visual impairment at a Kenyan national referral center occupational therapy clinic were CVI (48.7%), strabismic amblyopia (13.4%) and ocular causes (10.2%)[16]. Similarly, Smitha et al found CVI in 50% of children with cerebral palsy in their cross-sectional study at an Indian tertiary center[17].

2.0: LITERATURE REVIEW

2.1 PATTERN OF CEREBRAL VISUAL IMPAIRMENT

Perinatal hypoxia and ischemia are the most common mechanisms of injury, and the pattern of harm is largely determined by the age when the insult occurs. As a result, it differs between preterm and full-term newborns. In term babies, there is infarction of frontal and parieto-frontal cortices due to hypo perfusion caused by hypoxia- induced loss of vascular flow auto regulation. In preterm babies, damage to periventricular deep white matter leads to periventricular leukomalacia [8][18].

Other causes of CVI include central nervous system infection, traumatic brain injury, stroke, neonatal hypoglycemia, metabolic disease, hydrocephalus, seizures, congenital brain anomalies, maternal drug and alcohol use and chromosomal aberrations[17][19][20].

Meningitis (often caused by Hemophilus influenzae, Streptococcus meningitides, and Streptococcus pneumoniae) and encephalitis (Herpes simplex virus) are major causes of vision loss later in the course of infection. Visual pathway damage may be due to hypoxic-ischemic change, thrombophlebitis, arterial occlusions, venous sinus thrombosis or due to accompanying neurologic sequelae for example hydrocephalus and seizures[8].

Hydrocephalus may cause CVI due to damage to posterior visual pathways due to chronic distension of the posterior cortex[8].

Head trauma may cause cerebral visual impairment whereby damage may be transient or permanent. The damage may be primarily due to cortical injury or complications such as convulsions[8].

These causes of CVI were demonstrated by Njambi et al at KNH occupational therapy clinic which included neonatal sepsis/jaundice (92.5%), birth asphyxia (52.2%), meningitis (51.5%), congenital causes (44.4%) and prematurity (33.3%)[16].

Hypoxic-ischemic encephalopathy (47.2%), neonatal seizures (11.3%), hydrocephalus (4.9%), and neonatal hypoglycemia (4.2%) were the most common causes of CVI, according to Pehere et al at a tertiary eye hospital in South India. The majority had refractive error (50%), squint (48.2%), and ocular atrophy (82.4%). Furthermore, 72.5% of children with CVI had developmental

abnormalities, including motor delay (51.5%), cognitive delay (14.6%), and speech delay (2.9%)[15].

2.2 DIAGNOSIS

Poor vision in a child which cannot be explained by ocular examination findings should raise suspicion of CVI especially in the presence of preterm births, neurological deficits or a history of eventful perinatal period[9].

There is no globally approved diagnostic assessment method in place right now. Basic vision assessment, ophthalmological examination, standardized neuropsychological assessment, neuroimaging or any combination of these are used in the diagnostic process[6].

The ophthalmologist should perform a thorough vision assessment, eye examination, refraction in addition to identifying any medical history with a significant effect on vision. Associated abnormalities such as nystagmus, strabismus, refractive errors, anomalies of accommodation and optic atrophy should be sought out [21].

These children should be assessed for characteristic behavior which include delayed visual response (latency), color preference, attention to movement, complexity, visual field defects, absent visual novelty, absent reflex response, poor distant vision, light gazing and absent visually directed reach[22].

Suma et al retrospectively studied the presenting complaints of 88 children with neurological evidence of retro geniculate pathway pathology at an Indian tertiary eye institute. Majority of the patients were male (65.9%). Poor or no eye-to-eye contact (57.95%), squint (40%), poor hand-eye coordination (13.6%), and 'keeping eyes in a certain look' (10.1%) were found to be the most common complaints[23].

Visual assessment findings included fixing and following light (27.27%), normal visual acuity (17.04%) and non-specific response to light (14.77%). In addition, 17.04% had severe vision impairment or followed unilluminated bright objects, 12.5% had moderate vision impairment and 7.95% had severe vision impairment. Suma et al found simple myopia was more predominant than hyperopia in these 88 children. Examination findings included strabismus (67.04%), nystagmus (44.31%) and optic disc pallor in at least half the patients[23].

2.3 ASSOCIATED SYSTEMIC CO-MORBIDITIES

Development of normal vision occurs along with development of speech, motor and behavior in the first few years of life. As a result, poor vision can have an impact on these other functions and vice versa [(24)].

Common co-morbid conditions were impaired speech (29.9%), epilepsy (18.7%) and mental retardation (18.7%) as seen in a cross-sectional study by Njambi et al (2009) of children attending an occupational therapy clinic at a Kenyan national referral hospital, where majority had cerebral palsy (CP) (85.6%), attention deficit hyperactivity disorder (ADHD) (10.7%) and autism (9.6%)[(16)].

In a retrospective study conducted in Southern India, Pehere et al discovered delays in many developmental domains in 72.5% of children with CVI. Motor delay (51.5%), cognitive delay (14.6%), verbal delay (2.9%) and a combination of all three are among them (31.1%). The majority of these children had suffered severe injuries to several parts of the brain, resulting in profound vision impairment and delays in other areas of development [(17)(15)].

2.4 TREATMENT AND REHABILITATION

Early diagnosis of impairment and early intervention help affected children achieve a higher level of vision [(19)(25)].

While making an interventional plan, it is important to understand the underlying neurological issue, whether or not it is treatable, identifying the threshold of vision and how to work around it and finally vision intervention. The goal is to create an opportunity to acquire and interpret already available visual information.[(9)].

Treatment and rehabilitation must address the psychosocial impact on patients and their families hence counselling is important[(21)]. Each management plan is tailored to individual patients. Light reflex stimulation of each eye in a mostly dim and silent room is one kind of active visual stimulation therapy. Light is then shone on simple dark shapes such as circles and stars can be drawn on a bright cardboard and light shone on it to help develop an ability to identify object

outlines. All these are done several times a day. Eventually more details such as color are added to the shapes[7][8]. Overstimulation should be avoided in these children which would otherwise cause visual fatigue[26].

The age of onset, etiology and severity of impairment all have a role in vision improvement in these patients. Meningitis and epilepsy, for example, are linked to a worse prognosis [12]. There is limited data on the recommended duration of treatment. However, this may range from 7 – 13 months with improvement as much as age-appropriate reading[27].

Most children with CVI do not achieve normal visual acuity and will remain permanently impaired. Furthermore, children with CP in addition to CVI have a worse functional outcome than children who do not have CP. As a result, they need additional neurological and rehabilitative therapy[13][16].

