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# Clinical Profiles of Cerebral Visual Impairment in Suburban Area in Malaysia

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## Abstract

**Aim:** To describe visual potential, etiologies, and associated ocular and neurological deficits in children with Cerebral Visual Impairment (CVI) in suburban area in Malaysia, and to discuss the clinical profiles in relation to available published reports.

**Materials:** A retrospective study involving 31 children with CVI aged less than 17 years old was conducted at Hospital Universiti Sains Malaysia, Kelantan, Malaysia between January 2003 and December 2013. Demographic data and clinical profiles were reviewed.

**Results:** Both genders were affected equally. The majority of the children were categorized as blind at presentation. Five patients (16.1%) showed improvement of one to two lines on a Snellen visual acuity chart at one-year follow up, of which two had refractive errors and three had optic neuropathy. The most common etiology was perinatal asphyxia (58.1%). Optic disc atrophy (51.6%), global developmental delay (87.1%) and seizures (77.4%) were the most common ocular and neurological deficits observed.

**Conclusion:** The clinical profile of Malaysian children with CVI is fairly similar to published studies from the United States, Canada and New Zealand. Perinatal asphyxia was the most common etiology, but was two times higher than documented data from developed countries. This suggests a strong need for global improvement of the perinatal strategies in our policy. Rehabilitation programs are another issue that merits attention. More data from developing countries will be helpful for a better global approach.

**Keywords**: Cerebral Visual Impairment; Childhood Blindness; Developing Countries

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## Introduction

Cerebral Visual Impairment (CVI) is diagnosed by the exclusion or existence of many different etiologies and symptoms, which makes it non-categorized group overall [1- 3]. It is now recognized as a major etiology of childhood blindness in developed countries, ranging from 48-49% in the United Kingdom and 19% in the United States [4- 6]. In developing countries, childhood blindness due to CVI has been reported as 0.3% in India, 0.4% in China, 6.1% in Indonesia and 2.5% in Malaysia, respectively [7-10].

The current available data on clinical profiles of children with CVI are from the United States, Canada and New Zealand [2, 11-13]. Based on a PubMed literature search, we were unable to find any similar study from Asian countries or other developing nations. The objective of this study was to identify the common etiologies, associated ocular and neurological deficits, visual potential, and to discuss the

literature on CVI profiles from other developed countries. The outcomes of this study may provide new knowledge and possible strategies to identify and prevent treatable etiologies of CVI in developing countries.

## Materials

This study was conducted in a retrospective manner at Hospital Universiti Sains Malaysia, Kelantan, Malaysia. Records of 41 children with a diagnosis of CVI who were less than 17 years of age and attending the Ophthalmology Clinic from January 2003 until December 2013 were traced and reviewed. These children were monitored for at least 18 months to 5 years. Of 41 cases, 10 children were excluded due to missing or incomplete data. A total of 31 children with CVI were participated in our study.

Hospital Universiti Sains Malaysia is an academic-based institution located in the north east of peninsular Malaysia with pediatric ophthalmology and pediatric neurology services. It serves as a referral center in the east coast of peninsular Malaysia. This includes the states of Kelantan, Terengganu and Pahang, which make up a total estimated population of 3.8 million people, 90% of whom are of Malay ethnicity and covers an area of approximately 63846 $km^2$.

The clinical diagnosis of CVI was made on the basis that the visual dysfunctions were caused by damage to, or malfunctioning of the retro-schismatic visual pathways in the absence of damage to the anterior visual pathways or any major ocular disease. Children with associated ocular pathologies such as ptosis, corneal opacity, cataract, glaucoma, old optic neuritis, optic nerve infiltration, traumatic optic neuropathy, pigmentary retinopathy or a macular scar were excluded from this study.

Each record was reviewed for possible etiologies of CVI, age at presentation, visual acuities at presentation and one year after follow up, gestational age, presence of antenatal or postnatal complications, and other associated neurological deficits. Ocular findings including external, anterior and posterior segments signs, and funduscopic examination were documented. Imaging studies that were performed upon diagnosis, including computed tomography or magnetic resonance imaging of the brain/orbit were also reviewed.

Visual acuities of these children were divided into low vision and blind categories. Low vision was defined as corrected visual acuity of 3/60 (20/400) to 6/18 (20/60) in the better seeing eye, while blindness was defined as corrected visual acuity of worse than 3/60 (20/400) in the better seeing eye [14]. CVI registry forms were completed by a consultant pediatric ophthalmologist. Data were analyzed using the Statistical Package for Social Sciences (SPSS) Version 20.

