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Visual acuity in children with congenital changes in the organ of vision

Mirosława Grałek, Anna Niwald

Department of Pediatric Ophthalmology, M. Konopnicka University Pediatrics Center, Lodz, Poland
Independent Public Healthcare Center – Central Teaching Hospital of the Medical University of Lodz, Poland

ABSTRACT

The paper presents the most common congenital disorders reducing visual acuity encountered in pediatric ophthalmology. Decrease in visual acuity caused by ptosis, lacrimal duct obstruction, sclerocornea, cataract, and other anomalies involving the lens, as well as congenital abnormalities in the optic nerve and retina, are discussed. The diagnostic work-up and treatment options for these pathologies are also presented.

KEY WORDS: visual acuity, child, congenital eye abnormalities, diagnostics, management.

INTRODUCTION

The process of vision is a complex sequence of transformations of a diverse nature. Under normal circumstances, vision starts to develop in the initial months of the child's life, and becomes consolidated and refined in the subsequent years. Neonates are born with an absolute light-response reflex. As the organ of vision matures – in parallel with the child's overall physical and mental development – binocular vision is established and improved. Good vision is determined not only by the functional state of the organ of vision, but also by other factors including the child's general development, psychological predisposition, and environmental influences [1]. The process of vision involves the eyes and extraocular anatomical structures associated with different parts of the organ of vision, i.e. the protective apparatus of the eye, the globe of the eye (anterior and posterior segments), the orbit, and the visual pathway with the cortical centers of the brain. Light rays entering the eye from the external environment pass through the pupil and the transparent optical media of the eye to converge on the retina. There, they undergo a range of physicochemical and biochemical transformations within appropriate layers of retinal cells and in the optic nerve. The next step involves converting the acquired energy into electrical discharges within the cerebral cortex and transforming them into nerve impulses. The transformation process set in motion by stimulation of the retina with light results in the formation of images of the physical world and their perception by the child. As a result, the physiological ability of both eyes to focus on a single point simultaneously (stereoscopy) is established and perfected to ensure independence and high quality of daily life, schooling, flexibility in educational and career choices, and healthy social interactions [2]. These transformations may be disrupted by congenital developmental abnormalities that can affect any part of this sensory organ, leading to a decrease in visual acuity or, in severe cases, complete loss of vision. This paper discusses selected congenital eye abnormalities with a known potential impact on vision, which are quite commonly encountered in pediatric clinical ophthalmology.

One of them is congenital ptosis [3]. The condition presents as either unilateral or bilateral drooping of the upper eyelid, causing narrowing of the eyelid opening and, consequently, partial or complete obstruction of the pupil by the eyelid. Even the narrowest eyelid opening preserves vision, though to a considerably reduced extent. Congenital ptosis is evident from birth or develops within the first year of life. The defect is relatively rare compared to other congenital ophthalmopathies, with the overall prevalence of congenital ptosis in the general population ranging from 0.18 to 1.41% [4]. Even though it is essentially a non-progressive condition, congenital ptosis in children can still lead to functional, esthetic, and psychosocial problems. Zeng *et al.* highlight the impact of the condition, particularly when it occurs unilaterally, on the development of the eye and the risk of secondary visual impairment (amblyopia) of organic nature [4]. Amblyopia disrupts the normal development of the visual pathway. If left untreated, it can persist into adulthood, causing vision disability that restricts career and job choices and can lead to challenges in other aspects of life. Amblyopia affects approximately 2 to 5% of the general population and represents

CORRESPONDING AUTHOR

Prof. Mirosława Grałek, Department of Pediatric Ophthalmology, M. Konopnicka University Pediatrics Center, Independent Public Healthcare Center – Central Teaching Hospital of the Medical University of Lodz, 36/50 Sporna St., 91-738 Lodz, Poland, e-mail: miroslawa.gralek@wp.pl

the leading cause of vision disability in children, which can be prevented by early diagnosis and initiation of appropriate treatment [5]. Delayed eyeball development secondary to ptosis is induced by an imbalance in the axial length-corneal radius (AL/CR) ratio. Zeng *et al*. found the AL/CR ratio to be significantly and positively correlated with the severity of ptosis [4]. Consequently, the AL/CR ratio may potentially serve as a useful indicator of ocular development, though the association between congenital ptosis and axial elongation of the eye has not as yet been definitively established. Some reports suggest that congenital ptosis has a minor impact on the elongation of the axial length of the eye [4, 6]. However, in severe cases of ptosis, marked anisometropia can occur, while the risk of amblyopia and strabismus increases already in the early stages of the child's development and may run a progressive course during this period. In individuals with congenital ptosis, the prevalence of amblyopia varies across the studies, ranging from 14.7 to 43.9%, with anisometropia affecting up to 24.4% of patients [4, 6]. The complications can be minimized or avoided by early surgical correction of ptosis. Surgical treatment methods vary depending on the patient's age, severity of the condition, function of the elevating muscle of upper eyelid, and surgeon's preferences. Postoperative management consists of visual rehabilitation required for secondary refractive errors, amblyopia, and strabismus [6].