Vision rehabilitation centers with various experts such as low vision therapists offer services that cannot be provided by the caregiver. They also offer an opportunity for the caregivers to interact with similar children and offer each other support[26]. The process involves identifying what the child can see and ensuring the child uses his/her vision most efficiently with minimal interference in activities of daily living[28].

3.0 STUDY JUSTIFICATION

CVI is emerging as an important source of preventable blindness in low-income societies due to poor perinatal care and high under-five morbidity rates. There is also an increase in survival of preterm neonates with various aetiological factors for CVI. These include birth asphyxia, neonatal sepsis, jaundice and meningitis[5][16].

Early identification of vision impairment, diagnosis and treatment improves the overall outcome of treatment with reduction in impairments that would otherwise contribute to developmental delays.

This study will help identify the lacking data on the current prevalence of CVI as an important cause of vision impairment, its pattern of presentation and associated ocular and systemic co-morbidities at KNH which have a profound impact on a child's overall development. The data may be used to develop a standardized diagnostic tool for patients in our set up.

4.0 STUDY OBJECTIVES

Broad objective:

To describe the prevalence and pattern of cerebral visual impairment in children attending Kenyatta National Hospital eye clinic.

Specific objectives:

1. To determine the prevalence of cerebral visual impairment in children attending Kenyatta National Hospital eye clinic.
2. To describe the pattern of cerebral visual impairment in children attending Kenyatta National hospital eye clinic.
3. To identify known associated systemic co-morbidities in children with cerebral visual impairment at Kenyatta National hospital eye clinic.

5.0 STUDY METHODS

5.1 STUDY DESIGN

This was a retrospective, hospital-based study.

5.2 STUDY AREA

The study was done at Kenyatta National Hospital (KNH) ophthalmology clinic 35. KNH is the main national referral and teaching hospital in Kenya. It is located in Nairobi, off Ngong road on Hospital Road.

KNH ophthalmology clinic has many sub-specialty clinics amongst them pediatric ophthalmology. Pediatric ophthalmology clinic is held on two days every week, Wednesday and Thursday with an average of 50 patients reviewed per week. The children followed up in these clinics include those referred from other KNH clinics, referrals from KNH wards, referrals from other hospitals and self-referrals. The Wednesday clinic is run by two pediatric ophthalmologists. The Thursday clinic is run by a pediatric ophthalmologist and two consultant ophthalmologists. Other members of staff include ophthalmic nurses and ophthalmic clinical officers. Additionally, a minimum of three postgraduate students work in each clinic under the supervision of the consultants.

5.3 STUDY POPULATION

All children attending KNH pediatric eye clinic from 1st January 2019 to 28th February 2021.

5.4 INCLUSION CRITERIA

1. Patients under the age of 16 years with a clear diagnosis of cerebral vision impairment.

5.5 EXCLUSION CRITERIA

1. Patients without outpatient files (those with cards only).

5.6 SAMPLE SIZE

Determined by the formula:

$$n = \frac{Z^2 p(1-p)}{d^2}$$

n- Minimum sample size

p- lowest prevalence of CVI in study population (p=2.4%)[13]

d- degree of precision 0.012

Z - z score 1.96 (95% confidence interval)

n = 624 patients

5.7 MATERIALS AND EQUIPMENT

Questionnaires

Patient files

Patient registry

Stickers

5.8 DATA COLLECTION PROCEDURE

Hospital records department does not use ICD 10 codes for outpatient clinic files, therefore, patient file number, age and sex was obtained from clinic records entered into a patient registry. The files were then picked from the hospital records department from which patients who meet the inclusion criteria were identified and their files marked with a sticker to prevent missing files and repeat entries. These marked files were collected and a questionnaire was used to obtain data that included:

- Demographic variables (age at the time of clinic visit, age at diagnosis and sex).
- Clinical variables (causes of CVI, non-ocular co-morbidities, ocular symptoms, visual acuity, ocular examination findings and refractive status of each eye).

5.9 DATA ANALYSIS

The data was entered into Microsoft Excel 2010 and double entry of data into Excel was done.

SPPS Version 24 was used to analyze the data which was classified into demographic and clinical variables.

Demographic variables included sex, current age and age at diagnosis of cerebral visual impairment.

Clinical variables included non- ocular systemic co-morbidities, ocular symptoms, examination findings which included visual acuity, ocular findings and refractive status of each eye.

Descriptive analysis of data was done whereby, measures of central tendency and dispersion were calculated and data presented in appropriate tables, graphs and charts.

Inferential analysis was used to determine relationships among variables using regression coefficients and draw conclusions from the study population. Confidence interval was taken as 95%. A p value of <0.05 was taken as statistically significant.

5.10 ETHICAL CONSIDERATIONS

5.10.1 Approval

Kenyatta National Hospital/University of Nairobi Ethics and Research Committee approval was requested and given (approval number P21/01/2022).

5.10.2 Confidentiality

There was no use of patient names nor initials. A unique code which matched the code on patient's file was assigned to each patient's questionnaire. Data was stored in one computer, encrypted and was accessible only to the principal investigator who complied with data protection standards.

5.10.3 Potential benefits and risks

This was a retrospective study; therefore, no procedures were done on patients. The results may be used to establish the pattern of cerebral visual impairment at KNH eye clinic hence provide information to develop a standardized diagnostic tool for patients.

The investigator had no conflict of interest.

5.11 QUALITY ASSURANCE

A pre-designed questionnaire was used as a data collection tool. Data was collected by the principal investigator to minimize misinterpretation of data, wrong data classification or leaving out some data from the files.

5.12 STUDY DISSEMINATION

The results will be presented within ophthalmology department and published in ophthalmology scientific journals. A copy of the book will be handed over to KNH research office.

6.0 RESULTS

A total of 674 children's files were retrieved at Kenyatta National Hospital Eye Clinic between 1st January 2019 and 28th February 2021, of which 53 children met the inclusion criteria for this study. The prevalence of CVI was 7.9%.

6.1 DEMOGRAPHIC DATA

A total of 53 children with cerebral visual impairment were seen at KNH Eye Clinic, out of which, 31 (58.5%) were male while 22 (41.5%) were female. The male to female ratio was 1.4:1. Out of the 53 children, 18 (34%) were below 12 months of age at presentation. The youngest patient at presentation was 2 months and the oldest was 10 years of age. The mean age at presentation was 25.9 months (SD = ±28.5, median = 16.5 months, IQR = 18). The mean age at diagnosis was 21.7 months (SD = ±28.2, median =11.00 months, IQR = 14).

