PHOTOCOAGULATION TREATMENT OF DIABETIC RETINOPATHY

THESIS
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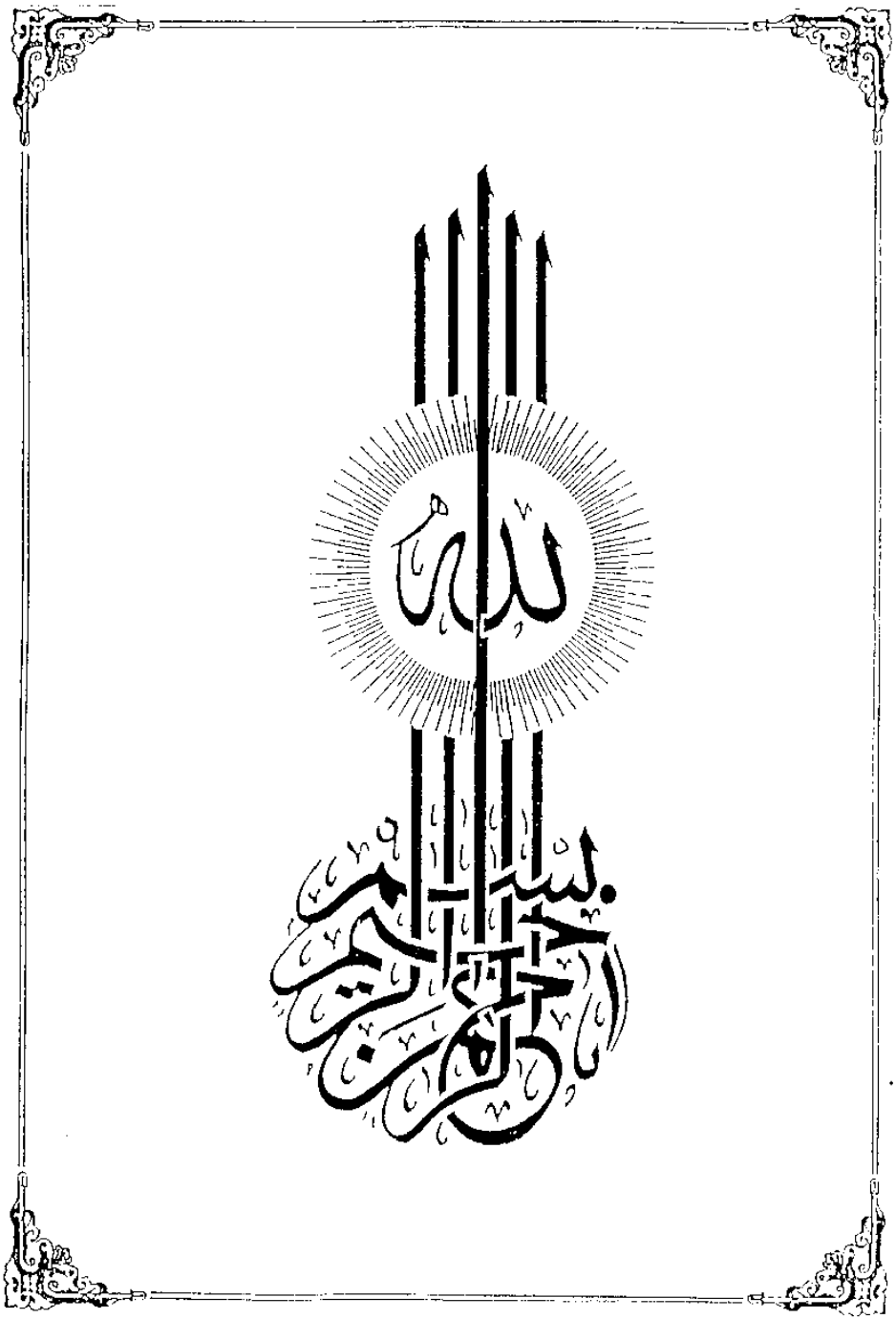

C O N T E N T S

INTRODUCTION

Diabetic retinopathy has become one of the leading known causes, if not the leading cause, of blindness in most of the countries throughout the world. Along with nephropathy and neuropathy, it represents a triad of disastrous complications of diabetes mellitus that can severely limit the functional capacity of an individual. The economic and emotional problems that ensue as the diabetic retinopathy process progresses can be horrendous. In many cases they can lead to decompensation of the patient.

Diabetic retinopathy causes progressive occlusion of the retinal capillaries and finally also of larger vessels. This gradually gives rise to map-like ischemic retinal zones which grow constantly and, because of metabolic deficiency, in turn stimulate the formation of new vessels.

Hence, the essential aim of photocoagulation is to eliminate such ischemic and hypoxic zones and convert them into metabolically inactive scars.

Moreover, the elimination of fairly large peripheral retinal areas leads to a hemodynamic improvement in the remaining central area of the retina.

CLASSIFICATION
OF
DIABETIC RETINOPATHY

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Historical classification :

Hirschberg (1891) presented the first comprehensive classification of diabetic retinopathy . He distinguished between exudative and hemorrhagic retinopathy and divided retinopathy into three types:

Type (I) (retinitis centralis punctata diabetica).

Hirschberg characterized this type as an inflammation of the central part of the retina; he noted that type (I) retinopathy tended to occur in patients aged 45 to 65 with mild diabetes of many years duration .