## Results

We recruited 31 children who were diagnosed with CVI; 5 children had low vision, 21 children were blind and the remaining 5 children were uncooperative during the initial consultation. There was no gender predominance. The majority of them were born at term, while there were 4 children who were born between 25 weeks to 34 weeks. Perinatal asphyxia (58.1%) was the most common etiology of documented CVI, followed by brain maldevelopment (29.0%), meningitis (9.7%) and shaken baby syndrome (3.2%). None of our patients had a history of head trauma, maternal drug exposure or intrauterine infection. These data are summarized in Table 1.

**Table 1:** Demographics and etiologies of Malay children with CVI

| | Low vision n=5 (%) | Blind n=21 (%) | Unable to test n=5 (%) |
|---|---|---|---|
| **Sex** | | | |
| Male | 2 (40.0) | 10 (47.6) | 4 (75.0) |
| Female | 3 (60.0) | 11 (52.4) | 1 (25.0) |

| | | | |
|---|---|---|---|
| **Age (years)** | | | |
| 0-5 | 4 (80.0) | 21 (100.0) | 4 (75.0) |
| 6-10 | 1 (20.0) | 0 (0.0) | 1 (25.0) |
| 11-17 | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| **Ethnicity** | | | |
| Malay | 4 (80.0) | 19 (90.5) | 5 (100.0) |
| Chinese | 1 (20.0) | 2 (9.5) | 0 (0.0) |
| **Gestational age** | | | |
| Preterm | 2 (40.0) | 1 (4.8) | 1 (25.0) |
| Term | 3 (60.0) | 20 (95.2) | 4 (75.0) |
| **Etiology** | | | |
| Perinatal asphyxia | 0 (0.0) | 14 (66.7) | 4 (75.0) |
| Brain maldevelopment | 4 (80.0) | 5 (23.8) | 0 (0.0) |
| Shaken baby syndrome | 1 (20.0) | 0 (0.0) | 0 (0.0) |
| Meningitis | 0 (0.0) | 2 (9.5) | 1 (25.0) |

Table 2 describes the visual acuities at presentation and one year after follow up. 21 children (67.8%) had presenting visual acuity worse than 3/60 (20/400), while 5 children (16.1%) had visual acuity ranging from 6/60 (20/200) to 6/18 (20/60). Visual acuity was not documented in the remaining 5 children as mentioned above.

At the one-year follow-up, 7 children (22.6%) had their documented visual acuity ranging from 3/60 (20/400) to 6/18 (20/60). None of these children had visual acuity of 6/12 (20/40) or better. 5 children (16.1%) showed improved visual acuity at one year follow up. These included 2 children with associated myopia who had shown improvement of two lines, while another 3 children with optic neuropathy showed consistent improvement of one line on a Snellen visual acuity chart compared to their presenting visual acuity. 24 children (77.4%) displayed their visual acuity worse than 3/60 (20/400), and this included those 5 uncooperative children during the initial assessment.

We documented that 7 children (22.6%) remained no perception of light during both assessments. This included 6 children who had perinatal asphyxia and one child with history of meningitis. We were unable to do further analysis of association of visual acuity and identified etiologies of CVI due to a small sample size.

**Table 2:** Visual acuities at presentation and one year follow up

| Visual acuity (VA) | Presenting VA n=31 (%) | One year follow up n=31 (%) |
|---|---|---|
| 6/12 and better | 0 (0.0) | 0 (0.0) |
| 6/18 - 6/36 | 3 (9.7) | 4 (12.9) |
| 6/60 - 3/60 | 2 (6.5) | 3 (9.7) |
| 2/60 - 1/60 | 1 (3.2) | 3 (9.7) |
| Counting Finger | 0 (0.0) | 0 (0.0) |
| Hand Movement | 0 (0.0) | 0 (0.0) |
| Perception of Light | 13 (41.9) | 14 (45.1) |
| No Perception of Light | 7 (22.6) | 7 (22.6) |
| Unable to test | 5 (16.1) | 0 (0.0) |

The associated ocular and neurological deficits were presented in Table 3. Optic disc atrophy (51.6%) was the most observed ocular signs, followed by nystagmus (32.3%), esotropia (19.4%), exotropia (12.9%), myopia (19.4%) and hypermetropia (3.2%). We noted that global developmental

delay (87.1%) and seizure (77.4%) were the most documented neurological deficits. The others included spastic quadriplegia and diplegia (54.8%), hearing impairment (9.7%) and abducens nerve palsy (3.2%).

