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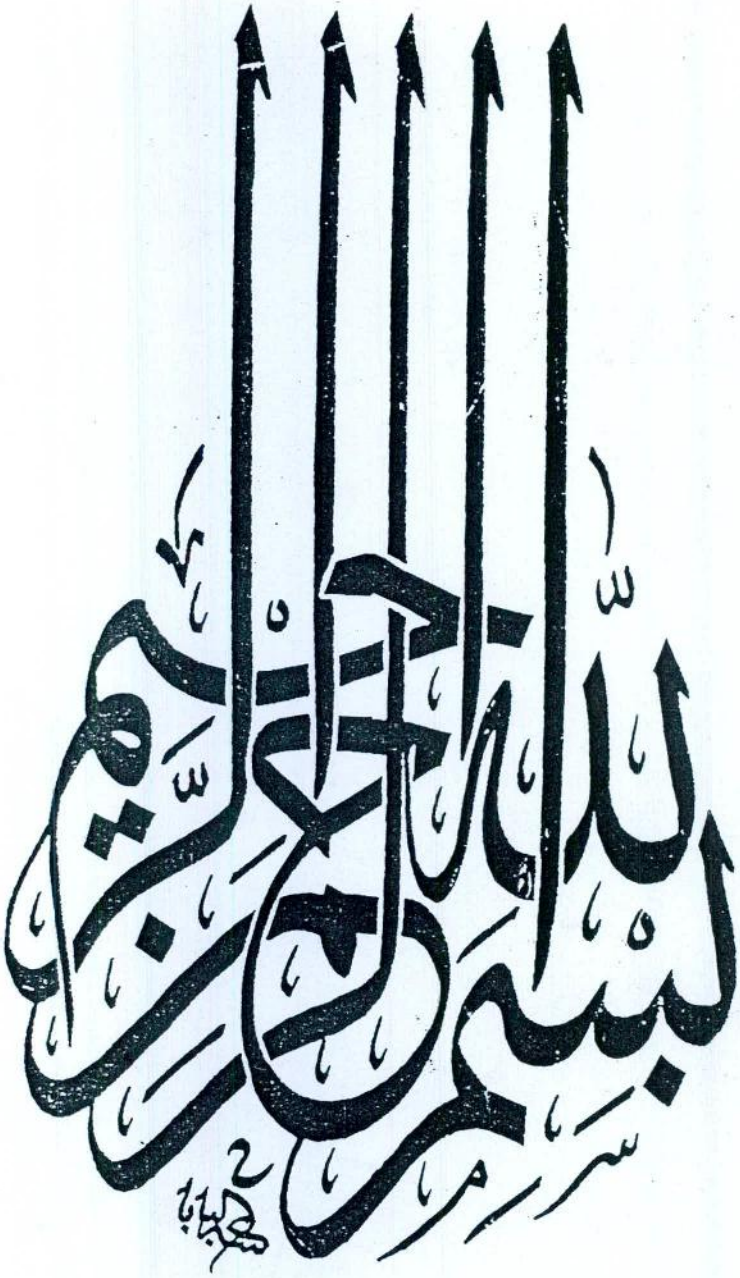

قـالـوا سبحانـك

لاَ عِلمَ لنا إلا ماَ علَمتنا إنكَ أَنت العَليمُ الحَكيم

صدق الله العظيم

سورة البقرة آية ٣٢

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COMPARATIVE STUDY BETWEEN THE USE OF GREEN ARGON LASER AND DIODE LASER PHOTOCOAGULATION IN THE TREATMENT OF CLINICALLY SIGNIFICANT MACULAR OEDEMA IN DIABETIC RETINOPATHY.

Thesis

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INTRODUCTION

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Diabetic retinopathy is one of the major tragedies of the ophthlmology in our present generation and leading to blindness in distressing percentage of cases.[1]

Diabetic retinopathy represent the second cause of legal blindness in many countries.[2]

The incidence of diabetes throughout the world has been estimated to be from 1.5% to 5% of the population.[3]

In Egypt, some surveys have been carried out to determine the incidence of diabetes in the general population and a report by Arab in 1987, revealed a prevalence rate of diabetes of 4.18%.[4]

Diabetic Maculopathy

The nature and extent of macular lesion is frequently the determining factor for visual acuity in eyes affected by diabetic retinopathy.[5]

Diabetic maculopathy is a characteristic component of diabetic retinopathy. Although the aneurysms, cotton wool spots, haemorrhages, serous fluid, and lipid deposits, which are the hallmarks of diabetic maculopathy, may be all found in other retinovascular diseases, the combination of their apperance, bilaterality, symmetry in both eyes, and progression make this a unique form of macular disease.[6]

Pathologic changes in diabetic maculopathy

The pathologic changes of diabetic maculopathy may be divided on anatomical basis into two broad categories.[7]

I- Intraretinal changes :- These include

- Macular oedema.
- Macular hard exudates, which result from increased retinal vascular permeability.
- Macular ischaemia which is caused by retinal vascular occlusion.[7]

II- Preretinal and vitreoretinal changes :- These include.

