Etiology and health considerations of visual disability during early childhood: a review

Etiología y consideraciones en salud de la discapacidad visual en la primera infancia: revisión del tema

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Abstract

Visual impairment is a condition that occurs throughout life. Although the disability goes beyond the biological aspect, visual deficiencies in the range of low vision or blindness can impact development and visual function depending on age, having a broad etiology and several important health considerations for its prevention, diagnosis, treatment, and rehabilitation. This is a review of the etiological spectrum and health considerations of visual impairment during early childhood, based on key aspects of the neurodevelopmental aspects of vision, the etiological classification, and the most common causes of visual impairments capable of causing visual disability during early childhood. Retinopathy of prematurity, congenital cataracts, corneal opacities, congenital retinal diseases, and uncorrected refractive errors stand out as preventable causes or reversible visual impairment. In addition, vitamin A deficiency, trauma blindness, and child abuse are significant causes that should be assessed in children with visual impairment, etiologies that go beyond visual health. It is a priority to identify, in a timely manner, the preventable and treatable causes, and to recognize the inevitable ones to establish functional and comprehensive rehabilitation processes.

Keywords: Disabled children. Low vision. Blindness. Health of the disabled.

Resumen

La discapacidad visual es un tópico transversal al ciclo de vida. Si bien la discapacidad va más allá de lo biológico, las deficiencias visuales en rango de baja visión o ceguera pueden impactar el desarrollo y la función visual acorde a la edad, teniendo una amplia etiología y varias consideraciones en salud importantes para su prevención, diagnóstico, tratamiento y rehabilitación. Se realiza una revisión sobre el espectro etiológico y las consideraciones en salud de la discapacidad visual en la primera infancia, partiendo de aspectos clave del neurodesarrollo de la visión, la clasificación etiológica y las causas más frecuentes de las deficiencias visuales capaces de producir una discapacidad visual en la primera infancia. Como etiologías más frecuentes de deficiencias visuales permanentes en rango de baja visión (agudeza visual entre 20/60 a 20/400) y ceguera (agudeza visual menor de 20/400) sobresalen la retinopatía del prematuro, la catarata congénita, las opacidades corneales y las enfermedades congénitas de la retina, y como causas prevenibles o de discapacidad visual

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reversible destacan los errores de refracción no corregidos. Además, son causas importantes para evaluar en los niños con discapacidad visual el déficit de vitamina A, la ceguera por trauma y el maltrato infantil, etiologías que van más allá de la salud visual. Es prioridad identificar de manera oportuna las causas prevenibles y tratables, y reconocer las inevitables para instaurar procesos de rehabilitación funcional e integral, según sea el caso.

Palabras clave: Niños con discapacidad. Baja visión. Ceguera. Salud de la persona con discapacidad.

Introduction

Visual impairment, including low vision and blindness, is a topic of great interest in public healthcare and clinical assistance, and it is a condition that occurs throughout life including gestational life, the newly born, early, and middle childhood.

Visual impairment in children has an impact on their development causing disadvantages in terms of educational processes, independence, learning, and adaptation; in addition, at least half of the children with moderate to serious visual impairments (low vision), and blindness show concomitant motor or sensory impairment¹. It has been reported that this population shows higher rates of hospitalizations and mortality in all countries, whether they are high or low-income countries¹. Evaluating the functionality of vision in children with visual impairment is crucial to undertake both the functional and comprehensive adaptation, and rehabilitation processes^{2,3}.

The World Health Organization (WHO), in its report on low vision management in the pediatric population, groups visual impairments in three types depending on the best-corrected visual acuity in the best eye: moderate visual impairment, between 6/60 (20/200) and 6/18 (20/60); serious visual impairment, between 3/60 (20/400) and 6/60 (20/200); and blindness with visual acuity under 3/60 (20/400). Moderate and serious visual impairments are known as low vision. In addition, the WHO recommends that an impaired contrast sensitivity and no adaptation to darkness should be included as parameters of visual impairment if they cause significant functional compromise².

It is estimated that 1300 million people show some form of disability worldwide, of these, 1.4 million are children^{4,5}. Of the total number of blind people, at least, 4% corresponds to the pediatric population, and of the total number of people suffering from some type of visual impairment, 1% are children⁵. We should mention that almost half of the cases of blindness in children can be prevented⁵.