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**Table 3:** Associated ocular and neurological deficits

| | Low vision n=5 (%) | Blind n=21 (%) | Unable to test n=5 (%) |
|---|---|---|---|
| **Ocular deficits** | | | |
| Nystagmus* | 1 (20.0) | 9 (42.8) | 0 (0.0) |
| Esotropia | 2 (50.0) | 4 (19.0) | 0 (0.0) |
| Exotropia | 1 (20.0) | 3 (14.3) | 0 (0.0) |
| Myopia | 3 (60.0) | 3 (14.3) | 0 (0.0) |
| Hyperopia | 0 (0.0) | 3 (14.3) | 0 (0.0) |
| Peripheral retinal degeneration | 0 (0.0) | 1 (4.8) | 0 (0.0) |
| Optic disc atrophy* | 3 (60.0) | 10 (47.6) | 3 (75.0) |
| **Neurological deficits** | | | |
| Global developmental delay** | 3 (60.0) | 19 (90.5) | 5 (100.0) |
| Seizures** | 2 (40.0) | 17 (81.0) | 5 (100.0) |
| Spastic quadriplegia | 0 (0.0) | 12 (57.1) | 2 (50.0) |
| Spastic diplegia | 2 (40.0) | 1 (4.8) | 0 (0.0) |
| Hearing impairment | 0 (0.0) | 3 (14.3) | 0 (0.0) |
| Abducens nerve palsy | 1 (20.0) | 0 (0.0) | 0 (0.0) |

* 16 children had more than one ocular problems

** 27 children had more than one neurological deficits

## Discussion

The global economic burden due to childhood blindness, referring to lost capacity of earning has been estimated to be between 6,000 USD and 27,000 million USD in 1993 [15]. This amount is expected to be much higher in the 21st century. Thus, an economic burden is unavoidable in developing countries such as Malaysia. Early identification and prevention play a vital role in the strategy management.

Table 4 summarizes the clinical profiles and identified etiologies of CVI from the published literatures [2, 11-13], including our clinical findings. We observed that both genders were equally affected, and our data are parallel with other reports from the United States and Canada [11, 12]. On the contrary, Chong and Dai from New Zealand reported that males were predominant in their study [13]. Huo et al. did not mention gender distribution in their report [2].

We observed that 67.8% of our children were categorized as blind at presentation. Our observation is consistent with published studies from the United States and New Zealand; blind children constituted to 70.6% to 78.6% of their study populations [2, 12, 13]. However, Matsuba et al. reported a higher percentage was noted in the low vision group (62.1%) when compared to the blind group (33.8%) in their study, which was conducted in Canada [11].

Our data showed 5 children (16.1%) had improvement of one to two lines on a Snellen visual acuity chart at one year follow-up. Two children from the low vision group showed a

slightly better outcome, while 3 children from the blind group demonstrated a minimal change in their visual acuity. This is most likely due to the treatment they received and increased age of the children in our study. Though the improvement in visual acuity was not remarkable, we suggest that the treatment of associated ocular problems, especially refractive errors should be optimized in children with CVI.

Similar to our study, Huo et al. and Matsuba et al. reported improvement in visual acuity of 60.4% and 46%, respectively, in their follow-up assessments [2, 11]. This clinical observation is interesting and consistent with a report by Lantzy and Lantzy [16]. They suggested that improvement in the vision of children with CVI was associated with only a few factors that included increasing age, child's neurological history parents' motivation and access to pertinent information about CVI, systematic functional vision evaluation and interventions designed to match the assessed level of CVI [16].

Perinatal asphyxia was the most common identified etiology of CVI in our study, and this is in keeping with previously published studies that reported a range from 22.4%-36.0% [2, 11-13]. However, our incidence rate was higher compared to the available data from developed countries [2, 11-13]. This suggests that there is a clear need for significant improvement in the health care system to prevent or reduce the incidence of perinatal asphyxia that ultimately results in CVI. Obstetricians and neonatologists in developing countries should be alerted about their active role in preventing childhood blindness.