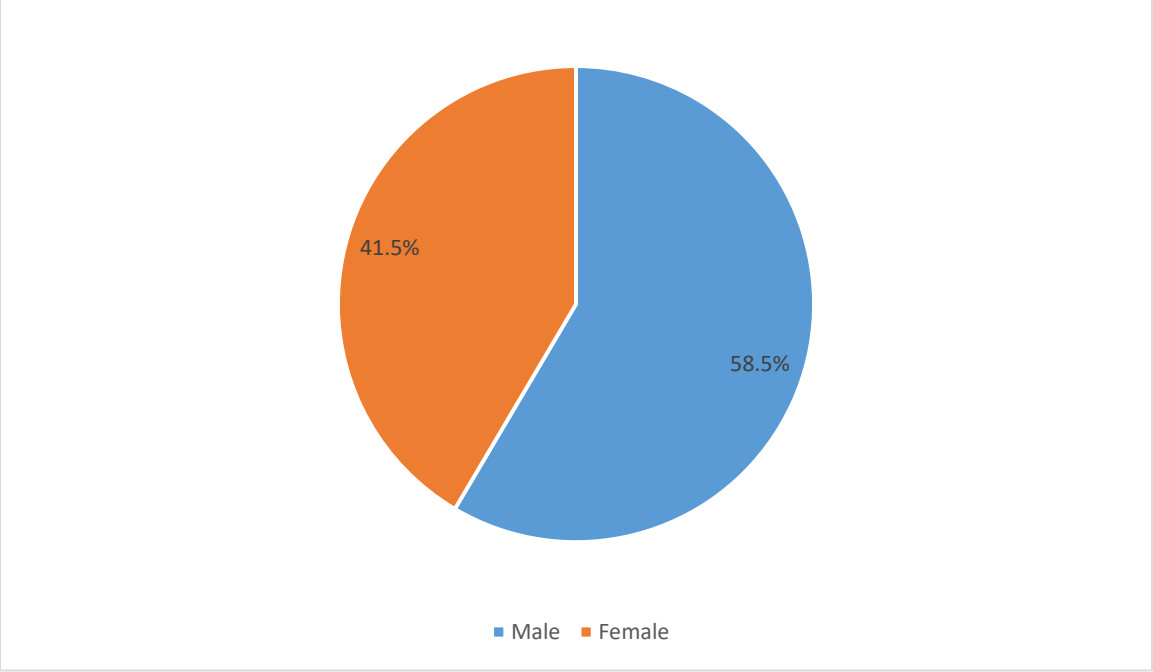

Figure 1: Distribution by sex of children with CVI (n=53)

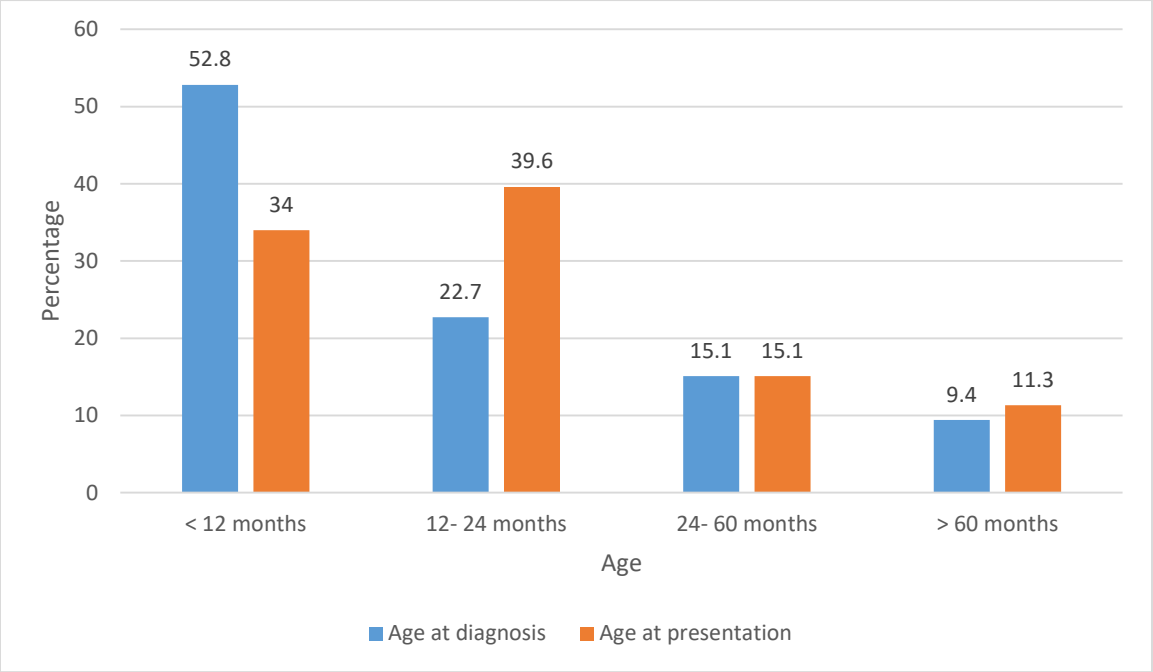

Figure 2: Distribution by age at diagnosis and age at presentation of children with CVI (n=53) (p=0.0001).

6.2 CAUSES OF CEREBRAL VISUAL IMPAIRMENT IN CHILDREN SEEN AT KNH EYE CLINIC

The most frequently reported causes of CVI among the 53 children were meningitis / encephalitis 24(41.4%), birth asphyxia 18 (31.0%) and hydrocephalus 13 (22.4%).