The main causes of blindness in children vary considerably from the geographical standpoint: in Scandinavian countries, Hungary, and the Czech Republic,

retinopathy of prematurity (ROP) is among the first five leading causes of severe loss of vision among school-age children^{4,6}. In Switzerland, it is reported that the five leading causes of blindness, in 76% of the population, are ROP, optic nerve hypoplasia, Leber's congenital amaurosis, optic nerve atrophy, and microphthalmia/anophthalmia⁷. In low- and medium-income countries congenital cataracts are prevalent, and in low-income countries, ROP prevails.⁸ In Latin America, the leading cause of blindness reported in children is ROP while the leading cause of low vision is uncorrected refractive errors⁴.

The objective of this study was to carry out a narrative review that would update the knowledge available on the etiology and health considerations of visual impairment during early childhood to show the broad etiological spectrum of this clinical, public health situation, and contribute to the understanding of health dynamics of children with visual deficiencies in the range of low vision or blindness.

A systematic search was conducted on the PubMed and Scielo search engines for articles published over the last 50 years on low vision and blindness during early childhood. The following keywords were used: "low vision", "blindness", "infants" and "child" (as well as their equivalents in Spanish). The results found were read and those that included information on the etiology and health considerations of visual impairment during infancy were selected.

Neurodevelopmental and vision milestones

Neurodevelopment is a complex, continuous, dynamic process that results from the child's interaction with the environment and parallel to the maturation of nervous structures, the acquisition of skills, and ultimately the formation of personality⁹.

The critical period of visual development occurs during the first 6 months of life and its maturation is completed at around 7 years of age^{10,11}. The development of the primary visual cortex (V1) begins on the 33rd gestational day, and it is completed when the baby

is born with the afferent pathway of the thalamus to the lamina four of the (primary visual) cortex V1¹². This occurs in several stages, the first one would be the formation of retinotopic maps on the cortex, which occurs by means of connections that develop between the axonal projections of the dorsal *pars* of the lateral geniculate nucleus with cells from the layer 4 of V1, a process that is guided by molecular signals on the cortex and spontaneous neuron activity. The second stage occurs after the first visual stimuli in the retina, causing greater orientation selectivity in V1 neurons. The next stage is known as a “critical period” because inadequate visual stimuli bring about consequences in the organization of the visual pathways that connect to the multipolar neurons of the brain cortex since it is in this stage that neuronal selective properties are perfected¹³.

Continuous development of visual acuity, movement perception, and contrast sensitivity is experienced during infancy, and visual developmental milestones are established as the motor, cognitive, and communicative development occurs (Table 1)¹⁴.

The first two years of life are more susceptible to altered binocular vision that causes amblyopia, which should be detected as early as possible to reach the greatest possible recovery of visual development¹². In children under 2 years of age, the V1 area experiences multiple synaptic connections, and different neural plasticity mechanisms are described to complete the developmental process¹².

The visual acuity test is essential in the child’s systematic evaluation throughout all the stages: newborn, infant, preverbal, and verbal child. Visual acuity should be tested as well as ocular alignment and the presence of structural ocular anomalies,¹⁵ in addition, it is recommended to look for signs that may be indicative of visual damage, such as lack of response to familiar faces or objects within the first few months of life, persistent nystagmus, erratic eye movements after three months of age, staring at bright lights, constant eye rubbing (oculodigital sign), and constant downward looking¹⁶. Table 1 details the main visual developmental milestones within the first year associated with motor and language progress.

Etiological classification of visual impairment in children

The classification of preventable and unpreventable causes of permanent visual deficiencies was conducted by the WHO including the moment when the condition first appears or the trigger factor (Table 2)^{18,19}.

Table 1. Visual developmental milestones and their association with motor and communicative development

Period	Milestone
First month	Visual curiosity, directed attention, following movement, patterns, and faces Brief visual contact and imitation of the adult’s facial expressions. Greater clarity at between 25 cm and 30 cm
After 3 months	Fixation is achieved: in this stage the child can already fix his eyes on an object and follow it. Maintained attention to faces and social smiles. Attention to the environment, following objects. Awareness of the use of hands to explore visualized objects. Interest in other people’s hands’ movement. Differentiation of white light colors improves.
After 6 months	Moves towards the observed objects that are out of reach. Understands some gestures and imitates actions observed such as greeting and hand clapping. Recognizes family members before they speak by their gestures and expressions. Individualizes objects by their shape and size. Integrates contours and edges. Shows fusion and stereopsis.
After 9 months	Understands communication expressions without dialogue. Knows where to look at to search for familiar situations or people. Perceives changes in height and surfaces as they move around.
After 12 months	Enjoys and recognizes familiar objects and people in photos

Adapted from de Swaminathan et al.¹⁷, Medina Alva et al.⁹ and Siu and Murphy¹².