Another cause of visual impairment, which is not always promptly detected, is congenital lacrimal duct obstruction, affecting 5 to 20% of children [7, 8]. Left untreated, it may lead to retention of tears and, frequently, mucous and/or purulent secretions on the corneal/ocular surface which can interfere with the normal development of the eye and predispose to amblyopia and refractive defects [7, 8]. During the initial weeks of life, which are the critical period for vision development, excessive meniscus and tear film along with inflammatory secretion on the cornea, change the optical characteristics of the anterior surface of the eye. Possible consequences include refractive errors and anisometropia, ultimately resulting in secondary amblyopia. In situations where both eyes are affected, there is a risk of binocular amblyopia. Early treatment, starting with conservative measures, and if these are ineffective, followed by surgical interventions (such as lacrimal duct probing, and surgical and endoscopic modalities like marsupialization of the lacrimal sac, nasolacrimal anastomosis, and periodic lacrimal duct intubation), helps mitigate the adverse effects of lacrimal duct obstruction on the child's visual development (Figure 1).

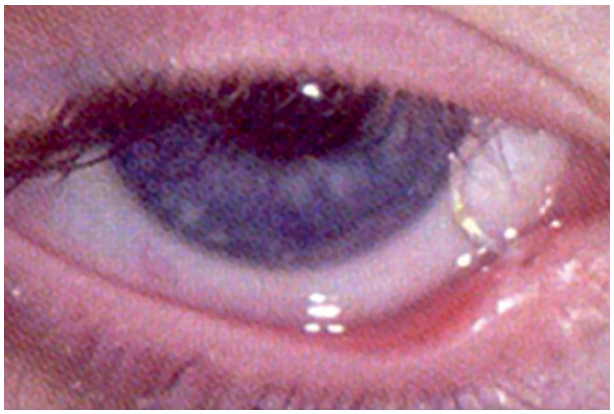

Figure 1. Tear duct probing with curved probe

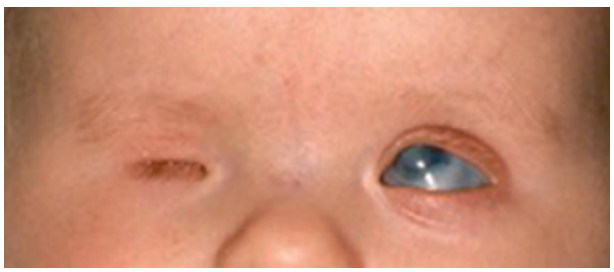

Figure 2. Left-sided corneal sclerotization. Right-sided anophthalmia

Sclerocornea is a congenital and primary developmental anomaly occurring in children, in which the cornea and other components of the anterior segment of the eye do not develop correctly. The condition leads to a significant reduction in visual acuity, potentially resulting in blindness (Figure 2) [9, 10].

Sclerocornea has a varying prevalence. Pantoja *et al.* [10] observed a relatively high prevalence of this anomaly – 1 in 397 people – in their study group consisting of subjects living in an isolated rural location. The value contrasts sharply with the overall incidence of corneal opacities (approximately 3 per 100,000 live births). This developmental anomaly is not progressive in nature. It is determined genetically and can be inherited in an autosomal dominant manner in milder cases or in an autosomal recessive pattern in more severe cases. Sclerocornea can manifest as either an isolated abnormality or as part of various syndromes. It is characterized by the absence of a clear demarcation between the cornea and the sclera, accompanied by varying degrees of corneal opacity. In patients affected by the anomaly, which is caused by bilateral ingrowth of vascularized opaque scleral tissue into the peripheral cornea, the sclera and cornea blend together, obliterating the boundary between the two structures. There is no distinct corneal limbus separating these distinct parts of the eye. Sclerocornea can be divided into two major types: total sclerocornea, which involves the tissues of the entire cornea, and peripheral sclerocornea, affecting the corneal periphery. In patients with total sclerocornea, the whole cornea is opaque and vascularized, while in the peripheral type, the affected area is vascularized, with regular arcades of superficial scleral vessels. The abnormality often occurs in association with cornea plana. In the majority of cases diagnosed in children lesions caused by sclerocornea result in complete corneal opacity and blindness, and the diagnosis is typically straightforward. Patients with peripheral sclerocornea may retain some useful vision, which can delay the accurate diagnosis of the condition. Sclerocornea must be primarily differentiated from congenital glaucoma, which can be another etiological factor for reduced visual acuity in children.