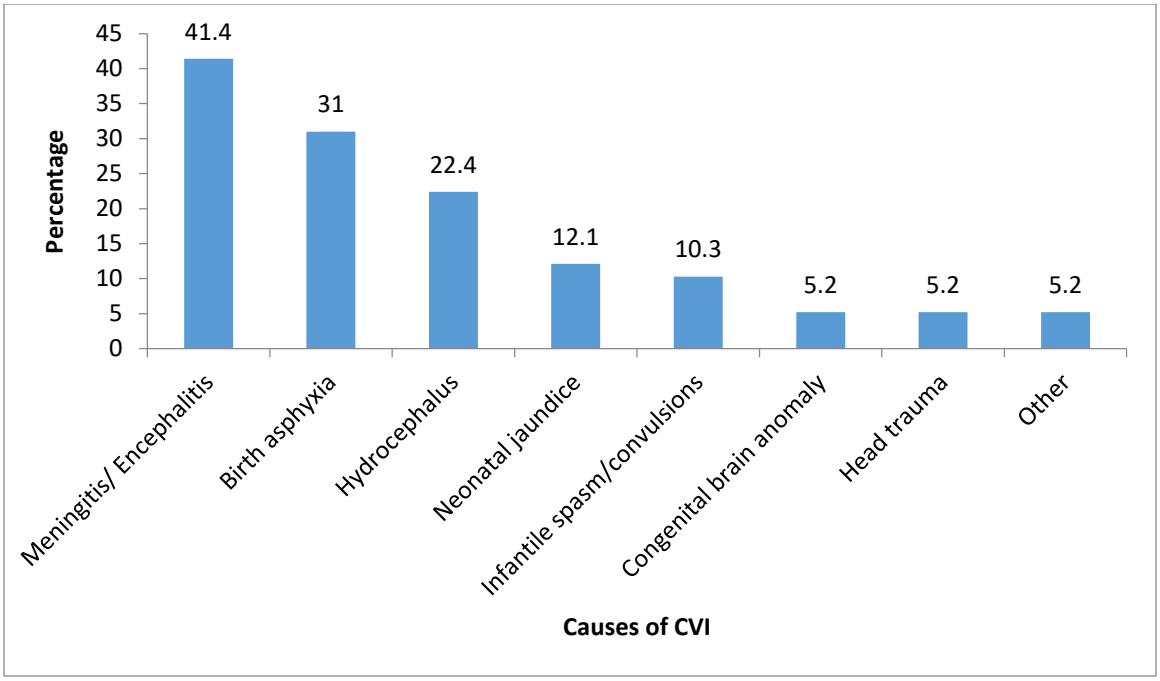

Figure 3: Causes of CVI among children seen at KNH eye clinic (n=53)

6.3 OCULAR SYMPTOMS IN CHILDREN WITH CVI

All children had poor vision, 6 (11.3%) squint and 4 (7.5%) nystagmus. Some children had more than one symptom such as poor vision and nystagmus or poor vision and squint.

Table 2: Distribution of ocular symptoms (n=53)

Symptom	Number	Percentage
Poor vision	53	100
Squint	6	11.3
Nystagmus (shaking eyes)	4	7.5
Photophobia	1	1.9
Others (specify) • Incomplete opening of right eye	1	1.9

*Some children had more than one ocular symptom.

6.4 OCULAR EXAMINATION FINDINGS

Table 3: Visual acuity at presentation (n=53)

Distance visual acuity	Without spectacles (n=53)
Not fixing and following light	29 (54.7%
Fixing and following light	14 (26.4%)
Fixing and following objects	4 (7.5%)
Blink reflex	2 (3.8%)
Picks 100/1000s at 33 cm	1 (1.9%)
Picks objects	1 (1.9%)
Snellen chart (6/60)	1 (1.9%)
Lea symbols (3/15)	1 (1.9%)

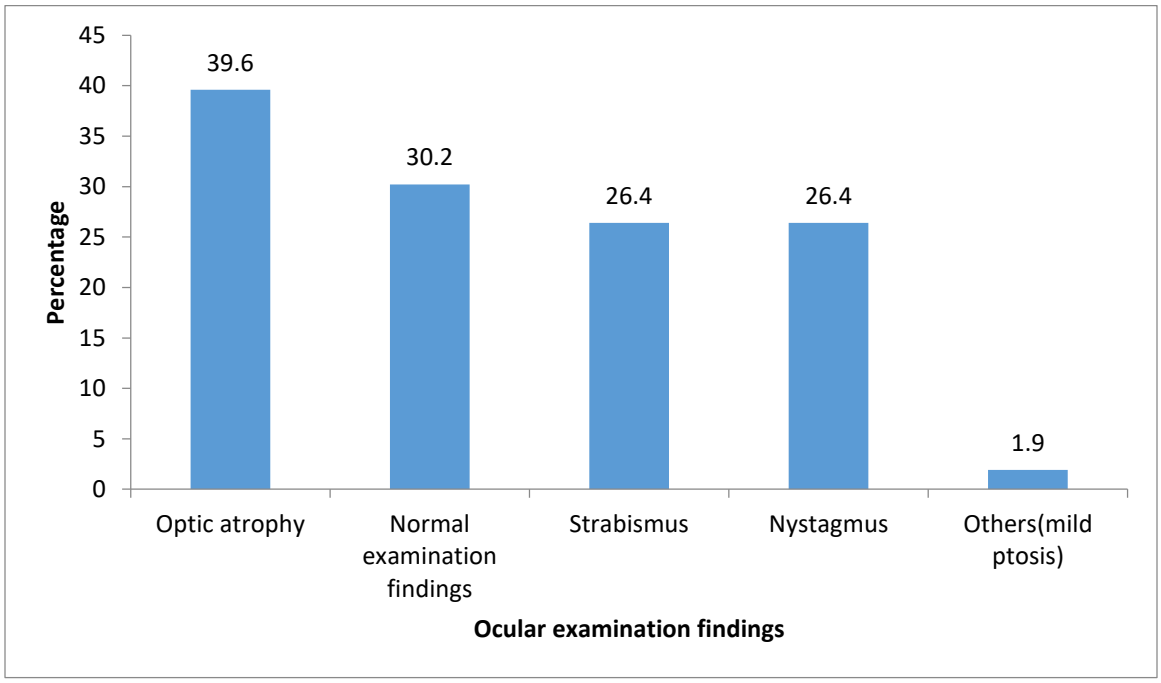

Figure 4: Ocular examination findings in children with CVI at KNH eye clinic (n=53)

Associated ocular examination findings were found in 37 (69.8%) children whereby optic atrophy was observed in 21 (39.6%) children. Normal ocular examination was found in 16 (30.2%) children. One patient had a mild right upper eyelid ptosis.

Table 4: Distribution of optic atrophy by age (n=21)

Age at presentation	Optic atrophy
<12 months	6 (28.5%)
12-24 months	9 (42.9%)
25-60 months	2 (9.5%)
>60 months	4 (19.1%)
Total	21 (100%)

Table 5: Distribution of strabismus and nystagmus by age (n=14)

Age at presentation	Strabismus	Nystagmus
<12 months	5 (35.7%)	4 (28.6%)
12-24 months	3 (21.5%)	6(42.8%)
25-60 months	5 (35.7%)	2 (14.3%)
>60 months	1 (7.1%)	2 (14.3%)
Total	14 (100%)	14 (100%)

Table 6: Refractive status of the children (n=24)

Type of Refractive error	Number	Percentage
Simple hyperopia	10	41.7
Mixed astigmatism	4	16.6
Simple myopia	2	8.3
Compound hyperopic astigmatism	2	3.0
Insignificant error	2	8.3
High myopia	1	4.2
Simple myopic astigmatism	1	4.2
Simple hyperopic astigmatism	1	4.2
Compound myopic astigmatism	1	4.2
Total	24	100

Out of the 53 children, 24 (45.3%) had their eyes refracted, while 29 (54.7%) did not. Among those who were refracted, 3 had spectacles which were retained. No spectacle correction was prescribed.