The WHO proposes another classification following the course of the physical examination of the eye depending on the damaged anatomical region (Table 3)¹⁸. This classification is useful when, in clinical practice, there is not enough information on the child’s medical history¹⁸.

Some studies have proposed classifying the causes into preventable, treatable, and inevitable^{20,21}. Preventable causes are conditions that can be prevented through basic interventions such as promotion, prevention, and education. These include vitamin A deficiency, trauma, chorioretinitis, and toxoplasmosis²⁰. Treatable causes are conditions in which vision can be restored or preserved by medical or surgical procedures including ametropia, cataracts, aphakia, glaucoma, keratoconus, retinal detachment, uveitis, and ROP²⁰. Inevitable causes include congenital ocular anomalies, retinal dystrophies, optic neuropathies, cortical blindness, and microphthalmia²⁰.

Table 2. Etiological classification of low vision and childhood blindness

Hereditary condition	Chromosomal, mitochondrial, autosomal dominant, recessive autosomal disease linked with the X, unspecific; it includes albinism, macular dystrophy, retinitis pigmentosa, Stargardt's disease, and corneal dystrophy.
Intrauterine factor	Rubella, toxoplasmosis, drugs or alcohol, congenital cataracts, nystagmus, optic nerve hypoplasia, coloboma, microphthalmia, and aniridia.
Perinatal or neonatal Factor	Hypoxic-ischemic encephalopathy (cortical blindness), retinopathy of prematurity, neonatal conjunctivitis (<i>ophthalmia neonatorum</i>)
Postnatal factor in infants and children	Vitamin A deficiency, measles, neoplasms, eye traumas, myopic degeneration, glaucoma, retinal detachment, corneal opacity, refractive errors, amblyopia.

Adapted from Gilbert et al.¹⁸ and Uprety et al.¹⁹.**Table 3.** Etiology of visual impairment according to anatomical location

Eyeball	Phthisis bulbi, anophthalmia, microphthalmia, buphthalmos, enucleation, others
Cornea	Staphyloma, scarring, keratoconus, dystrophy, leucomas
Lens	Cataract, aphakia, other
Uvea	Aniridia, coloboma, uveitis, other
Retina	Dystrophy, albinism, retinopathy of prematurity, retinoblastoma, other
Optic nerve	Atrophy, hypoplasia, glaucoma, other
Normal-looking eyeball	Refractive error, amblyopia, cortical blindness, idiopathic nystagmus

Adapted from Gilbert et al.¹⁸.

Most common causes of visual impairment during early childhood.

Retinopathy of prematurity

ROP is characterized by impaired retinal vasculogenesis affecting its normal development. The clinical spectrum goes from spontaneous regression to total bilateral retinal detachment and blindness²².

It is the leading cause of childhood blindness in developing countries such as India and China, and in Latin American and Eastern European countries²³. The

main risk factors of this condition are premature birth, low weight at birth, supplementary use of oxygen, and neonatal sepsis related to immaturity during the histological, neuronal, and vascular development of the retina. These factors predispose the child to be more susceptible to certain aggressors, and trigger compensatory mechanisms that result in an aberrant vascularization^{22,24}. The growing rates of premature births, along with the greater survival of newborn babies thanks to the development of neonatal care units, although with diagnostic delays, have led to an increase of ROP blindness²².

ROP is classified clinically depending on the findings found in the fundus examination²⁵. A large number of premature patients develop some degree of ROP, but most cases of ROP are moderate and they resolve spontaneously; the remaining cases are severe and require treatment²⁶.

Back in 2008, Gilbert²⁷ estimated that over 50 000 children worldwide had gone blind due to ROP. However, recent papers suggest that this figure could have been underestimated because in 2010 almost 32 200 newborn babies suffered from visual impairment due to ROP²².