The other identified etiologies in our study were brain maldevelopment, meningitis and shaken baby syndrome which are fairly comparable with the available data from the United States, Canada and New Zealand [2, 11-13]. Four of our patients (12.9%) were born premature. Prematurity was also reported by the other published studies [2, 12, 13].

None of our patient had a history of head trauma, while a small percentage of children in the other published studies had documented head trauma [2, 11-13]. We believe that this is another preventable etiology that merits attention. Global developmental delay, seizure and optic disc atrophy are the most common neurological and ocular deficits encountered in our series. These data are also parallel with those published in studies from developed countries [2, 11-13].

**Table 4:** Comparison of clinical profiles of CVI in previously published studies

| Variables | Present Study n=31 (%) | Huo et al[2] n=170 (%) | Khetpal et al[12] n= 98 (%) | Matsuba et al[11] n=423 (%) | Chong and Dai[13] n=182 (%) |
|---|---|---|---|---|---|
| Country/year | Malaysia / 2014 | USA / 1999 | USA / 2007 | Canada / 2006 | New Zealand / 2014 |
| Sex | | | | | |
| Male | 16 (51.6) | NA | 56 (57.1) | 225 (53.2) | 116 (63.7) |
| Female | 15 (48.4) | | 42 (42.9) | 198 (46.8) | 66 (36.3) |
| Age at presentation, years | 0-17 | 0-15 | 0-19 | <3 , >3 | 3-16 |
| Presenting visual acuity | | | | | |
| Low vision | 5 (16.1) | 50 (29.4) | 26 (26.5) | 263 (62.1) | 39 (21.4) |
| Blind | 21 (67.8) | 120 (70.6) | 72 (73.5) | 143 (33.8) | 143 (78.6) |
| Unable to test | 5 (16.1) | 0 (0.0) | 0 (0.0) | 6 (1.4) | 0 (0.0) |

| | | | | | |
|---|---|---|---|---|---|
| Visual acuity at follow up | | | | | |
| Low vision | 7 (22.6) | NA | NA | 159 (61.2) | NA |
| Blind | 24 (77.4) | | | 62 (23.8) | |
| Unable to test | 0 (0.0) | | | 6 (2.3) | |
| Aetiology | | | | | |
| Perinatal asphyxia | 18 (58.1) | 38 (22.4) | 35 (36.0) | 151 (35.7) | 43 ( 24.0) |
| Brain maldevelopment | 9 (29.0) | 16 (9.4) | 30 (30.0) | 99 (11.8) | 5 (2.7) |
| Shaken baby syndrome | 1 (3.2) | 0 (0.0) | 7 (7.0) | 0 (0.0) | 13 (7.1) |
| Meningitis and infections | 3 (9.7) | 21 (12.4) | 15 (15.0) | 50 (11.1) | 12 (6.6) |
| Cerebral vascular accidents | 0 (0.0) | 24 (14.1) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Hereditary causes | 0 (0.0) | 0 (0.0) | 0 (0.0) | 42 (9.9) | 7 (3.8) |
| Brain tumour | 0 (0.0) | 4 (2.4) | 0 (0.0) | 0 (0.0) | 2 (1.1) |
| Head trauma | 0 (0.0) | 7 (4.1) | 4 (4.0) | 1 (0.2) | 12 (6.6) |
| Intracranial cyst | 0 (0.0) | 9 (5.3) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Congenital microcephaly | 0 (0.0) | 5 (2.9) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| In utero drug exposure | 0 (0.0) | 3 (1.8) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Acquired hypoxia | 0 (0.0) | 17 (10.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Idiopathic | 0 (0.0) | 16 (9.4) | 7 (7.0) | 0 (0.0) | 1 (0.5) |
| Seizures | 0 (0.0) | 7 (4.1) | 10 (10.0) | 6 (1.4) | 9 (4.9) |
| Metabolic | 0 (0.0) | 0 (0.0) | 1 (1.0) | 43 (10.2) | 2 (1.1) |
| Maternal drug use | 0 (0.0) | 0 (0.0) | 2 (2.0) | 0 (0.0) | 0 (0.0) |
| Static encephalopathy | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 1 (0.5) |
| Intrauterine infection | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 1 (0.5) |
| | | | | | |
| Neurological deficits | | | | | |
| Global developmental delay | 27 (87.1) | NA | NA | 368 (87.0) | 153 (84.0) |
| Seizures | 24 (77.4) | 90 (52.9) | 60 (61.0) | 279 (66.0) | 103 (56.6) |
| Cerebral palsy | 17 (54.8) | 44 (25.8) | 37 (37.0) | 313 (74.0) | NA |
| Hearing impairment | 3 (9.7) | 3 (1.8) | 11 (11.0) | 63 (14.9) | 11 (6.0) |
| Abducens nerve palsy | 1 (3.2) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Facial nerve palsy | 0 (0.0) | 3 (1.8) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Hemiparesis | 0 (0.0) | 20 (11.8) | 21 (21.0) | 0 (0.0) | NA |
| Microcephaly | 0 (0.0) | 26 (15.3) | 21 (21.0) | 0 (0.0) | NA |
| Behavioral problems | 0 (0.0) | 0 (0.0) | 4 (4.0) | 0 (0.0) | NA |
| Hypotonia | 0 (0.0) | 9 (5.3) | 0 (0.0) | 0 (0.0) | NA |
| Hydrocephalus | 0 (0.0) | 0 (0.0) | 0 (0.0) | 70 (16.5) | NA |