6.4 NON-OCULAR CO-MORBIDITIES IN CHILDREN WITH CVI

A total of 39 (73.6%) children had non-ocular co-morbidities and 14 (26.4%) had none. Of these, the commonest reported non-ocular co-morbidities were delayed milestones/ motor impairment 24 (61.5%), cerebral palsy 18 (46.2%), epilepsy 12 (30.8%), impaired speech 4(10.3%). Specific cases classified as others (10.2%) were left sided hemiplegia and rickets. Many children had more than one co-morbidity.

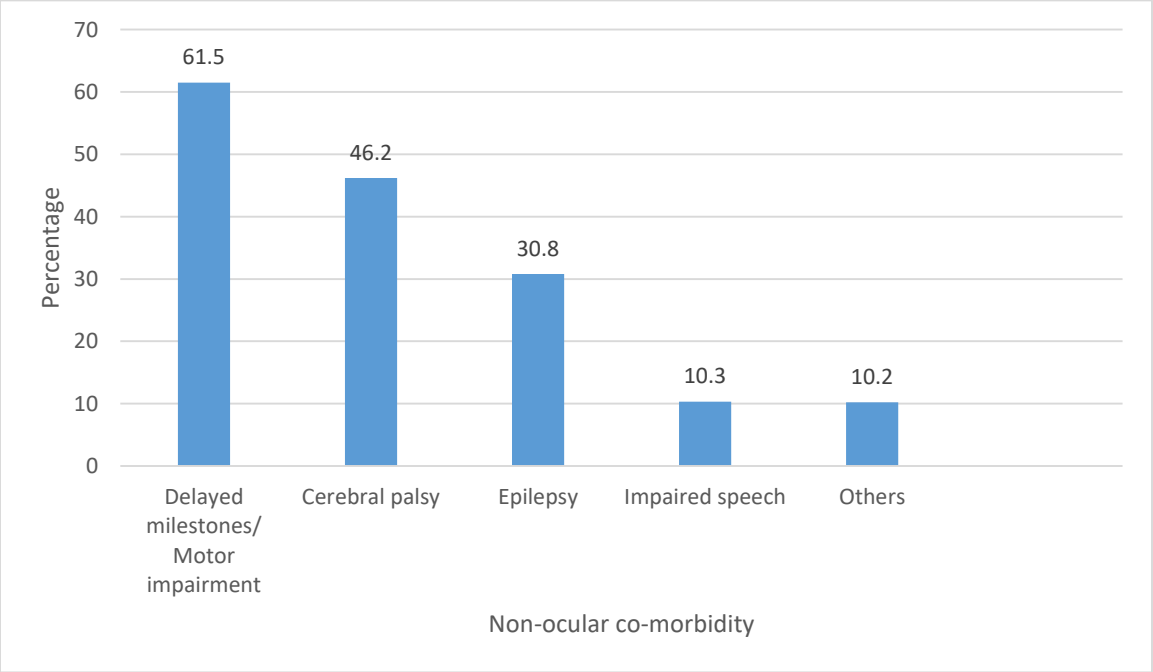

Figure 5: Distribution of non-ocular co-morbidities (n=39)

7.0 DISCUSSION

7.1 PREVALENCE

The prevalence of CVI among children presenting at KNH Eye Clinic was 7.9%. This was three times higher than 2.4% in a retrospective study done by Huo et al at a large pediatric ophthalmology practice over 15 years and published in 1999 where CVI was an important cause of vision impairment due to higher survival rates of neonates[13]. A much higher CVI prevalence of 33% was seen in a study by Pehere et al. However, this study was done in children in a vision impairment clinic where CVI was the leading cause[15]. In a cross-sectional study by Njambi et al, among children attending an occupational therapy clinic, CVI had a prevalence of 48.7% [16]. These study populations are not comparable to our CVI patients who were attending a general pediatric ophthalmology clinic.

There is no study done in our population that describes the prevalence of CVI. Therefore, an increase or decrease in prevalence cannot be accurately reported.

7.2 DEMOGRAPHIC DATA

CVI was seen more in male children than female children with a ratio of 1.4:1. This is comparable to a cross-sectional study by Smitha et al in a CVI clinic at a Southern Indian tertiary hospital, at 1.8:1[17]. A retrospective hospital-based study by Rojeeta et al found the male to female ratio was 3:2 [29]. A higher percentage of male patients (65.9%) with CVI were also seen in a retrospective study by Suma et al[23].

Majority of the children with CVI were above 12 months (60.4%). The youngest patient was 2 months and the oldest was 10 years of age (at presentation). The youngest patient was 1 month old when the diagnosis of CVI was made. The mean age at diagnosis was 21.7 months and mean age at presentation was 25.9 months (p=0.0001). This is comparable to a study by Njambi et al at a KNH occupational therapy clinic where the mean age of children seen was 2.3 years (27.6 months)[16]. Pehere et al found 29.03% of children with CVI were below 12 months[30]. The age when cerebral insult occurred in our set up was much earlier than when the diagnosis of CVI was made. Many children in the paediatric ward with causes of CVI such as meningitis were admitted with depressed level of alertness hence detecting poor vision was difficult. It was only noted when the child regained consciousness much later. The difference between the mean age of diagnosis

and presentation in our set up could be that a diagnosis was made while the child was admitted in the paediatric ward where the care giver or paediatrician noted poor vision in the child and requested for ophthalmology review who thereafter referred the child to KNH eye clinic.

7.3 CAUSES OF CVI

The commonest cause of CVI was meningitis/encephalitis (41.4%) followed by birth asphyxia (31%). Causes grouped as others included brain abscess, medulloblastoma and subdural hygroma (5.2%). Some patients had more than one causative factor for CVI. Common combinations included meningitis and hydrocephalus. Hydrocephalus occurred either as a primary disorder or as a sequelae of meningitis/encephalitis. Other combinations included birth asphyxia and infantile spasms/convulsions with or without neonatal jaundice. This differs from a study by Huo et al in a developed country where the commonest cause was perinatal asphyxia followed by cerebral vascular accident and meningitis/encephalitis. Hypoxic ischaemia was the commonest cause of CVI in the developed world[13][31].

In a South Indian tertiary facility, Sowmya et al found the commonest cause of CVI was perinatal asphyxia, epilepsy and structural MRI brain abnormalities. Majority of the patients had multiple aetiologies(32). Pehere et al, in their South Indian study found the commonest cause was hypoxic-ischaemic encephalopathy[15].

Causes of CVI found in a cross-sectional hospital based study by Njambi et al included neonatal sepsis/jaundice (92.5%), birth asphyxia (52.2%) and meningitis (51.5%)[16].