The clinical practice guidelines recommend conducting visual tests through indirect ophthalmoscopy in all newborn babies under 32 gestational weeks or in those who are born weighing less than 1500 grams²³. Primary prevention requires identifying the risk factors for ROP²⁸, and secondary prevention is based on identifying the disease early, and treating patients who need it in a timely manner. The gold standard regarding the management of this condition to prevent central vision loss in children with severe ROP is laser ablative therapy (laser photocoagulation)²⁹. Other management options are antibodies to block the vascular endothelial growth factor such as bevacizumab³⁰, and in more advanced cases, palliative surgical treatment²³.

Congenital cataracts

Any opacity of the lens of the eye occurring within the first year of life is defined as a congenital cataract³¹. It is the first preventable cause of blindness in children worldwide, and it was one of the priorities of the VISION 2020 initiative. It causes between 5% and 20% of all the cases of pediatric blindness, and nearly 200 000 children worldwide are blind due to cataracts. Some studies report that this cause of blindness among children is ten times higher in low-income countries due to

malnutrition, inadequate healthcare systems, and higher perinatal infection rates³².

Early diagnosis is important because a good intervention can yield good visual results³³.

In children, cataracts can be congenital or acquired; bilateral or unilateral³². In many cases, the etiology remains unknown, but approximately 50% is due to genetic mutations that encode proteins involved in the structure of the lens. Other important causes are maternal infections, genetic anomalies, metabolic disorders, systemic diseases³³, too many endogenous or exogenous corticosteroids, and child abuse. This condition, without timely treatment, can often cause visual deficit due to amblyopia and strabismus when it is unilateral, and blindness when it is bilateral³⁴.

Management is surgical management in most cases together with vision rehabilitation processes³⁴. Regarding prognosis, surgery performed within the first 2 months of life is associated with good results, being binocular cataracts less amblyogenic. The presence of nystagmus is a sign of poor prognosis³⁵. In spite of performing early surgery and intensive optical rehabilitation, children can develop amblyopia due to deprivation, nystagmus, strabismus, and glaucoma³³.

Corneal opacities

Corneal opacities are defined as a group of diseases characterized by loss of transparency in corneal tissue. Its etiology can be genetic, infectious, traumatic, toxic or a combination of them all, which alters the differentiation of the anterior segment between gestational weeks 6 and 16³⁶.

Most cases are bilateral and other eye malformations and concomitant systemic disorders are often reported³⁷. It is an uncommon disorder whose incidence rate is 2.2 to 3.11 per 100 000 newborn babies³⁷. Timely diagnosis and treatment are essential to reduce the risk of amblyopia or permanent visual impairment in bilateral cases³⁷, and it is important to detect other ocular anomalies or systemic compromise³⁶.

Another classification divides causes into primary and secondary. Primary causes are corneal opacities without a defined etiology, which are often present at birth, such as corneal dystrophy, corneal dermoid, peripheral sclerocornea, and CYP1B1 cytopathy. Secondary causes can be congenital or acquired; congenital ones are kerato-irido-lenticular dysgenesis (Peters anomaly type 1 and 2) and irido-trabecular dysgenesis. The acquired ones can be due to bacterial infections, herpes simplex virus, trauma or metabolic disorders³⁸.

Several studies report that the leading cause is Peters anomaly that often occurs with central opacity^{37,39}. It has been described as an anomaly in the development of the anterior segment that can involve Descemet's membrane, the stroma, and the endothelium. We should mention that 30% to 70% of the patients have concomitant glaucoma, which complicates visual prognosis⁴⁰.

Management will depend on the degree of compromise of corneal opacity. Surface keratectomy is indicated to eliminate surface corneal deposits, lamellar keratoplasty for deposits that are a little deeper, and penetrating keratoplasty for opacities at multiple levels⁴¹. In children, the corneal transplant does not have such positive results due to its higher rejection rate, the formation of new scarring tissue, and the onset of infection⁴².

We should mention that amblyopia, glaucoma, the compromise of other anterior segment structures, and intellectual disability can reduce the chances of vision rehabilitation⁴⁰.

Refractive defects

As the eye grows, its optical properties change to cause the emmetropization process or balance between the lens and refraction systems of the eye to focus images in the fovea^{10,11}. It is defined as ametropia when parallel rays of light don't focus in the fovea⁴³. The main etiology is hereditary, but malnutrition has been reported in the early stages of life as an important risk factor⁴⁴.

