Vitrectomy in pediatric Age Group

Essay

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By

Tamer Mostafa Ahmed

M.B.;B.Ch.

Supervised by

Professor Dr. Omar El Zawahry

Professor of Ophthalmology

Faculty of Medicine

Cairo University

Dr. Ahmed Reda Awadein

Lecturer of Ophthalmology

Faculty of Medicine

Cairo University

Faculty of medicine

Cairo University
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Abstract

Vitrectomy is a closed system technique that typically utilizes 3 ports, one port is used to allow intraocular infusion of a balanced saline solution, and the remaining ports are used to introduce various instruments to the vitreous cavity

The incompletely developed infant eye has different spatial relationship than the adult eye, and these differences directly affect vitreoretinal surgical techniques. Because of the anterior insertion of the retina, sclerotomies are placed through the pars plicata to avoid creation of iatrogenic retinal tears

The aim of this essay is to review the indications and complications of vitrectomy in the pediatric age group, and to compare techniques of vitrectomy in pediatric and adult age group, and to highlight the new techniques in pediatric vasectomy.

Key Word:

Vasectomy , Pediatric , age group and to highlight the new technique in pediatric vasectomy

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List of abreviations

- Age-related macular degeneration (AMD).
- Anterior vitreous cortex (AVC).
- Bimanual bipolar diathermy (BBD).
- Cystoid macular edema (CME).
- Epiretinal membrane (ERM).
- Fluid-air exchange (FAX).
- Hyaluronic Acid (HA).
- Intraocular pressure (IOP).
- Internal limiting lamina (ILL).
- Microvitreoretinal(MVR).
- Operating Room (OR).
- Panretinal photocoagulation (PRP).
- Perfluorocarbon (PFC).
- Persistent hyperplastic primary vitreous (PHPV).
- Posterior Vitreous cortex (PVC).
- Posterior vitreous detachment (PVD).
- Retinopathy of prematurity (ROP).
- Transconjunctival sutureless vitrectomy (TSV 25).
- Vascular endothelial growth factor (VEGF).

INTRODUCTION

Vitrectomy is a closed system technique that typically utilizes 3 ports, one port is used to allow intraocular infusion of a balanced saline solution, and the remaining ports are used to introduce various instruments to the vitreous cavity (**Fujii et al.**,2002).

The incompletely developed infant eye has different spatial relationship than the adult eye, and these differences directly affect vitreoretinal surgical techniques. Because of the anterior insertion of the retina, sclerotomies are placed through the pars plicata to avoid creation of iatrogenic retinal tears (**Kim et al.,**1997).

Indications of vitrectomy in children include, lensectomy complications as vitreocorneal adhesions or immersion of lens mass into the vitreous, persistent hyperplastic primary vitreous (PHPV), uveitis, advanced retinopathy of prematurity, trauma consequences as dense vitreous hemorrhage, retinal detachment and traumatic macular holes (**Kohnen**,2001).

Despite potential surgical and postoperative complication, favorable visual outcomes can be achieved following early vitrectomy in dense vitreous hemorrhage (**Simon et al.**,2005).

Pars plana vitrectomy with silicone oil injection can be a viable option in some pediatric cases with complicated retinal detachment (**Karl et al.**,1997).

The transconjunctival sutureless vitrectomy (TSV 25) is a new technique showing substantial progress, which decreases surgical time and postoperative inflammation, with optimal postoperative patient comfort, but currently the accurate selection of patients remains important (**Romero et al.**,2006).

Aim of Work

The aim of this essay is to review the indications and complications of vitrectomy in the pediatric age group, and to compare techniques of vitrectomy in pediatric and adult age group, and to highlight the new techniques in pediatric vitrectomy.

Chapter 1

EMBRYOLOGY OF VITREOUS AND RETINA

VITREOUS EMBRYOLOGY

Interfaces

During invagination of the optic vesicle, the basal lamina of the surface ectoderm enters the invagination along with the ectodermal cells that have become specialized neural ectoderm. The ectodermal cells that are on the surface of the inner with the outer basal lamina give rise to retinal pigment epithelium attached to Bruch's membrane, while the invaginating neural ectoderm and its basal lamina give rise to the neural retinal cells adherent to the internal limiting lamina (ILL). Thus, the basal laminae of both the retina and RPE have the same embryologic origin (Fig.1) (**Sebag and Hageman**,2000).

It is important to appreciate that these basal laminae serve as interfaces between adjacent ocular structures. In the case of the ILL, this basal lamina is the interface between the retina and vitreous. Bruch's membrane separates the RPE and retina from the choroid (neural crest origin).

