Neovascular Glaucoma

Essay

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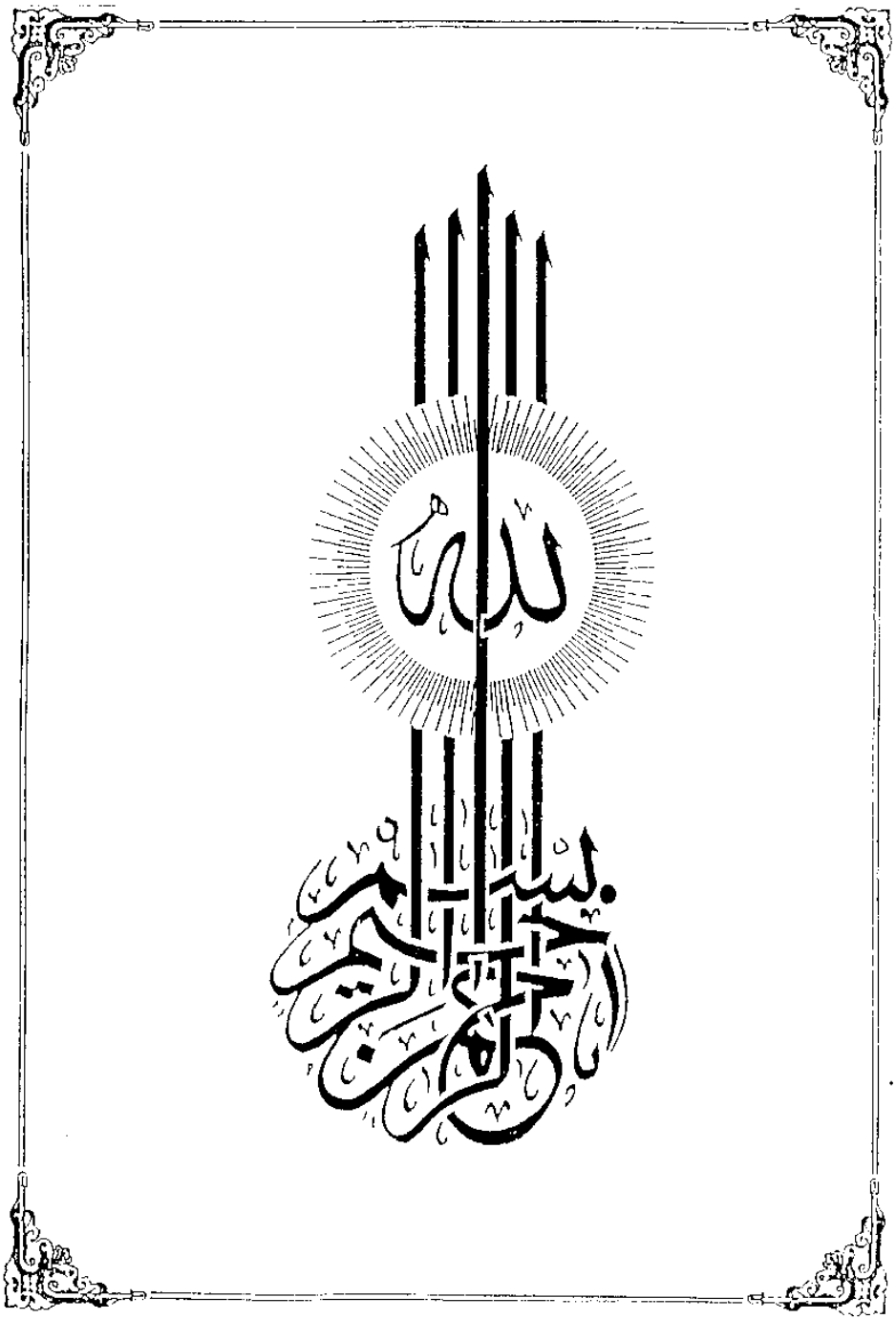

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Introduction

INTRODUCTION

Neovascularization of ocular tissues is one of the most important clinical problems in ophthalmology. The pathogenesis of new blood vessel formation developing within ocular tissues is still unclear and its clinical management is often unsatisfactory. Blood vessels are our link with the external world, bringing nutrient elements to our tissues and carrying away our waste products. The eye has many different vascular channels both in the anatomical and physiological senses, each performing its own special work dependent upon the requirements of the tissue they serve, and no other organ contains such a variety of microvascular systems. As the eye develops in embryonic life, vascular channels are laid down. More channels are provided than are needed for the mature organ and many of the excess vessels atrophy in a remodeling process that leaves a mature vascular system capable of serving normal needs over an entire lifetime. As we age, our blood vessels tend to

degenerate and their degeneration is more than anything else, leading to aging and senescence.

In many disease situations the various mature vascular beds of the eye grow beyond their normal limits; this is neovascularization. Generally in the body, new blood vessels are not bad. Indeed, in most situations they are an important element in the healing process of a damaged tissue. Unfortunately, in the eye neovascularization often interferes with ocular function and thus is undesirable, neovascularization of the iris is often termed "*rubeosis iridis*" (*Henkind, 1978*).