Uncorrected ametropia in children is highly prevalent in developing countries⁴⁵, and in general they are a cause of correctible or reversible visual impairment. It is considered a public health problem due to its negative impact on the child's learning and developmental processes⁴⁶.

It is classified as spherical or axial ametropia when patients' refraction defect depends on the length of the eye (myopia-hypermetropia), cylindrical ametropia when the refractive error is due to the curvature of the cornea and parallel rays of light cannot focus on the same point of the retina, and mixed ametropia when the patient has both types. The former is corrected using spherical lenses and the latter with cylindrical lenses; in addition, refraction surgery and rehabilitation can be used^{44,47}. Pediatric studies conducted in the United States, Chile, and Colombia suggest that almost half of the patients with low vision would improve with the right refractive correction procedure⁴⁸.

Uncorrected ametropia during childhood causes amblyopia, strabismus, poor academic performance, and visual impairment; therefore, detection and correction make a great positive impact and are among the most cost-effective measures in public health⁴⁹.

Brain and cortical disorders affecting vision

They are a spectrum of conditions due to neurological damage that can compromise optical radiations, the cortex or the association areas and trigger heterogeneous visual disorders that go from difficulties in visual processing to binocular blindness^{29,50}. Its prevalence is difficult to establish because there is not a unified international definition⁵¹. In addition, on several occasions global brain damage does not make it possible to differentiate whether it is the intellectual or motor deficiency the one that prevents the correct use of vision or whether there is a true neurological visual disorder⁵⁰.

A representative etiology is hypoxic-ischemic encephalopathy of the newborn baby, especially if they are severe disorders. Other causes are central nervous system malformations, trauma, epilepsy, and infections^{29,51}.

The classic signs of these patients are significantly diminished visual acuity or visual field disorder in the absence of anterior visual pathway damage with a history or evaluation suggestive of neurological compromise⁵⁰. Its determination is considered an exclusion diagnosis due to the etiological and clinical variety it shows; it is necessary to conduct an eye examination, a neuropsychological evaluation, and a brain imaging test^{50,52}. The current evidence available indicates that there is no correlation among etiology, location of the lesion, and visual perception; therefore, it should be defined mainly by the patient's visual disorders rather than his neuro-anatomical disorders^{50,52}.

Optic nerve hypoplasia

It is a congenital malformation that compromises the formation of one or both optic nerves with or without central nervous system malformations that affect the midline, and the hypothalamus- hypophyseal function⁵³. It is considered one of the leading causes of low vision and childhood blindness in developed countries such as Switzerland and the United States^{7,29,54}.

Its etiology is unknown in up to 70% of cases, but it can be associated with gestational factors (eg, teenage

pregnancy, nulliparity, first trimester bleeding, poor maternal and fetal weight gain) and gestational exposure to toxics (eg, alcohol, tobacco, and psychoactive drugs). The HESX1 genetic mutation is among the genetic factors, but it is only present in 1% of cases^{29,54,55}.

This condition has a worse visual prognosis compared to cortical visual deficiencies, ROP, and albinism⁵⁴. In 2013 it was reported that the annual incidence rate was 2.4 per 100 000 children under 19 years of age⁵⁴.

Diagnosis is clinical through direct ophthalmoscopy, in which it is possible to observe a small optic disc with augmented vascular formation. There is no consensus regarding the imaging assessment of the optic nerve in children, but the optical coherence tomography is useful^{29,54,55}. Due to the fact that this condition can be accompanied by other neurological disorders, comprehensive assessment includes brain magnetic resonance imaging scanning with especial emphasis on the sella turcica, where the main anatomical disorders seen are agenesis of the corpus callosum, absence of *septum pellucidum*, hypophyseal hypoplasia, and hydrocephalus^{54,56}. It is also recommended to request an early hormone profile test since the main associated endocrinopathy is growth hormone deficiency, in 82% of the patients who have some hormone deficit⁵⁶. It is also necessary to assess the need for correcting refractive defects, amblyopia, and strabismus that can be associated, and conduct a neuropsychological assessment to detect behavioral disorders and failure to thrive^{54,56}.