These interfaces play an important role in a significant biologic event that underlies one of the most devastating causes of blindness in humans: neovascularization. At the ILL interface between vitreous and retina, neovascularization in advanced diabetic retinopathy and other ischemic retinopathies, including retinopathy of prematurity, is a significant cause of vision loss. At the level of Bruch's membrane, an interface of identical embryologic origin as the ILL, neovascularization in age-related macular degeneration is a significant and growing problem. Both of these conditions result from vascular endothelial cell migration and proliferation onto and into interfaces of the same embryologic origin: the basal lamina of the surface ectoderm. Improving our understanding of endothelial cell interaction with these interfaces should provide new

insights into therapy and prevention of these important disorders (**Sebag and Kenneth**,2007).

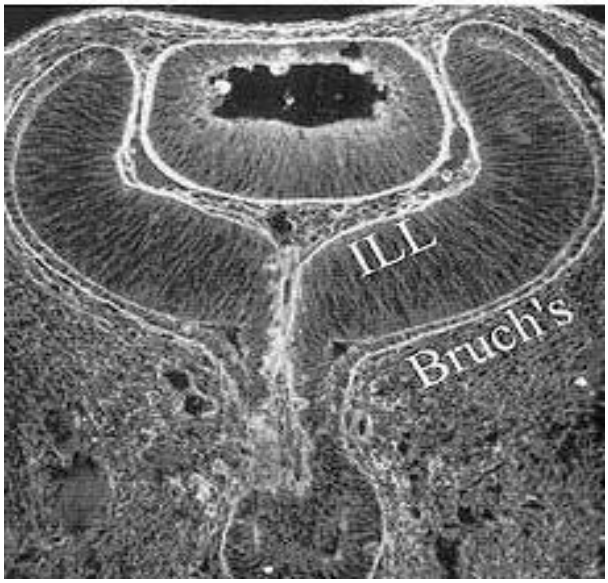

Figure1. This specimen, taken at about the 9-week stage of embryogenesis, was stained with an anti-ABA fluorescent marker that binds to extracellular components of the basal laminae. The continuity of the basal laminae destined to become the internal limiting lamina (ILL) and Bruch's membrane is evident.(**Sebag and Kenneth**,2007)

Vitreous Body

Early in embryogenesis, the vitreous body is filled with blood vessels known as the vasa hyaloidea propria. This network of vessels arises from the hyaloid artery, which is directly connected to the central retinal artery at the optic disc. The vessels branch many times within the vitreous body and anastomose anteriorly with a network of vessels surrounding the lens, the tunica vasculosa lentis. This embryonic vascular system attains its maximum prominence during the ninth week of gestation or 40-mm stage (**Mann**,1964).

Atrophy of the vessels begins posteriorly with dropout of the vasa hyaloidea propria, followed by the tunica vasculosa lentis. At the 240-mm stage (seventh month) in the human, blood flow in the hyaloid artery ceases. Regression of the vessel itself begins with glycogen and lipid

deposition in the endothelial cells and pericytes of the hyaloid vessels. Endothelial cell processes then fill the lumen and macrophages form a plug that occludes the vessel. The cells in the vessel wall then undergo necrosis and are phagoctosed by mononuclear phagocytes (**Jack**,1972).

The sequence of cell disappearance from the primary vitreous begins with endothelial and smooth muscle cells of the vessel walls, followed by adventitial fibroblasts and lastly phagocytes, consistent with a gradient of decreasing oxygen tension (**Balazs et al.**,1980).

It is not known precisely what stimulates regression of the hyaloid vascular system, but studies have identified a protein native to the vitreous that inhibits angiogenesis in various experimental models. Theoretically, such activity seems necessary if a transparent tissue is to inhibit cell migration and proliferation and minimize light scattering to maintain transparency. This may also be the mechanism that induces regression of the vasa hyaloidea propria. Thus, activation of this protein and its effect on the primary vitreous may be responsible for the regression of the embryonic hyaloid vascular system as well as the inhibition of pathologic neovascularization in the adult (**Lutty et al.**,1985).

Recent studies have suggested that vasa hyaoidea propria and the tunica vasculosa lentis regress via apoptosis. Mitchell and colleagues pointed out that the first event in hyaloid vasculature regression is endothelial cell apoptosis and propose that lens development separates the fetal vasculature from VEGF-producing cells, decreasing the levels of this survival factor for vascular endothelium, inducing apoptosis (**Mitchell et al.**,1998).

After endothelial cell apoptosis, there is loss of capillary integrity, leakage of erythrocytes into the vitreous, and phagocytosis of apoptotic endothelium by macrophages, which were felt to be important in this process (**Ito and Yoshioka**,1999).

Meeson and colleagues in 1996 proposed that there are actually two forms of apoptosis that are important in regression of the fetal vitreous vasculature. The first (initiating apoptosis) results from macrophage

induction of apoptosis in a single endothelial cell of an otherwise healthy capillary segment with normal blood flow. The isolated dying endothelial cells project into the capillary lumen and interfere with blood flow. This stimulates synchronous apoptosis of downstream endothelial cells (secondary apoptosis) and ultimately obliteration of the vasculature. Removal of the apoptotic vessels is achieved by hyalocytes (**Meeson et al.**,1996).