Type (II) (retinitis hemorrhagica diabetica).

Retinal hemorrhages predominated the fundus picture in this type of retinopathy,followed by other inflammatory changes and abnormalities.The hemorrhages were often small or dot like . These often produced severe visual loss. The larger hemorrhages could also break into the vitreous forming cloudy mobile masses.

In one patient with type II retinopathy, Hirschberg described a tent-like "preretinal connective tissue structure

" protruding above the optic disc. In another patient he described hemorrhagic " clumps " along the retinal surface.

Patients with type II retinopathy could develop venous occlusions or hemorrhagic glaucoma, which indicated a particularly poor visual prognosis .

Type (III) .

This type consisted of rare forms of retinal inflammation and degeneration, but Hirschberg was unsure that these were related to diabetes. Hirschberg's classification did not include proliferative retinopathy specifically, although he described apparent proliferative changes under his type (II) retinopathy. Very little was added to Hirschberg's classification for almost half a century .

In 1934, based upon a study of 1052 diabetics, Wagener, Dry, and Wilder elaborated upon Hirshberg's classification. They distinguished between hard and soft exudates ; venous changes were emphasized, and proliferative retinopathy was noted as specific category .

This classification presented a theory of the natural evolution of diabetic retinopathy, progressing through five distinct phases. Each phase represented a more severe form of retinopathy than the previous phase.

In 1943, Ballantyne and Loewenstein presented their classic paper in which microaneurysms were rediscovered. This work led Ballantyne, in 1946 to a classification that emphasized microaneurysms as distinctive and early lesions of diabetic retinopathy.

Ballantyne noted that microaneurysms represented a breakdown within the capillary circulation, followed by what to day might be termed intraretinal microvascular abnormalities . This, to Ballantyne , indicated a chronic condition of stasis and anoxia on the venous side of the retinal circulation ,followed by grosser changes in the larger retinal veins and ,finally ,by increasingly severe forms of proliferative retinopathy.

Ballantyne and Michaelson's classification, (1962) :

1_ Microlesions

Microaneurysms alone or accompanied by minute hemorrhages and punctate exudates.

2_ Macrolesions

This is the traditional form. Dot and blot hemorrhages ; waxy exudates , discrete or confluent , sometimes circinate but never in the form of macular star .

3_ Vascular changes

Retinal hemorrhages , irregular expansion of the veins , and formation of knobs , loops, coils and leaches ; sheathing of the veins; new-formed intraretinal and preretinal vascular tufts in the vitreous; thrombosis.

4_ Destructive changes .

Intraocular hemorrhages; retinitis proliferans, detachment of the retina and vitreous , secondary glaucoma.

5_ Mixed forms .

The diabetic changes are associated with those characteristics of arteriosclerosis and /or hypertension.

Fabrykant and Gelfand (1965) proposed an even simpler classification , based upon presumably reversible versus irreversible lesions :

[A] Reversible :

Class 1 : Venous changes , microaneurysms.

Class 2 : Microaneurysms, hemorrhages, exudates.

[B] Irreversible :

Class 3 : Above, plus neovascularization .

Class 4 : Above ,plus fibrotic bands and membranes

Beetham(1968)noted that the most useful classification divides retinopathy into only two types, proliferative and nonproliferative. This classification would probably be agreed upon most universally among clinicians today.

In 1973, Scuderi proposed a classification based upon three forms of retinopathy, subdivided in terms of severity:

I Background retinopathy :

A- Early :

(1) Microaneurysms ,punctate hemorrhages.

(2) Punctate retinitis.

B- Advanced :

(1) Hemorrhagic.

(2) Exudative .

C- Severe :

(1) Retinal and preretinal hemorrhages,venous changes.

(2) Vitreous hemorrhage .

(3) Venous thrombosis.

II Proliferative retinopathy .

III Mixed retinopathy .

A- Diabetic and arteriosclerotic

B- Diabetic and renal.

Biochemical classification

Beaumont and Hollows (1972) proposed a classification correlated with biochemical abnormalities present in diabetes. This classification emphasized the potential role of medical treatment for diabetic retinopathy, in conjunction with specific recommendations for pituitary ablation and in selected cases, photocoagulation.

Type I : Diffuse capillary retinopathy .

Defect :

Abnormal growth hormone.

Insulin insensitivity .

Treatment .

Improve diabetic control.

Chemical suppression of growth hormone.

Pituitary ablation.

Type II : Lipid retinopathy .

Defect :

Impaired lipolysis.

Treatment .

Medical, restore normal lipid metabolism.

Type III : Obstructive retinopathy .

Defect :

Obstructive vascular disease .

Altered blood rheology.

Treatment :

Lower intraocular pressure.

Medical, improve fibrinolysis.

Type IV : Minimal retinopathy .

Defect :

Minimal

Treatment :

Reassurance.

VAHEX classification . (Fig. 1)

A classification of the diabetic retinopathy that embodies both the ophthalmoscopic appearance and dynamic factors affecting the ultimate prognosis for eye with diabetic retinopathy. This classification is useful for determining which patient might benefit from photocoagulation.

The VAHEX arcronym is derived from the features of nonproliferative retinopathy.

V, venous dilation; A microaneurysms; H, hemorrhagic activity; E, retinal edema; X, exudative formation.

(L'Esperance 1983)