Congenital retinal diseases

This is a group of diseases associated with over 250 genetic mutations that lead to progressive photoreceptor damage, which is associated with visual loss, and blindness⁵⁷. The leading pathologies that occur during childhood are Leber's congenital amaurosis, retinitis pigmentosa, and achromatopsia^{29,57,58}. It is estimated that they affect 1/2000 people across the world, and are more common in developed countries since in these countries, the incidence of preventable causes decreases⁵⁷.

Retinitis pigmentosa is a recessive autosomal disease associated with the rhodopsin gene RHO and gene USH2A in 25% and 20% of cases, respectively. At first, it compromises the rods and cones and then it

degenerates the cones, which leads initially to nyctalopia followed by peripheral vision compromise, and eventually central vision impairment⁵⁸.

Dyschromatopsia is also a recessive autosomal disease considered a channelopathy that is diagnosed within the early few months of life, in which loss of vision and absence of color vision are associated with photophobia and pendular nystagmus⁵⁸.

Leber's congenital amaurosis is characterized by the severe loss of retinal cellular structures and, therefore, of visual function in the early stages of life. In Leber's type 2 congenital amaurosis cellular loss does not occur, but rather a mutation of gene RPE65 that is in charge of retinal isomerase for the regeneration of the chromophore that makes up photoreceptor pigments^{29,58}; in this type, to this date, genetic therapy has proven to be safer and more effective. However, it is necessary to conduct new studies because these therapies do not show lasting results due to posterior retinal degeneration^{29,58}.

Retinoblastoma

It is the malignant intraocular tumor that occurs most frequently during childhood; it represents 3% of childhood malignancies⁵⁹. It develops due to the malignant transformation of primitive retinal cells, and its main diagnostic age is before the child turns 3⁶⁰. In the United States and Europe, its incidence rate is approximately 11 new cases per million children under 5 years of age, which corresponds to 1 case per 15 000 to 20 000 live births. The worldwide incidence rate is 9000 new cases per year⁶¹.

This neoplasm can be familial or sporadic. Between 30% and 40% of the cases are inherited with a dominant autosomal pattern and are associated with mutation of the Rb1 oncosuppressor gene located in chromosome 13q14. That is why parents should be informed and the patient's siblings, if any, should be tested regularly^{59,60}.

The main clinical signs of these patients include leukocoria and strabismus, as well as chronic red eye, glaucoma, low vision, orbital cellulitis, and vitreous hemorrhage^{59,60}. Ophthalmoscopy is the initial diagnostic modality in addition to magnetic resonance imaging scanning to determine the extent of the disease⁶². In the advanced stages of the disease, enucleation is often preferred to improve survival by determining the need for adjuvants based on risk stratification⁵⁹. If disease is bilateral, an attempt is made to keep at least one of the eyes with the help of chemotherapy, cryotherapy or brachytherapy, and use of new technologies

such as selective infusion of intra-arterial chemotherapy, and intravitreal injections⁶².

The prognosis of this disease is good when the diagnosis is performed in a timely manner, but follow-up is advisable to detect the development of secondary tumors such as sarcomas, melanoma, and lung cancer or central nervous system cancer^{60,62}.

Vitamin A deficiency

Vitamin A is important to preserve the integrity of the eyeball, the vision process (especially in scotopic conditions), and to preserve the epithelial surfaces of the cornea⁶³. It is one of the leading causes of blindness in developing countries and it accounts for 70 deaths per 1000 live births⁶⁴. Around 190 million preschoolers have it and nearly 56% are cases located in Africa. In Latin America, it occurs mainly in Haiti, Bolivia, Peru, and Brazil⁶⁵, and are often associated with maternal and child malnutrition, diarrhea, malabsorption, and measles in children⁶⁶.

The main signs that can appear are exophthalmia, xerosis, Bitot's spots, keratomalacia, and predisposition to infectious keratitis⁶⁶. Supplementation and enriching some foods with this vitamin is essential to prevent up to 70% of blindness resulting due to its deficiency⁶⁴.

Trauma-induced blindness

It is one of the leading causes of unilateral non-congenital blindness, causing one-third of eye losses within the first 10 years of life⁶⁷. We should mention that traumas do not only happen in the context of the school or the household, but also that it is one of the consequences of war, violence, and armed conflicts involving children and adults, especially in Southeast Asia, Sub-Saharan Africa, and Afghanistan⁶⁸. Some authors point out that between 5% and 28% of the people who have been the victim of a terrorist attack present with eye traumas⁶⁹.