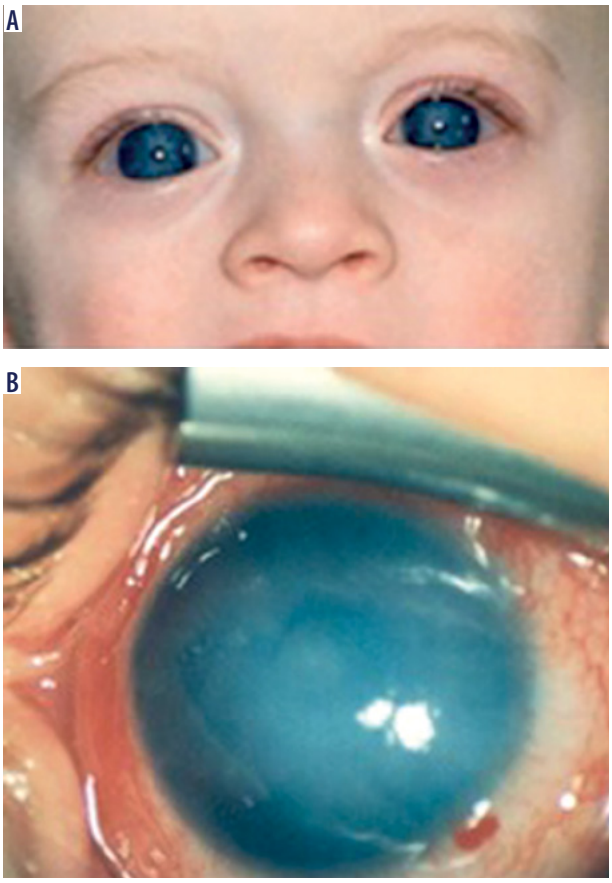

Figure 3. A) Congenital bilateral glaucoma, large eyeballs and large corneas – "pretty eyes". **B**) Congenital acute glaucoma, eye irritation, single hemorrhage in ocular conjunctiva, corneal edema

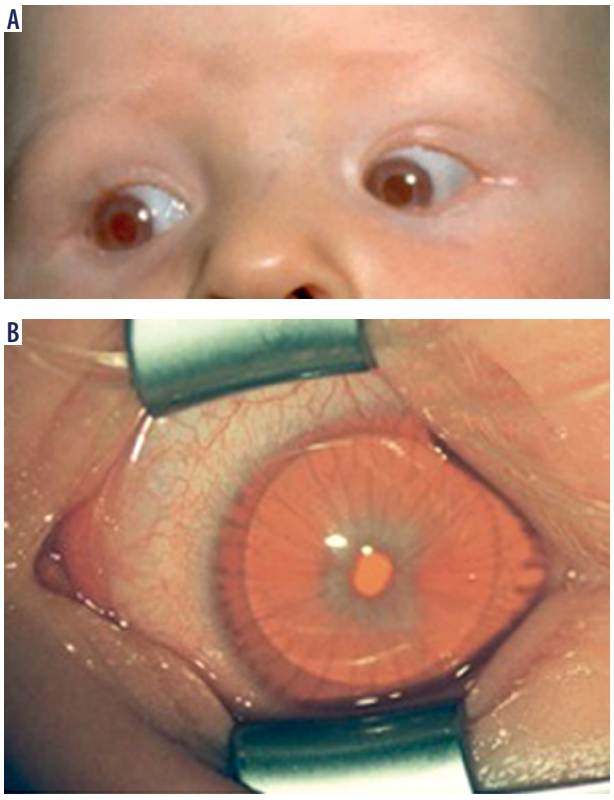

Figure 4. A) Albinism. **B**) Iris transillumination, absence of pigment in ocular structures

The primary treatment for sclerocornea is surgical, involving corneal transplantation. Pediatric keratoplasty is challenging on account of the technical intricacies of the procedure and high risk of graft rejection [11]. Successful management in children undergoing keratoplasty requires personalized clinical and surgical care along with extended postoperative rehabilitation [12].

