# OCULAR COMPLICATIONS OF PREMATURITY

Essay
Submitted for partial fulfillment of
Master Degree in Ophthalmology

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2009

# *Acknowledgement*

*At first and foremost thanks GOD who gave me the power to finish this work.*

*I would like to express my deepest thanks and appreciation to* **Professor Dr. Hoda Mohamed Saber Naeim ,** *Professor of ophthalmology, faculty of Medicine, Ain Shams University, for her great support, valuable time, kind supervision and continuous advices which helped me to overcome many difficulties.*

*I am also deeply gratiful and would like to express my sincere thanks and gratitude to* **Dr. Safaa Saleh Mahmoud Saleh,** *assistant professor of Ophthalmology, faculty of Medicine, Ain Shams University, for her great help, valuable advice and criticism.*

*Also I would like to thank my parents, my wife and all my relatives who participated in a way or another in this work and by being patient with me and providing a good environment to complete this work.*

Victor Girgis Abdel Aziz

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# List of abbreviations

| ABBREVIATION | WORD |
|---|---|
| CMV | Cytomegalo virus |
| FEVR | Familial exudative vitreoretinopathy |
| IUGR | Intrauterine growth retardation |
| LBW | Low birth weight |
| MIGB | Metaiodobenzylguanidine |
| MRI | Magnetic Resonance Image |
| NF | Nerve fiber |
| PM | Postmortem |
| RDS | Respiratory distress syndrome |
| RLF | Retrolental fibroplasia |
| ROP | Retinopathy of prematurity |
| SGA | Small for gestational age |
| VEGF | Vascular endothelial growth factor |
| VLBW | Very low birth weight |
| → | Follow-up or followed by |

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# Introduction

Prematurely born infants are known to have an increased rate of ophthalmological morbidity. The overall incidence of subnormal vision and strabismus in children born prematurely was higher than in a full term population of the same Age (**Holmstrom et al., 1999** ).

Prematurity is especially associated with eye pathology, including retinopathy of prematurity, amblyopia, strabismus and refractive errors. When detected early, amblyopia and many other childhood vision abnormalities are treatable, but the potential for correction and normal visual development is related to age. Since many affected children are asymptomatic, early detection of abnormal visual function requires effective screening throughout early childhood (**Monte and Mills, 1999**).

Amblyopic is, reduction in vision, usually in one but sometimes in both eyes, that is not attributable to anatomic eye or optic nerve pathology. Binocular vision requires that a focused image is formed in each eye that can be fused by the brain into a single image Any problem that interferes with a focused, fusible image during the first 8 to10 years of life is capable of causing amblyopia. The critical sensitive period in the development of amblyopia begins within the first weeks of life and lasts until about 8 to 10 years of age **(Friendly, 1993**).

Strabismus includes an extremely heterogenous group of eye movement problems ranging from, constant to latent, and from congenital to those acquired late in life. Before the age of 6 monthes, coordination of eye movements is poor , and the eyes in a normal infant may be misaligned. Therefore, it is difficult or impossible to diagnose strabismus before six weeks of age (**Wright, 1995**).

Retinopathy of prematurity (ROP) is a disorder of developing blood vessels that occur in premature newborns. Normal retinal vascularization is usually complete around 40 weeks post conceptional age in normal term pregnancies. ROP occurs when the normal pattern of progressive blood vessel growth within the retina is interrupted by premature birth. Vessel growth becomes poorly regulated and excessive in advanced stages, abnormal blood vessels proliferate massively on the retina and into the normally avascular vitreous space in the centre of the eye . These abnormal vessels can leak, bleed and, in subsequent late phases of ROP, may contract, causing distortion or detachment of the retina, leading to severe visual loss or blindness. Severe intravitreal vascularization, fibrosis and retinal detachment lead to the formation of membrane posterior to the lens ( retrolental fibroplasia ) in the most severe cases (**Demorest, 1996**).

The incidence and severity of ROP are highly correlated with the degree of prematurity. The disorder occurs almost exclusively in infant with a birth weight less than 1,500 gm and gestational age of less than 32 weeks. The incidence of ROP in infants with a birth weight less than 750 gm is 90 % and drops to 47 % in those from 1,000 to 1,250 gm. Other associations include the length and concentration of supplemental oxygen use, anemia and other conditions, such as intraventricular haemorrhage and necrotizing entercolitis (**Fuchino et al., 1995**).

Late ocular complications of prematurity include high refractive errors, strabismus and amblyopia. Children with significant prematurity should continue to have periodic complete eye examination (**Repka et al. 2006**).

# Aim of the work

The aim of this work is to discuss the ocular complications of premature infants and how to manage them.

# OCULAR EMBRYOLOGY

## Development of the Eye:

The formation of the eye begins at about the 22nd day of fetal life. During the next 6 – 8 weeks; development is nearly complete. The eye is formed from both ectoderm and mesenchyme. The ectoderm is derived from the neural tube that gives rise to the retina, the fibers of the optic nerve, and the smooth muscles of the iris. The surface ectoderm on the side of the head forms the corneal and conjunctival epithelium, the lens, and the lacrimal and tarsal glands. The mesenchyme forms the corneal stroma, the sclera, the choroid, the iris, the ciliary musculature, part of the vitreous body, and the cells lining the anterior chamber (**Thomas, 2001**).