Eyeball trauma can be open or closed. Closed trauma is more frequent in boys compared to girls (2:1 and 4:1 ratio), caused mainly by toys, and its prognosis is better⁶⁷. Open trauma usually has a worse prognosis, above all if there is loss of vitreous humor, if the wound is > 10 mm, and if the patient can only recognize hand movements⁷⁰.

Toxoplasmosis, rubella, cytomegalovirus, and herpes (TORCH)

– Toxoplasmosis: infection due to *Toxoplasma gondii* by the intake of contaminated food and water. It is

generally associated with primary infection in the mother; its transmission and severity in the fetus depend on gestational age. Congenital infection causes chorioretinitis, hydrocephalus, intracerebral calcifications, epilepsy, cognitive deficiency or psychomotor retardation, although 90% of them are asymptomatic⁷¹. If the newborn baby shows criteria for congenital toxoplasmosis, he should receive treatment, since 82% of newborn babies who go untreated have retinal lesions when they reach adolescence, and 60% of those who are symptomatic at birth have visual loss⁷².

- Cytomegalovirus: it is the leading cause of non-genetic neurosensorial hearing loss and intellectual disability acquired during childhood. It affects 0.5% to 2% of live births, but its prevalence in pregnant women depends on their socioeconomic level since in 6% of them it is associated with poor levels compared to 2% (higher levels)⁷³. Its transmission is transplacental: primary infections, reinfections or reactivations. A total of 22% of children born with this condition have visual defects, and 10% to 15% have chorioretinitis since birth, which is indistinguishable from toxoplasmosis, but it can be differentiated by its slow progression^{73,74}.
- Rubella: immune preventable condition transmitted by droplets, contact, or transplacental infection. Congenital compromise is severe including congenital cataracts, neurosensorial hearing loss, hypoplasia of the iris, microphthalmia, and salt and pepper retinopathy. No specific treatment is available, hence the importance of vaccination⁷⁵.
- Herpes simplex: virus transmitted through lesions in the skin or mucosa through saliva, sperm, or vaginal secretion. A total of 1:3000 to 1:20 000 live newborn babies become infected (5% intrauterine, 85% perinatal, and 10% postnatal). Clinical signs include microphthalmia, chorioretinitis, eye atrophy, neurological symptoms, and 45% of cases show skin-eye-mouth infections on days 10-12 of life. Prevention is achieved through Caesarean section in pregnant women with active lesions⁷³.

Child abuse

It is defined as the set of signs and symptoms that appear in children who have been physically abused, bringing on permanent physical, mental, and emotional trauma⁷⁶. It can be suspected when eye injuries improve during hospitalization and get worse at home. If they are bilateral, they appear after birth and if damage predominates in the lower corneal segment since Bell's phenomenon protects the upper segment⁷⁷. Common

eye signs are wounds in the eyelids, posterior subcapsular cataracts, peripheral chorioretinal atrophy, and preretinal and retinal hemorrhages⁷⁶; in addition, the diagnosis of abuse should be considered whenever the child has a retinal disease, and optic atrophy⁷⁸.

Conclusions

The etiology of permanent visual deficiencies causing visual impairment in early childhood varies geographically. The clinical spectrum is broad, and it ranges from optical and ophthalmological conditions to neuro-ophthalmological and brain conditions.

The visual system and functions do part of the development; the first 6 months and 7 years of life are crucial for visual development purposes. There is an ongoing development of visual functions during early childhood.

The etiology of visual impairment during childhood can be classified depending on the moment when the condition appears for the first time or the anatomical region affected during the physical examination or else as preventable, treatable, and inevitable causes.

Congenital cataracts are the main preventable cause of childhood blindness. In developing countries, the leading treatable cause of childhood blindness is ROP. Uncorrected ametropias are the main group of preventable conditions causing low vision and reversible childhood blindness.

Clinically, in addition to ROP, congenital cataracts, and refractive defects, the following conditions should be considered as potential causes of visual impairment during early childhood: corneal opacities, brain disorders affecting vision, optic nerve hypoplasia, congenital retinal diseases, retinoblastoma, vitamin A deficiency, eye traumas, infections (TORCH), and child abuse.

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