The difference between this study and others could be explained by the paediatric diagnosis given at the time of presentation with vision complaints. Whereby, meningitis/encephalitis and its sequeale is a common cause of childhood morbidity and mortality in our population.

7.4 OCULAR SYMPTOMS

The parents or guardians of all the children with CVI had complaints of poor vision. Some in addition to poor vision had squint (11.3%) and nystagmus (7.5%). Poor vision was described as

'not following faces', 'not following light' and 'inattentiveness'. One child was noted to have a drooping right upper eyelid with no systemic co-morbidity. This was a coincidental finding.

These findings are comparable to a retrospective study by Pehere et al where poor vision was the commonest complaint followed by squint[30]. Suma et al reported that the most common complaint was poor eye to eye contact which is a specific description of poor vision[23].

7.5 OCULAR EXAMINATION FINDINGS

Vision assessment in this study was not age appropriate for most children because of the multiple systemic deficits and advanced degree of vision impairment. About half of the children were not fixing or following light while less than a third were fixing and following light. Others were described as fixing and following light or objects slowly indicating a latency in visual response to stimulus which is a significant feature in CVI.

Visual assessment findings in a study by Suma et al included fixing and following light (27.27%), normal visual acuity (17.04%), severe vision impairment (17.04%) and non-specific response to light (14.77%)[23].

The commonest ocular examination finding was optic atrophy (39.6%) further categorized as temporal optic pallor (47.6%) and diffuse optic pallor (52.4%). Other findings included strabismus (26.4%) and nystagmus (26.4%). Of note, a third of the patients had normal ocular examination findings. One patient had a mild right upper eyelid ptosis and not significant to cause poor vision. Some patients had more than one ocular finding. Optic atrophy and nystagmus were mostly found in the 12-24 months age group. This age group was on follow up during the study period and injury happened much earlier than that.

The frequency of optic atrophy was less than that found by Suma et al which was 60.22% of the patients. Strabismus (67.04%) and nystagmus (44.31%) also had higher frequency [23]. Pehere et al also found a higher frequency of optic atrophy (82.4%). However, this was in children who all had severe visual impairment and therefore not comparable to our study [15]. Njambi et al found strabismus and refractive errors were common ocular findings among cerebral palsy patients. This is however not comparable to this study population[16].

Refraction was done in 24 children (45.3%). Simple hyperopia was the predominant refractive error (41.7%) followed by mixed astigmatism (16.6%). Of note is that only three patient records had indicated whether objective dry or cycloplegic refraction was done. One had cycloplegic refraction done with simple hyperopia and two with dry refraction with mixed stigmatism and compound myopic astigmatism. However, standard practice in KNH paediatric ophthalmology clinic involves dilated fundoscopy of all children with poor vision which is achieved using tropicamide and phenylephrine eye drops. Therefore, all children underwent wet non-cycloplegic refraction and only one had cycloplegic refraction. Three children had spectacles and retained their spectacle correction. The patients were 8 months, 3 years and 10 years with no visual acuity improvement with their spectacles. Majority had severe visual impairment, therefore vision stimulation therapy using light at home was prescribed.

Pehere et al found compound hyperopic astigmatism was the predominant refractive error in cycloplegic refraction[15]. Njambi et al found myopia was the predominant refractive error in non-cycloplegic refraction[16]. Simple myopic astigmatism was the predominant refractive error in the study by Suma et al[23]. No similarities were found in all the studies.

7.6 NON-OCULAR CO-MORBIDITIES

The most commonly reported non-ocular comorbidities were delayed milestones, cerebral palsy and epilepsy. Less than a third did not have any co-morbidity. Delayed milestones included inability to perform age appropriate tasks such as sustained neck support, inability to sit without support amongst others. All children with cerebral palsy had delayed milestones and only six had delayed milestones with no other co-morbidity. Some children had more than one diagnosed co-morbidity such as epilepsy and cerebral palsy.

Visual impairment frequently occurs more in children with various disabilities and can be combined with motor and cognitive disabilities[7][23]. This is similar to a study by Pehere et al. They however classified their co-morbidities into motor, cognitive and speech delays. Half of their patients had motor delays and 31.1% had delays in multiple areas of development. The association between CVI and delays in multiple areas of development is explained by the fact that these children suffer injury to multiple parts of the brain[15]. Suma et al found systemic associations in

85.2% of their patients whereby the commonest non-ocular co-morbidities were developmental delay and cerebral palsy[23].

8.0 STUDY LIMITATIONS

The main limitation was tracing of files in KNH records department. There was a record of all patients seen in KNH eye clinic in 2019. However, there were a lot of patient cards which did not meet the inclusion criteria. In addition, many of the file numbers were duplicated several times throughout the year, while many more files were missing. These challenges made it difficult to meet the required sample size required therefore necessitating to extend the study period to February 2021.

9.0 CONCLUSION

- All children with CVI presented with poor vision and a few had squint and nystagmus.
- The commonest known cause of CVI was meningitis/encephalitis followed by birth asphyxia.
- More than half of the children with CVI had profound vision impairment on examination.
- The commonest ocular examination finding was optic atrophy.
- Simple hyperopia was the commonest refractive error found.
- The commonest non-ocular co-morbidity was delayed milestones/motor impairment.

10.0 RECOMMENDATIONS

- All children with cerebral damage should be referred to an ophthalmologist for evaluation to avoid delayed diagnosis of CVI and so timely interventions can be done.
- Ophthalmologists should closely work with obstetricians and paediatricians to inform them of the causes of CVI so they can address causes such as birth asphyxia and early childhood illnesses which would help lower the incidence and prevalence of CVI.
- A study on visual outcome of CVI treatment in our set-up is recommended as follow-up to this study.

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APPENDICES

APPENDIX I: RESEARCH BUDGET

Item	**Quantity**		**Total cost (ksh)**
Proposal/Ethical approval			
Proposal printing (32 pages)	6 copies		3000
Binding of proposal	6 copies		600
Ethics proposal cost			2000
Internet			3000
		Subtotal	**8600**
Data Collection			
Printing of questionnaires	4 pages		40
Photocopying of questionnaires	4 pages (130 copies)		1040
Stationery			400
Box file for questionnaires	5		825
Flash disc (16gb)	1		1500
		Subtotal	**3805**
Transport		**Subtotal**	**1500**
Contracted services			
Statistician		**Subtotal**	**30000**
Dissemination			

Printing of final book (Approximately 50 pages)	8 copies		10000
Binding of finished book	8 copies		800
		Subtotal	**10800**
KNH research permission letter			1500
Dissemination costs			
		Total	**56,205**

APPENDIX II: QUESTIONNARE

A. BIODATA

Patient code: ……

Age ……..