Vitiligo (albinism) is another congenital anomaly that can have a significant impact on visual acuity and visual comfort in children (Figure 4).

Albinism has an estimated prevalence of 1 in 20,000 people [13]. Oculocutaneous and ocular albinism comprise a group of hereditary conditions which can be inherited in two patterns – autosomal recessive or autosomal dominant – and is linked to the X chromosome in boys. The conditions are characterized by a reduction or absence of melanin in ocular tissues and other pigmented structures in the skin and/or hair. The severity of symptoms can vary depending on the inheritance pattern. Ocular changes are a common and characteristic feature of albinism in all affected individuals, and include photophobia, nystagmus, iris transillumination, hypopigmentation of the fundus, underdevelopment of the central retina with foveal hypoplasia, and conduction abnormalities in the optic nerve [14]. In children with albinism, these manifestations result in diminished visual acuity, sometimes to a considerable degree. Features of cutaneous albinism may or may not be present. The condition is typically treated symptomatically, with cosmetic and colored contact lenses used for the relief of symptoms. Cataract is another cause of reduced visual acuity and, at the same time, one of the main causes of preventable childhood blindness. A decrease in visual acuity due to cataract, even in patients with severe lens opacification, never leads to the loss of light perception in the eye. The absence of light perception is indicative of damage to the retina, optic nerve, or brain cortex. Cataract affects approximately 200,000 children worldwide, with an estimated incidence of 3-6 cases per 10,000 live births [15]. The pathogenesis of the condition is multifactorial, and not yet fully understood. Congenital cataract is idiopathic in 60% of cases, but it may also be genetically determined. It is most commonly inherited in an autosomal dominant pattern, with a familial history of the disorder. Other factors contributing to cataract formation include ocular developmental anomalies, metabolic diseases, and intrauterine infections, particularly those caused by TORCH pathogens. Pediatric cataract may be congenital if present within the first year of life, or developmental if present after infancy. Lenticular opacity may also coexist with abnormalities in the shape of the lens, such as lens defect or dislocation (Figure 5).

Developmental cataract may progress in the later years of the child's life. In addition to the congenital and developmental types, children can also be diagnosed with acquired cataract, developing as a complication of diverse eye conditions