A better understanding of this phenomenon may provide insights into new ways to induce the regression of pathologic angiogenesis or inhibit neovascularization in such conditions as proliferative diabetic retinopathy and exudative age-related macular degeneration (AMD). Indeed, the recently developed synthetic VEGF inhibitors seem to be of limited usefulness in treating pathologic neovascularization in exudative AMD and this or a superior inhibitory mechanism may prove to be useful in other proliferative retinopathies, such as retinopathy of prematurity.

When secondary vitreous formation occurs normally, the result is a clear, viscoelastic gel that fills the center of the eye. Because of the intricate interaction between Hyaluronic Acid (HA) and collagen, the vitreous body is transparent centrally, with a dense periphery known as the vitreous cortex. At times there is a visible central structure, known as Cloquet's canal, which is the site of the former hyaloid artery. The solid vitreous body of youth scatters little or no incident light, remaining relatively clear through the first few decades of life until changes occur with aging, initially on a molecular level and ultimately on a macroscopic structural level that affects the entire vitreous (**Sebag and Kenneth**,2007).

RETINAL EMBRYOGENESIS

The eyeball starts to develop from neuroectoderm around the twenty-second day of fetal life. A pair of optic primordium, identified as optic pits, form on both sides of the midline in the ventrolateral region of the primitive forebrain. At approximately the 3-mm stage of development, the optic pits extend outward as hollow spheres ventrolaterally from the neural tube to form the optic vesicles (**Duke-Elder and Cook**,1963).

They become separated from the forebrain by a constriction, or stalk. The cavity of the future third ventricle is continuous with the cavity of the optic stalk and vesicles. The next event is the invagination of the optic vesicles, by differential growth and buckling, to form the optic cup. This occurs around the fourth week of fetal life. The optic cup forms a fold inferiorly and ventrally to form the embryonic fissure through which the mesenchymal and vascular tissues enter the globe. The embryonic fissure begins to close midway in the fissure and extends anteriorly and posteriorly. The process is completed by the end of the seventh week of gestation. Incomplete closure of the fissure can result in colobomas of the iris, retina, or choroid (**Edward and Kaufman**,2003).

The first stage is the differentiation of the cells of the optic vesicle into a two-layered tissue, the neuroepithelium. This stage of development commences simultaneously with the formation of the optic cup. During this process, a layer of ependymal cells that show numerous mitosis appears at the edge of the vesicle. At the base of these cells is a basement membrane from which extend numerous fine cilia. Interdigitation of the cilia with the cells of the outer layer of the optic vesicle is a precursor to the subsequent arrangement of the insertion of the photoreceptor outer segments into the spaces between the microvilli of the pigmented epithelium . Immediately internal to the ependymal layer is the thick layer of primitive undifferentiated cells, which are characterized by cytologic evidence of high mitotic and metabolic activity large oval nuclei with

darkly staining nucleoli and a prominent chromatin net. The ependymal layer and the aggregation of the nuclei of the undifferentiated cells form the primitive zone, constituting the outer 90% of the neuroepithelial thickness. The inner 10% is made up of the marginal zone, which consists of the cytoplasmic extensions of the multitudinous outer primitive cells. The marginal layer is microscopically differentiated by its lack of nuclei until the 10-mm stage at 6 weeks of development (**Duke-Elder and Cook**,1963).

At the beginning of the invagination of the optic vesicle, the distal end of the primitive ophthalmic artery grows into the embryonic fissure from below. When the fissure closes, the artery becomes trapped within the cavity separating the marginal zone from the lens vesicle. Intraocular branches of this transitory hyaloid artery presumably meet the nutrient requirements of the developing retina until its own vasculature is established during the fourth month of gestation (**Susanna**,2007).

From the sixth week to the third month, the neuroepithelium gradually develops into the neuroblastic layers, from which the mature retina subsequently develops. The pattern of this change resembles the morphogenesis of the central nervous system, with the spread of cellular proliferation directed from the inner layer toward the outer layers. Because of its invagination, the neuroepithelium's direction of proliferation is translated into progression from the outer toward the inner layers. The primitive cells internal to the ependymal layer divide and migrate inward to form a distinct new layer of nuclei where the marginal layer had been. This results in the formation of the inner and outer neuroblastic layers, which are separated by the transient nerve fiber layer of Chievitz. This distinct, three-layered structure is temporary. It gradually takes on the more complex architecture of the retina by cellular differentiation. This differentiation of the neuroblastic layers progresses from the inner layer (future ganglion layer) to the outer layer (future photoreceptor layer) such that the outer retina takes on its mature appearance last. In addition, this differentiation of the neuroblastic layers is more precocious in the posterior pole as compared with the periphery