| | | | | | |
|---|---|---|---|---|---|
| Others | 0 (0.0) | 5 (2.9) | 0 (0.0) | 0 (0.0) | 13 (7.1) |
| Physical disability | NA | NA | NA | NA | 98 (53.8) |

| Visual acuity (VA) | Presenting VA n=31 (%) | One year follow up n=31 (%) |
|---|---|---|
| 6/12 and better | 0 (0.0) | 0 (0.0) |
| 6/18 - 6/36 | 3 (9.7) | 4 (12.9) |
| 6/60 - 3/60 | 2 (6.5) | 3 (9.7) |
| 2/60 - 1/60 | 1 (3.2) | 3 (9.7) |
| Counting Finger | 0 (0.0) | 0 (0.0) |
| Hand Movement | 0 (0.0) | 0 (0.0) |
| Perception of Light | 13 (41.9) | 14 (45.1) |
| No Perception of Light | 7 (22.6) | 7 (22.6) |
| Unable to test | 5 (16.1) | 0 (0.0) |

| | | | | | |
|---|---|---|---|---|---|
| Ocular deficits | | | | | |
| Nystagmus | 10 (32.3) | 19 (11.2) | 21 (21.0) | 72 (17.2) | NA |
| Esotropia | 6 (19.4) | 32 (18.8) | 19 (19.0) | 0 (0.0) | NA |
| Exotropia | 4 (12.9) | 31 (18.2) | 40 (41.0) | 0 (0.0) | NA |
| Refractive errors | 9 (29.0) | 14 ( 8.2) | 20 (20.0) | 0 (0.0) | NA |
| Retinal diseases | 1 (3.2) | 5 (2.9) | 4 (4.0) | 20 (4.7) | NA |
| Optic disc atrophy | 16 (51.6) | 28 (16.5) | 42 (42.0) | 56 (13.2) | NA |
| Ocular motor apraxia | 0 (0.0) | 26 (15.3) | 0 (0.0) | 88 (20.8) | NA |
| Amblyopia | 0 (0.0) | 0 (0.0) | 12 (12.0) | 0 (0.0) | NA |
| Optic nerve hypoplasia | 0 (0.0) | 0 (0.0) | 0 (0.0) | 18 (4.3) | NA |
| Ocular structural abnormalities | 0 (0.0) | 0 (0.0) | 0 (0.0) | 14 (3.3) | NA |
| Photophobia | 0 (0.0) | 0 (0.0) | 4 (4.0) | 0 (0.0) | NA |
| Other | 0 (0.0) | 2 (1.2) | 0 (0.0) | 0 (0.0) | NA |
| Neurological deficits | | | | | |
| Global developmental delay | 27 (87.1) | NA | NA | 368 (87.0) | 153 (84.0) |
| Seizures | 24 (77.4) | 90 (52.9) | 60 (61.0) | 279 (66.0) | 103 (56.6) |
| Cerebral palsy | 17 (54.8) | 44 (25.8) | 37 (37.0) | 313 (74.0) | NA |
| Hearing impairment | 3 (9.7) | 3 (1.8) | 11 (11.0) | 63 (14.9) | 11 (6.0) |
| Abducens nerve palsy | 1 (3.2) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Facial nerve palsy | 0 (0.0) | 3 (1.8) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Hemiparesis | 0 (0.0) | 20 (11.8) | 21 (21.0) | 0 (0.0) | NA |
| Microcephaly | 0 (0.0) | 26 (15.3) | 21 (21.0) | 0 (0.0) | NA |
| Behavioral problems | 0 (0.0) | 0 (0.0) | 4 (4.0) | 0 (0.0) | NA |
| Hypotonia | 0 (0.0) | 9 (5.3) | 0 (0.0) | 0 (0.0) | NA |
| Hydrocephalus | 0 (0.0) | 0 (0.0) | 0 (0.0) | 70 (16.5) | NA |
| Others | 0 (0.0) | 5 (2.9) | 0 (0.0) | 0 (0.0) | 13 (7.1) |
| Physical disability | NA | NA | NA | NA | 98 (53.8) |