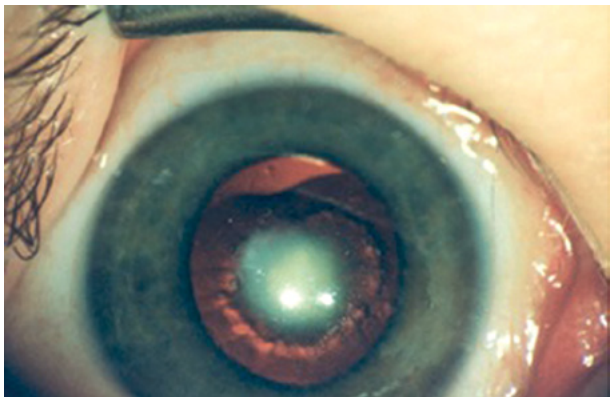

Figure 5. Partial lens opacity, coloboma lentis

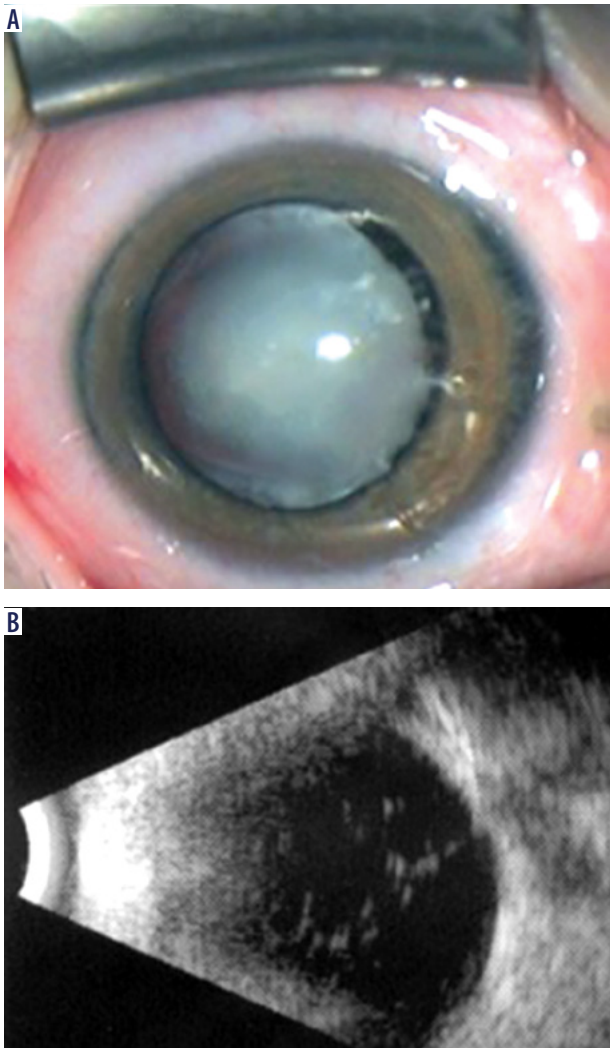

Figure 6. A) Persistent fetal vasculature (PFV), solid retrolental vascularized membrane. **B)** PFV on ocular ultrasound, echo signal of persistent fetal artery

or associated with underlying systemic pathologies. Cataracts in children exhibit a broad range of morphological variability, based on which they are classified as nuclear, cortical or subcapsular. Opacification may involve the complete lens or be limited to the central or peripheral parts. It can present in various forms including wedge cataract, punctate cataract, crystalline cataract, coralliform cataract, running along the sutures of the lens or in any other location and extent. Early diagnosis and treatment are crucial to prevent irreversible amblyopia which occurs in children due to a lack of visual stimulation in the cataract-affected eye. This is known as amblyopia ex anopsia, and the lack of visual stimuli can lead to visual deprivation syndrome, which affects children above 16 weeks of age. A central cataract larger than 3 mm in diameter, unilateral cataract associated with strabismus, and bilateral cataract with nystagmus are regarded as visually significant. The primary treatment involves surgical removal of the opaque lens, followed by immediate and consistent visual rehabilitation [16, 17]. Bilateral cataract requires surgery within the first few weeks of life to prevent visual deprivation, with a short interval (typically one to two weeks) between procedures to remove the cloudy lens to avoid amblyopia in the eye operated on later.

Congenital cataract may occur in association with a congenital developmental disorder termed persistent fetal vasculature (PFV), formerly known as persistent hyperplastic primary vitreous (PHPV) (Figure 6) [17].

In certain cases, the symptoms of PFV may be suggestive of cataract. The clinical presentation of the condition can vary depending on the location and manifestation of persistent vascular abnormalities. PFV is classified into three types: anterior, posterior, or a combination of anterior and posterior, which is most commonly diagnosed. Characteristic features of anterior PFV include a shallow anterior chamber, dilated iris vessels, persistent fibrovascular membrane of the lens, cataract, and/or pathological involvement of the ciliary processes. The formation of membranous retrolental structures and involvement of deformed (drawn-in or elongated) ciliary processes may result in tractions contributing to a decrease in aqueous humor production and potentially leading to ocular hypotony. Disc-like membranous cataract develops as a result of opacity arising from spontaneous resorption of the contents of the natural lens. Posterior PFV is characterized by falciform retinal septum formed by a retinal fold extending from the optic disc to peripheral retina or the retrolental area (feature typical of this form of PFV). Other manifestations include vitreous traction, epiretinal membrane, and optic nerve hypoplasia. There is no lens opacification. Anteroposterior PFV has the clinical characteristics of both anterior and posterior PFV, such as punctate opacities of the posterior lens capsule, solid retrolental vascularized membrane, retinal pigment changes, retinal detachment, hypoplasia or dysplasia of the optic nerve, leukocoria, and microphthalmia. Microphthalmia is a common finding in almost every type of PFV, occurring in approximately 85% of cases. The presence of microphthalmia is correlated with poor visual acuity even with appropriate therapeutic management [18, 19]. PFV may coexist with a malformation of the optic disc referred to as morning glory disc anomaly, which can also result in reduced final visual acuity (Figure 7). The deformity was first described by Kindler in 1970 [16, 17].