## The Eye Ball:

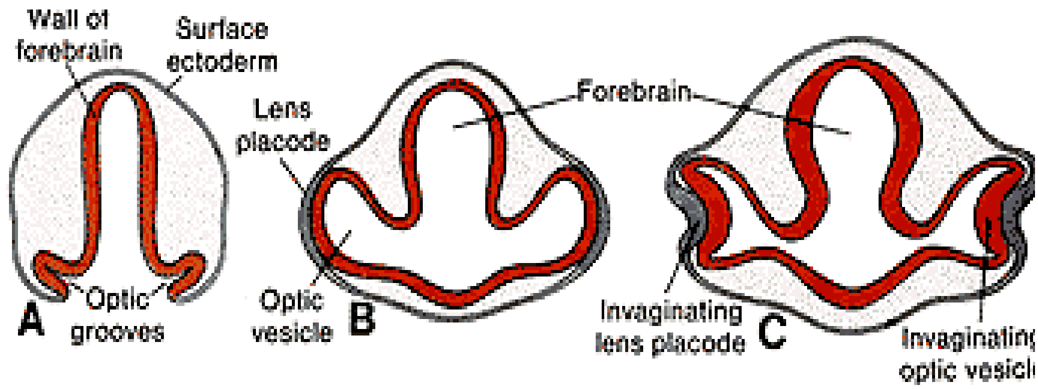
The rudimentary eyeball develops as an ectodermal diverticulum from the lateral aspect of the forebrain. The diverticulum grows out laterally towards the side of the head, and the end becomes slightly dilated to form the optic vesicle, while the proximal portion becomes constricted to form the optic stalk **(fig. 1).** At the same time, a small area of surface ectoderm overlying the optic vesicle thickens to form the lens placode. The lens placode invaginates and sinks below the surface ectoderm to become the lens vesicle. Meanwhile, the optic vesicle becomes invaginated to form the double – layered optic cup. The inferior edge of the optic cup is deficient, and this notch is continuous with a groove on the inferior aspect of the optic stalk called the optic fissure and takes with it the hyaloid artery. Later, this fissure becomes narrowed by growth of its margins around the artery, and by the seventh week of embryonic development the fissure closes, forming a narrow tube, the optic canal , inside the optic stalk **(Fig. 2).** Failure of the fissure to close completely results in coloboma formations, which may include the pupil, ciliary body, and the choroid or optic nerve. By the fifth week, the lens vesicle loses contact with the surface ectoderm and lies within the mouth of the optic cup, the edges of which form the future pupil **(Fig. 3)** (**Thomas, 2001**).

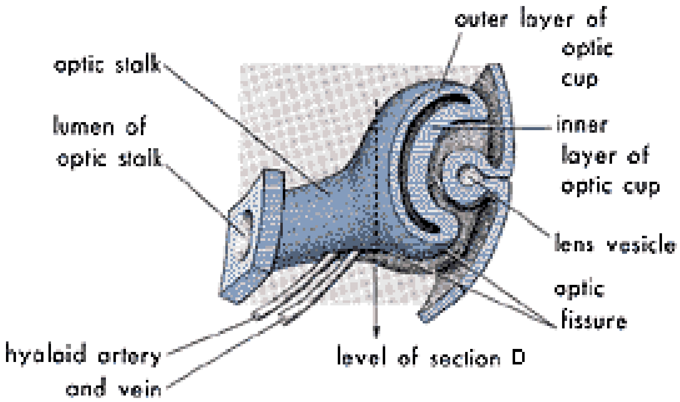
# Primordia of ocular structures:

**Table 1:** primordial of ocular structures (Elizabeth, 2000).

| Surface Ectoderm | Mesoderm | Neural Ectoderm |
|---|---|---|
| lens | Corneal stroma | Sensory retina & R.P.E. |
| Corneal Epithelium | Corneal endothelium & Descement's membrane | Ciliary body Epithelium, Pigmented & nonpigment |
| Conjunctival epithelium | Blood vessels | Pigmented Epithelium of iris |
| Cilia | Sclera | Sphincter pupillae muscle |
| Epithelium, tarsal glands | choroid | Dilator pupillae Muscle |
| Epithelium, Zeiss & Moll glands | Conjunctival stroma, Episclera & tenon capsule | Neural portions of The optic nerve |
| Epithelium lacrimal Gland & accessory lacrimal | Iris stroma & Extrinisic eye muscle | Melanocytes |
| Epithelium lacrimal passages | Ciliary muscle | |
| | Bones of orbit | |
| | vitreous | |

*Eye lids and zonules arise from surface ectoderm and mesoderm.
Φ Bruch's membrane arises from neural ectoderm and mesoderm.




**D**

**Fig.1.:** ( A) Shows the formation of the optic vesicle, which grows out a diverticulum from the lateral aspect of the forebrain. (B) coronal section of diencephalon. Shows a thinkening of the surface ectoderm overlying the optic vesicle to form a lens placode. (C) Coronal section of diencephalons. Shows the lens placode invaginating and sinking below the surface ectoderm. Note that the optic vesicle is also becoming invaginated. (D) Shows the formation of the lens vesicle, the optic cup, and the choroidal fissure (**Thomas,2001**)