*NA means not available

Our study has some limitations. The small sample size limits further analysis in our study. The association of visual acuity and identified etiologies of CVI will perhaps give more impact to the merit of analysis. This is a single center experience, while a nationwide study is preferable. A multicenter study will be ideal in a future research.

More data from other developing countries will address this shortfall in current knowledge. This is a real challenge for developing countries especially in South East Asian region. Various strategies should be planned and implemented in the health care system, especially improvement of rehabilitation program, availability of facilities and the well-being of this pediatric population.

In conclusion, the clinical profile of Malay children with CVI is fairly similar to published studies from the United States, Canada and New Zealand. Though perinatal asphyxia is the most common etiology, but we documented a tremendously higher incidence compared to developed countries. This suggests a clear need for significant improvement of perinatal management and treatment strategies. Rehabilitation program for children with CVI is another issue to address in developing countries.

## References

1. Boot FH, Pel JJ, van der Steen J, Evenhuis HM (2010). Cerebral visual impairment: Which perceptive visual dysfunctions can be expected in children with brain damage? A systematic review. Res Dev Disabil 31: 1149-1159.
2. Huo R, Burden SK, Hoyt CS, Good WV (1999). Chronic cortical visual impairment in children: etiology, prognosis, and associated neurological deficits. Br J Ophthalmol 83: 670-675.
3. Swaminathan M (2011). Cortical visual impairment in children - A new challenge for the future? Oman J Ophthalmol 4: 1-2.
4. Rahi JS (2007). Childhood blindness: a UK epidemiological perspective. Eye 21: 1249-1253.
5. Rogers M (1996). Visual impairment in Liverpool: prevalence and morbidity. Arch Dis Child 74: 299-303.
6. Steinkuller PG, Du L, Gilbert C, Foster A, Collins ML, et al (1999). Childhood blindness. J AAPOS 3: 26-32.
7. Titiyal JS, Pal N, Murthy GV, Gupta SK, Tandon R, et al (2003). Causes and temporal trends of blindness and severe visual impairment in children in schools for the blind in North India. Br J Ophthalmol 87: 941-945.
8. Hornby SJ, Xiao Y, Gilbert CE, Foster A, Wang X, et al (1999). Causes of childhood blindness in the People's Republic of China: results from 1131 blind school students in 18 provinces. Br J Ophthalmol 83: 929-932.
9. Sitorus R, Preising M, Lorenz B (2003). Causes of blindness at the Wiyata Guna school for the blind, Indonesia. Br J Ophthalmol 87: 1065-1068.
10. Patel DK, Tajunisah I, Gilbert C, Subrayan V (2011). Childhood blindness and severe visual impairment in Malaysia: a nationwide study. Eye 25: 436-442.
11. Matsuba CA, Jan JE (2006). Long term outcome of children with cortical visual impairment. Dev Med Child Neurol 48: 508-512.
12. Khetpal V, Donahue SP (2007). Cortical visual impairment: Etiology, associated findings, and prognosis in a tertiary care setting. J AAPOS 11: 235-239.
13. Chong C, Dai S (2014). Cross sectional study in childhood cerebral visual impairment in New Zealand. J AAPOS 18: 71-74.
14. World Health Organization. ICD-10 International statistical classification of diseases and related health problems. In: The epidemiology of eye disease. Johnson GJ, Minassian D, Weale R (eds). London: Champan and Hall Medical;1998:8-30.
15. Smith AF, Smith JG (1996). The economic burden of global blindness: a price too high! Br J Ophthalmol 80: 276-277.
16. Lantzy CAR, Lantzy A (2010). Outcomes and opportunities: a study of children with cortical visual impairment. J Vis Impair Blind 104: 649-653.