Among the various diagnostic methods, ocular ultrasound with vascular flow assessment and magnetic resonance

imaging are helpful in the diagnosis of PFV. Persistent fetal vasculature and cataract need to be considered in the preoperative diagnostic work-up and surgical management [20]. In some cases, the presence of PFV is not evident until the time of cataract surgery, when it requires additional intraoperative measures. The resulting aphakia is routinely corrected surgically – from the age of around two – through the implantation of an artificial intraocular lens immediately after cataract removal. In infants and young children, refractive errors, such as hyperopia, are corrected with contact lenses or eyeglasses with appropriate optical lens power. In some cases, the option of implanting an artificial intraocular lens may be considered at a later stage. In unilateral aphakia, eyeglasses are prescribed to manage amblyopia in the affected eye. Recent advances in surgical techniques and optical rehabilitation methods have led to significant improvements in both the anatomical and functional outcomes of pediatric cataract surgery [20]. Posterior capsule opacification and secondary glaucoma remain the primary postoperative complications, necessitating appropriate treatment and long-term specialized care, in children operated on for cataract in early life.

Decreased visual acuity can also be attributed to other vitreous abnormalities associated with syndromes such as Stickler syndrome, which presents with developmental disorders of connective tissue. Stickler syndrome has a complex genetic background. In most cases, it is inherited in an autosomal dominant manner, but there are also patients with the autosomal recessive variant. The primary ocular manifestations in young patients include myopia exceeding –3.0 Dsph, cataract, and retinal detachment. On slit-lamp examination, vitreous abnormalities are characterized by the presence of an "optically empty" vitreous with sterile avascular strands and veils. The presentation can also include skeletal malformations, craniofacial abnormalities, and inner ear complications. The clinical manifestations of Stickler syndrome frequently require differentiation from other systemic disorders [21].

Other causes of visual disorders in children are vascular anomalies: vascular tumors (primarily infantile hemangiomas) and vascular malformations, located in strategic anatomical areas, within the eyelids, in epibulbar tissues or in the orbit (Figure 8) [22].

Hemangiomas occur in approximately 5-10% of full-term newborns. An important role in the development of vascular tumors is attributed to abnormalities in embryo- and angiogenesis processes and an imbalance of pro- and antiangiogenic factors. Hemangiomas have a cyclical growth pattern consisting of the proliferation phase, plateau phase, and involution phase. In the growth phase, which coincides with the period of vision development and consolidation of visual processes, proliferating hemangiomas have the potential to displace the eye, restrict ocular movement, induce strabismus and exophthalmos, and lead to mechanical drooping of the eyelid (pseudoptosis) due to the mass effect of the developmental anomaly, resulting in impaired visual function. Vascular malformations, which affect approximately 4.5% of the general population, originate from localized abnormalities in morphogenesis processes during embryonic and vascular development. While present from birth, vascular malformations may not be noticeable until later in the child's life. They can develop in various regions of the orbital and periorbital space, contributing to a marked loss in visual acuity. Both infantile hemangiomas and vascular malformations may cause aesthetic discomfort, prompting parents to seek medical attention. In addition to the ophthalmic examination, the diagnostic work-up includes imaging diagnostic tests, including ultrasound with vascular flow assessment, magnetic resonance imaging, and computed tomography. The first-line treatment for hemangiomas is pharmacological management

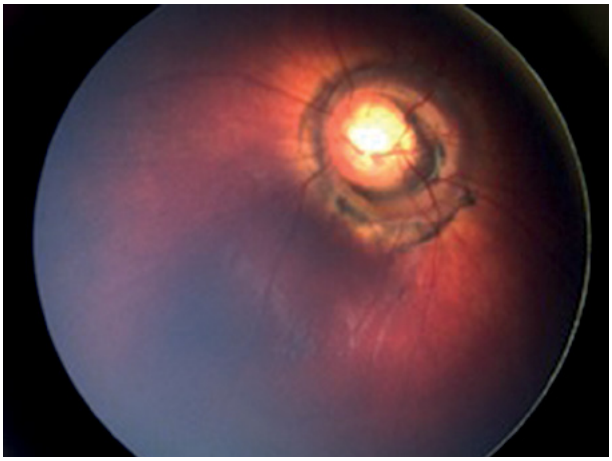

Figure 7. Morning glory disc anomaly. Giant optic disc, disc depression filled with glial tissue. Ridge-like border of the disc with clusters of pigment around the perimeter. Radial pattern of retinal vessels emerging from the disc