Rubeosis iridis is the term for the clinical counterpart of histologically present neovascularization of the iris. It is present in less than 5% of diabetic patients without proliferative retinopathy, but approximately 50% of patients with proliferative changes in their retinae. The new vessels of the iris arise from the iris venules (*Yanoff, 1989*).

The new vessels grow initially within the iris stroma from the pre-existing iris capillary beds found near the iris root and at the pupillary portion, the intervening iris tissue contains few capillaries.

Fluorescein angiography of the anterior segment may suggest the presence of these hidden intrastromal new vessels because of their abnormal permeability. This intrastromal stage is followed by one in which fine tufts of vessels are found lying anteriorly on the iris either near the pupil or in the angle. These new vessels, unlike normal iris vessels leak fluorescein. They may remain stationary for months or even years without causing problems, or they may enlarge, often rapidly in cases secondary to retinal vein occlusion, cover the entire iris surface and also occlude the angle. When this occurs a severe often intractable neovascular glaucoma develops (*Henkind, 1978*).

Early anterior chamber angle neovascularization causes secondary open angle glaucoma that proceeds rapidly to

closed angle glaucoma because of the peripheral anterior synaechia (*Yanoff, 1989*).

Aetiology

AETIOLOGY

Neovascular glaucoma results from growth of fibrovascular tissue over the surface of the iris (rubeosis iridis) and trabecular meshwork. This results in marked impairment of outflow facility and dramatic elevation of intraocular pressure, often with recurrent hemorrhages into the anterior chamber (hemorrhagic glaucoma). neovascularization usually begins around the pupillary border appearing as fine twig-like new vessels on the iris surface. In some instances the process begins near the iris root with vessels visible only by gonioscopy. Vessels subsequently extend over the entire iris surface.

Contraction of the neovascular tissue leads to peripheral anterior synaechia and a zipper like pull of the iris to the cornea. Before this stage one often sees rubeosis iridis with impaired outflow facility and newly formed blood vessels arborizing over a wide deep angle.

In advanced cases the pupil becomes rigid and non-reactive with ectropion uvea. Pressure elevation may occur abruptly with marked congestion of the globe, corneal edema and severe pain (*Kolker & Hetherington, 1983*).

It may appear that the vessels of the iris are the most sensitive within the eye to stimuli which cause new vessels growth. For example, though it is little appreciated clinically, 80% of eyes with retinoblastoma develop some degree of iris neovascularization. This occurs even though there is neither hint of retinal neovascularization in the majority of such eyes, nor neovascularization elsewhere in the eye. Similarly, iris neovascularization may develop after branch retinal vein occlusion in which only a small area of retina develops new vessels. Central retinal vein occlusion has long been recognized as a major cause of iris neovascularization. However, it is only recently appreciated that there are two forms of central retinal vein occlusion; the ischemic and non-ischemic varieties. Only the former one

leads to iris neovascularization. This finding supports the view that a product formed by hypoxic retinal tissue may cause iris neovascularization. Also the observation that panretinal photocoagulation of conditions with retinal hypoxia can lead to regression of new formed vessels is a further evidence that at least one stimulus of iris neovascularization comes from diseased retinal tissue (*Laatikainen, 1977*).

The clinical diseases leading to rubeosis iridis can be grouped into five main categories (Kottow, 1978) as following:

I. The primary arterial insufficiency:

In these cases no abnormality can be found apart from hypoxic conditions created by insufficient arterial blood supply to the eye or parts of it, as seen in cases of:

- Carotid artery occlusion.
- Ophthalmic artery occlusion.

- central and branch retinal artery occlusion.
- Aortic arch syndrome.
- Cranial giant cell arteritis.
- Sickle cell anemia.
- Retrolental fibroplasia.
- Ciliary artery occlusion.

II. Proliferative vascular diseases of the eye:

These include all tumoral diseases in which vascular malformation is the primary feature such as:

- Hemangioma either retinal or choroidal.
- Coat's disease.
- Leber's miliary aneurysms.
- Persistent hyperplastic primary vitreous.

This group of diseases may have a vasculogenic factor in common which might influence the appearance of rubeosis iridis.

III.Arterial insufficiency with impaired venous drainage:

The combination of failing arterial supply and impaired venous return, causes a disturbance of circulation in these instances:

- Diabetic microangiopathy.
- Eale's disease.
- Stagnation thrombosis.
- Thrombotic retinal vein occlusion.
- Carotid-cavernous fistula.

IV. Neovascular diseases of the eye:

These include all neoplastic growths for which vascular proliferation may be concomitant, but not an essential part of the process, such as choroidal melanoma and retinoblastoma. It is well documented that the tumour cells release vasogenic substances to induce neovascularization for their own metabolic requirements and that some of these substances reach the iris vessels and cause them to proliferate.