Sex: Male ☐ Female ☐

Age at diagnosis: ……

B. CAUSES *(Tick all that apply)*

Hydrocephalus ☐ Congenital brain anomaly ☐ Meningitis/ Encephalitis ☐

Head trauma ☐ Birth asphyxia ☐ Neonatal jaundice ☐

Infantile spasm/convulsions ☐ Others ☐

C. NON-OCULAR CO-MORBIDITIES *(Tick all that apply)*

Cerebral palsy ☐ Impaired hearing ☐ Impaired speech ☐

Mental retardation ☐ Epilepsy ☐ Others (Specify) ☐

None ☐

D. OCULAR SYMPTOMS *(Tick all that apply)*

Nystagmus (shaking eyes) ☐ Photophobia ☐ Squint ☐

Poor vision ☐ Light gazing ☐ Others ☐

E. OCULAR EXAMINATION

Distance visual acuity	Without spectacles		With spectacles	
	Right eye	Left eye	Right eye	Left eye
Fixing and following light				
Not fixing and following light				
Picks 100/1000s at 33 cm				
Lea gratings test				
Lea symbols				
Can't be tested				
Not tested				

Ocular findings associated with CVI *(Tick all that apply)*

Strabismus ☐

Nystagmus ☐ Others ☐

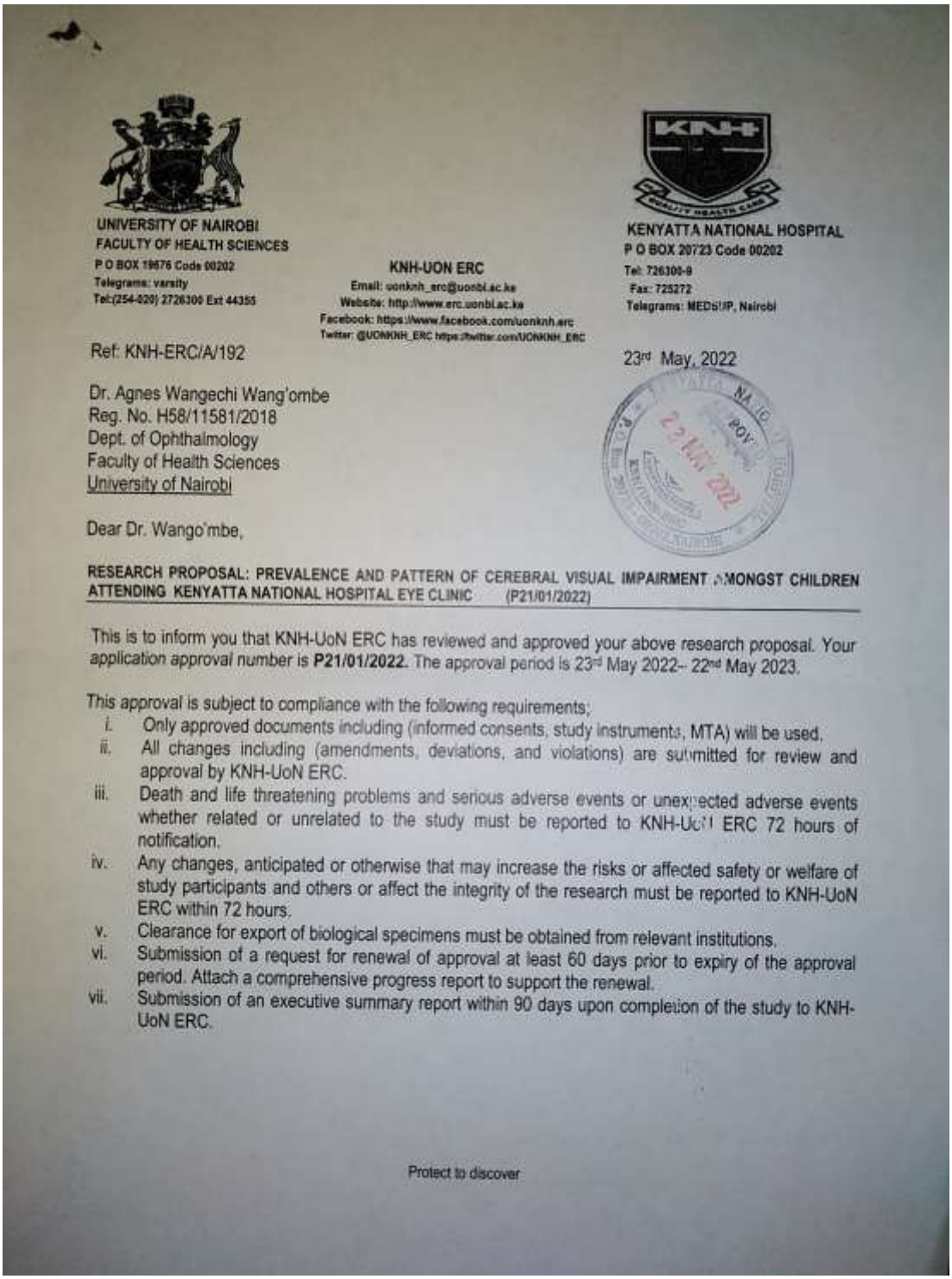
Optic atrophy ☐ None ☐

Has patient been refracted? Yes ☐ No ☐

If yes, refractive status of each eye:

	Refractive status	No spectacles given	Spectacles given(specify)
Right eye			
Left eye			

APPENDIX III: ETHICAL APPROVAL CERTIFICATE



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Ref: KNH-ERC/A/192

23rd May, 2022

Dr. Agnes Wangechi Wang'ombe
Reg. No. H58/11581/2018
Dept. of Ophthalmology
Faculty of Health Sciences
University of Nairobi

Dear Dr. Wango'mbe,

RESEARCH PROPOSAL: PREVALENCE AND PATTERN OF CEREBRAL VISUAL IMPAIRMENT AMONGST CHILDREN ATTENDING KENYATTA NATIONAL HOSPITAL EYE CLINIC (P21/01/2022)

This is to inform you that KNH-UoN ERC has reviewed and approved your above research proposal. Your application approval number is **P21/01/2022.** The approval period is 23rd May 2022– 22nd May 2023.

This approval is subject to compliance with the following requirements;

i. Only approved documents including (informed consents, study instruments, MTA) will be used.
ii. All changes including (amendments, deviations, and violations) are submitted for review and approval by KNH-UoN ERC.
iii. Death and life threatening problems and serious adverse events or unexpected adverse events whether related or unrelated to the study must be reported to KNH-UoN ERC 72 hours of notification.
iv. Any changes, anticipated or otherwise that may increase the risks or affected safety or welfare of study participants and others or affect the integrity of the research must be reported to KNH-UoN ERC within 72 hours.
v. Clearance for export of biological specimens must be obtained from relevant institutions.
vi. Submission of a request for renewal of approval at least 60 days prior to expiry of the approval period. Attach a comprehensive progress report to support the renewal.
vii. Submission of an executive summary report within 90 days upon completion of the study to KNH-UoN ERC.