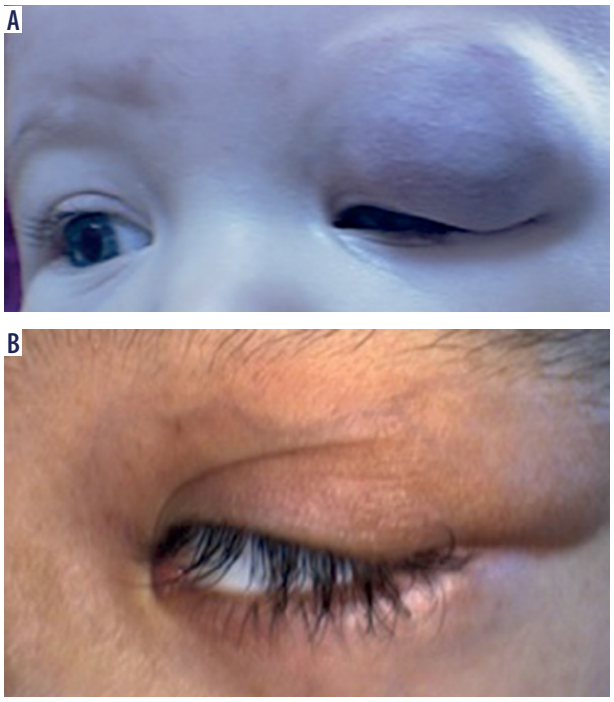

Figure 8. A) Left-sided deep infantile hemangioma of the upper eyelid and orbit. **B**) Left-sided lymphatic malformation of the upper eyelid and orbit

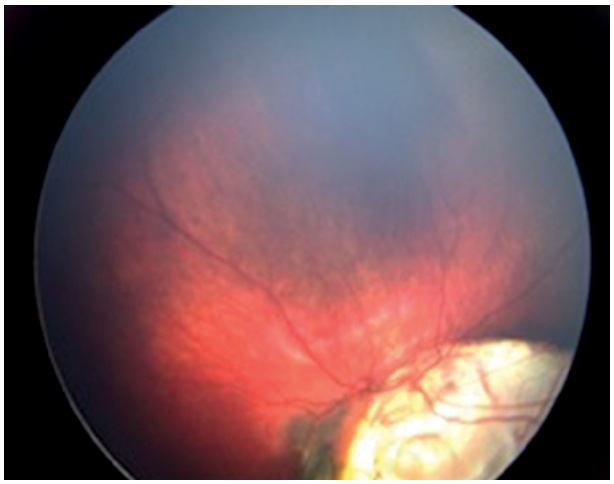

Figure 9. Retinal, choroidal and optic disc coloboma

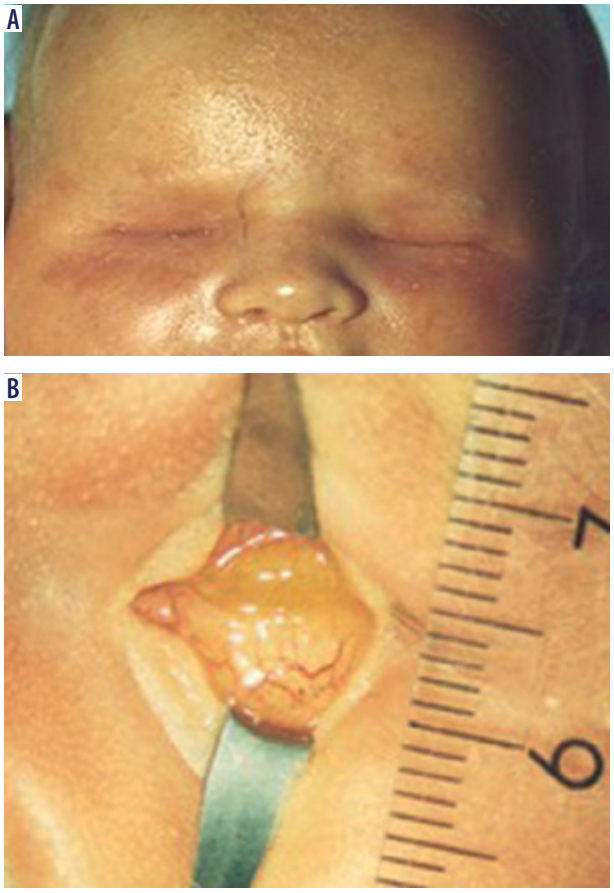

Figure 10. A) Bilateral anophthalmia, sunken eyelids, absence of eyes after eyelid opening. **B**) Anophthalmia; small, shallow empty orbit

with propranolol administered orally in increasing doses (1 to 2.0-2.5 mg/kg bw/day, divided into three doses). Surgical procedures and laser therapy may also be considered as treatment options. Vascular malformations are primarily treated operatively. In some cases, other surgical methods, such as sclerotherapy and laser therapy, are employed.