- Thickening of the posterior hyaloid face.
- New preretinal membrane formation which results from the proliferation of fibrous, glial and fibrovascular tissues.
- Tractional detachement of the macula which is caused by subsequent shrinkage of these tissues and of the vitreous.[7]

Intraretinal Changes

1- Macular Oedema :-

It reflects an increase in retinal vascular permeability, and is defined as excess accumulation of extracellular fluid in the macular region.[8,9]

2- Macular hard exudates:

This is the second type of intraretinal diabetic maculopathy and was called previously exudative maculopathy.

Hard exudate can be defined as yellowish material deposits in the outer layer of the retina [outer plexiform layer] or in the subretinal space in severe cases. They are formed as a result of leakage of plasma

lipoproteins and lipid through abnormally permeable retinal capillaries and microaneurysms.[7]

There are three forms of hard exudates:-

(a) Cluster form:

This is a collection of small white exudates giving a speckled appearance to a localized area of the retina usually a half to one disc diameter in extent. This cluster form is the most transient type of exudative lesion and it appears and disapears in a short period of about 4 months.[10]

(b) Ring form circinate retinopathy:

Here lipoid deposits exist in two forms, a small ring of exdates of about one sixth of the disc area, and a larger aggregation of several disc diameters which often encloses the macula.Their general pattern of formation is that of a continuous process of deposition and reabsorption.[11]

They are located typically temporal, above or below the macula but rarely medial to it. This complex consists of a central area of moderately oedematous retina with usually large microaneurysms and dots haemorrhages.[10]

(C) Large waxy lipid plaques:

They are plaque like deposits either covered or involved in the macular area, variable in size from massive confluent deposits two or three disc diameter in size to small aggregation of about one tenth of disc diameter in extent.

Examination with Hruby lens shows them to extend through all layers of the retina, and the retina itself is often distended two to three times its normal thickness, by what appears to be oedema fluid. Of the three forms

these wax plaques are the most stable and probably remain in the same sites for years, eventually undergoing further degenerative changes.[10]

This typical picture of the so called exudative maculopathy disapears spontaneously with time and often leaves after effects in the macula such as crystaline plaques, fibrinous nodules or pigmentary degeneration.[12]

Hard exudates can affect central visual acuity in two ways:

1- The oedema in the central part of the ring can encompass the fovea.
2- Hard exudates can themselves extend into the fovea causing irreversible damage to neuronal components of the sensory retina.[13]

(3) Macular Ischaemia

It is defined as loss of significant areas of retinal capillary blood flow caused by diabetic vascular disease.[9]

The condition is suspected on ophthalmoscopy when some or all of the following findings are present:

1) Retinal arterioles with diffusely opacified walls.
2) Cotton wool spots or soft exudates (retinal nerve fibre infarct).
3) Large dark, blot haemorrhage (haemorrhagic infarct).
4) Zones of prominent intraretinal microvascular abnormalities (IRMA)[9].

In many eyes, ischaemic changes are accompanied by macular oedema, in which, the relative contribution of oedema and ischaemia to the visual function deficits cannot be separately assessed.[14]

The closure of retinal capillaries is one of the early observable changes in diabetic retinopathy. Small areas of capillary non-perfusion are

so often associated with capillary microaneurysm formation which is considered to be proliferative response to focal ischaemia.[14]

Capillary dilatation, is often accompanied by patchy capillary closure, the dilatation may represents a compensatory response to local ischaemia and hypoxia.[15]

Grading of Macular Ischaemia:

The severity of macular ischaemia may be divided into three grades:

(1) Focal Capillary Dropout:

This is the mildest form of macular ischaemia, the macula appears normal ophthalmoscopically. Small area of capillary non- perfusion surrounded by dialated capillaries are seen by fluorescein angiography. This degree of capillary occlusion is accompanied with normal visual acuity.[7]

(2) Englargement of the normal foveal avascular zone (FAZ):

Englargement of the normal FAZ secondary to occlusion of perifoveal capillaries is a more severe form of ischaemia[7].

The foveola can function fairly normally with FAZ dimensions within the usual normal range, even if obliteration of capillaries from diabetic retinopathy occurs, but once these dimensions are exceeded, visual function deficits can be expected.[14]

So the visual consequence of enlargement of the FAZ is unpredectable. The fovea appears normal by ophthalmoloscopy, so that fluorescein angiography is required to show enlargement of foveal avascular zone which is 500 μ in diameter normally.[7]