Protect to discover

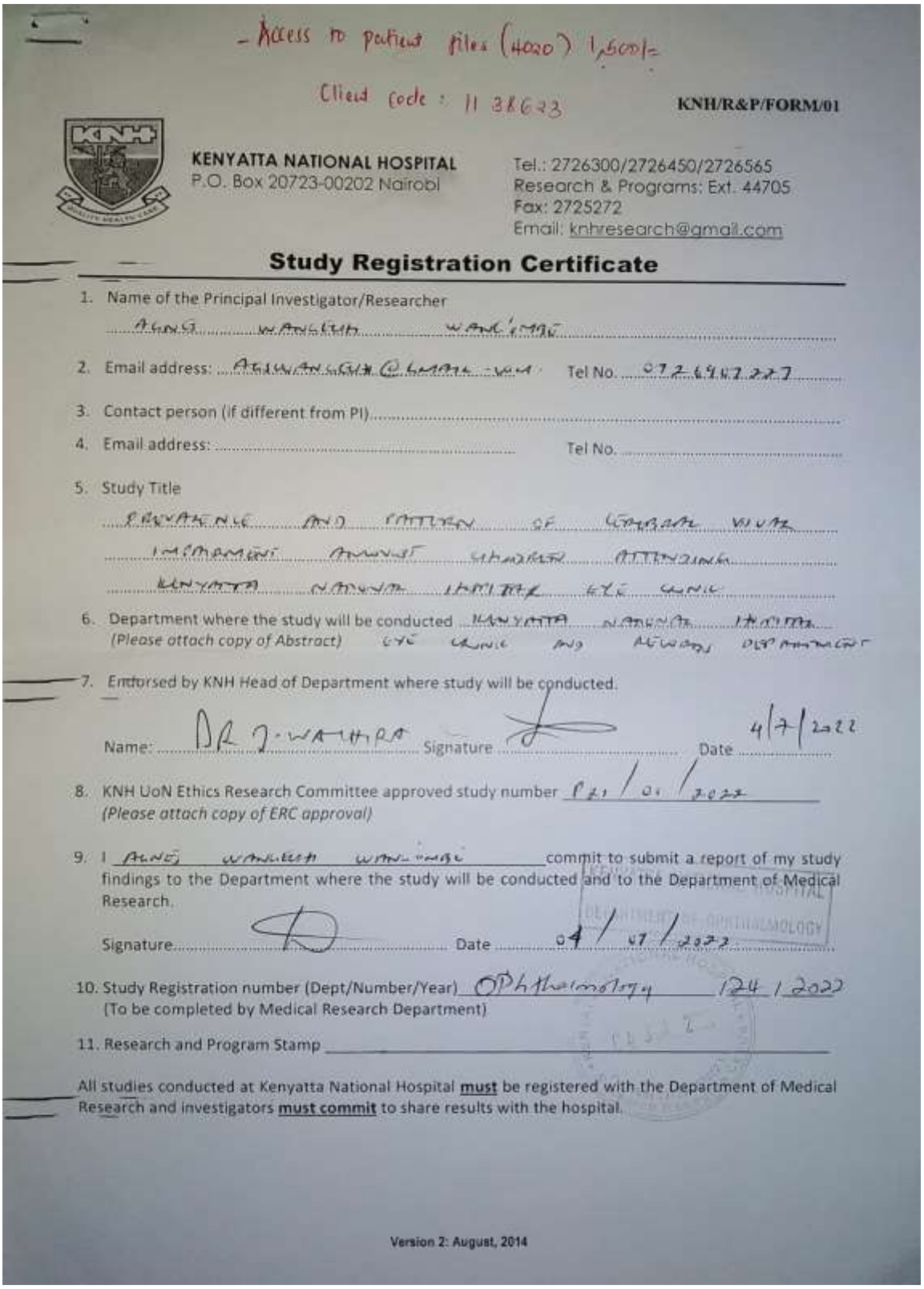
Prior to commencing your study, you will be expected to obtain a research license from National Commission for Science, Technology and Innovation (NACOSTI) https://research-portal.nacosti.go.ke and also obtain other clearances needed.

Yours sincerely,

DR. BEATRICE K.M. AMUGUNE
SECRETARY, KNH-UoN ERC

c.c. The Dean, Faculty of Health Sciences, UoN
The Senior Director, CS, KNH
The Chairperson, KNH- UoN ERC
The Assistant Director, Health Information, KNH
The Chair Dept. of Ophthalmology UoN
Supervisors: Dr. Lucy Njambi, Dept. of Ophthalmology, UoN
Dr. Margaret Njuguna, Dept. of Ophthalmology, UoN

APPENDIX III: STUDY REGISTRATION CERTIFICATE



- Access to patient files (4020) 1,500/=

Client code : 11 38623

KNH/R&P/FORM/01

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Research & Programs: Ext. 44705
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Study Registration Certificate

1. Name of the Principal Investigator/Researcher
   ALNES WANGECHI WANG'ONDU
2. Email address: AGIWANGECHI@GMAIL.COM Tel No. 0726947227
3. Contact person (if different from PI).....
4. Email address: ..... Tel No. .....
5. Study Title
   PREVALENCE AND PATTERN OF GLAUCOMA VISUAL IMPAIRMENT AMONGST SHAMIRAN ATTENDING KENYATTA NATIONAL HOSPITAL EYE CLINIC
6. Department where the study will be conducted KENYATTA NATIONAL HOSPITAL EYE CLINIC AND ACTIVE DEPARTMENT
   (Please attach copy of Abstract)
7. Endorsed by KNH Head of Department where study will be conducted.
   Name: DR J. WAMALA Signature ..... Date 4/7/2022
8. KNH UoN Ethics Research Committee approved study number P21/01/2022
   (Please attach copy of ERC approval)
9. I ALNES WANGECHI WANG'ONDU commit to submit a report of my study findings to the Department where the study will be conducted and to the Department of Medical Research.
   Signature ..... Date 04/07/2022
10. Study Registration number (Dept/Number/Year) Ophthalmology 124/2022
    (To be completed by Medical Research Department)
11. Research and Program Stamp .....

All studies conducted at Kenyatta National Hospital **must** be registered with the Department of Medical Research and investigators **must commit** to share results with the hospital.

Version 2: August, 2014