Intraocular malignant tumors, especially retinoblastoma, can be a major factor leading to vision loss in the youngest

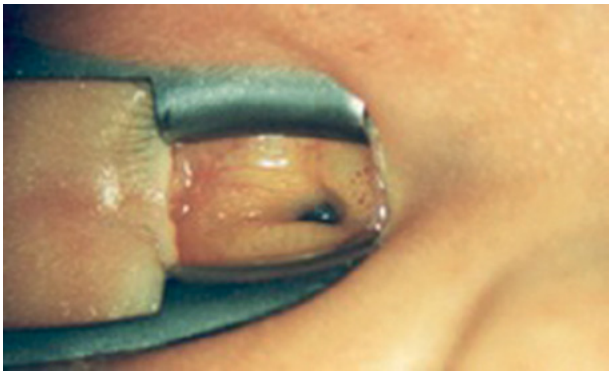

Figure 11. Right-sided microphthalmia, small eyeball located deep in the orbit

children, with a potential risk of losing the affected eye as well. These tumors pose a distinct challenge in the field of pediatric ocular oncology, both in terms of diagnosis and treatment [23].

Visual acuity in children can also be adversely affected by congenital lesions involving the posterior segment of the eye, which are associated with developmental changes in the retina and optic nerve [24]. These are congenital defects, referred to as coloboma, resulting from incomplete closure of the embryonic fissure of the eye during embryogenesis.

Coloboma may occur sporadically or be inherited. It can also be associated with chromosomal abnormalities. Located in the areas of the retina responsible for normal visual processes, such as the fovea or optic nerve disc, coloboma can lead to a decline in visual acuity followed by functional limitations. Typically, coloboma manifests as defects in the inferior nasal portion of the eye fundus. Not uncommonly, abnormalities involving the optic disc, choroid, retina, ciliary body, iris, and lens occur concurrently, forming a complex of abnormalities that can significantly impair children's visual acuity. Congenital coloboma developing in other areas of the fundus is described as atypical. Developmental macular disorders associated with significantly reduced visual acuity may be accompanied by nystagmus, while choroidal coloboma may be a predisposing factor to retinal detachment. In addition to clinical ophthalmological examinations, the diagnostic process comprises imaging diagnostic assessments including ocular ultrasound scanning, optical coherence tomography (OCT) of the macula and optic nerve, and visual evoked potential tests. There is no causative treatment for congenital ocular coloboma. Retinal detachment is typically treated operatively, using advanced surgical techniques.

The diagnosis of anophthalmia and microphthalmia in children is a distressing situation for parents, and especially for the young patients affected by these severe and irreversible disabilities (Figure 10).

Anophthalmia and microphthalmia have an estimated incidence of 3 cases per 100,000 population and 14 cases per 100,000 population, respectively. However, other statistics suggest that the overall incidence of births with these malformations could be as high as 30 cases per 100,000 population [25]. Based on epidemiological data, the risk factors

for anophthalmia and microphthalmia include: maternal age over 40 years, multiple births, and preterm birth with low birth weight and low gestational age. Complete absence of the ocular primordia in the orbit is referred to as anophthalmia, while microphthalmia is defined as an abnormally small eye. The maximum mean normal length of the ocular axis in neonates and adults is approximately 17 mm and 23.8 mm, respectively, while the corneal diameter in patients with microphthalmia is less than 10 mm. Microphthalmia is diagnosed in 3.2-11.2% of blind children (Figure 11).

Mild and moderate microphthalmia is treated conservatively with conformers stimulating the growth of soft and bone tissues within the orbit, with the option of ocular prosthetics. The development of vision in microphthalmic patients depends on the condition of the retina and other changes in the abnormally small eye. It is crucial to optimize the potential of the small, poorly sighted eye which, in some cases, may be the only functional one. In anophthalmia, the primary goal of treatment is esthetic improvement through preparation of the orbit and conjunctival sac for the placement of ocular prosthesis. In cases of severe anophthalmia and microphthalmia, medical management involves reconstruction strategies that aim to enlarge the endo-orbital area, such as implants, expanders, and dermal grafts, and reconstruction of orbital soft tissues, followed by the placement of eye prosthesis [25].

To summarize, each of the congenital ocular abnormalities discussed above can contribute to a significant extent to reduced vision or loss of sight. Any ocular changes noticed by parents or caregivers in newborns, infants, and young children should prompt an immediate ophthalmological examination to assess the condition of the eye and take appropriate action based on the findings.

DISCLOSURE

The authors declare no conflict of interest.

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