

TREATMENT OF DIABETIC
RETNIOPATHY

Thesis

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Requirements for the Master Degree
of ophthalmology



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CHAPTER I
HISTORY

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HISTORY

The word diabetes is derived from a Greek word which means "to run through a siphon", and was given by Aretaeus of Cappadocia (30-90 A.D), (Abd El-Twab 1957; Duncan, 1964). He wrote of the progressiveness and fatal prognosis of the disease. His description of diabetes is the first clear account of his disease we possess (Major, 1964).

Avicenna, the famous Arabian physician (980-1037 A.D.), in his famous textbook [Al Kanoon], gave the first complete clinical picture of the disease with all its complications; stressing gangrene, farunculosis and phthisis (Duncan, 1964; Poulsen, 1967; Ghareeb, 1969), and commented on the sweet taste of urine, which Thomas Willis (1621-1675) described as if "there had been sugar and honey in it." (Abd El-Twab, 1957; Major, 1954; Duncan, 1964; Poulsen, 1967). Since then the disease has been known as Diabetes Mellitus (Poulsen, 1967).

Rollo (1796) suggested the use of a special restricted diet in the treatment of diabetes mellitus

(Abd El-Twab, 1957) and was the first to record the significance of diabetic cataract (Duncan, 1964).

Already before the introduction of the ophthalmoscope it was realized that blindness might occur in diabetes mellitus; either owing to a visible cataract or because of disorders deeper in the eye (Ehlers, 1953). The first description of changes in the fundus of the eye in diabetes was published five years after the invention of the ophthalmoscope and was given by Jaeger in 1856. This was the first recognition of the occurrence of specific retinal changes in diabetes mellitus. (Ballantyne and Michaelson 1962), and it was well described by Mackenzie and Nettelship in 1877. A more detailed description of these changes was given by Hirschberg in 1890.

Since the introduction of Insulin, diabetics lives have been prolonged, opening an era for research workers to study the so called late manifestations of the disease, viz: nephropathy, retinopathy, and others. "They are not really "Late", but have been reported in diabetics of recent onset or even in prediabetics, which are probably inherited rather than acquired" (Ghareeb, 1971).

CHAPTER II

CLASSIFICATION OF DIABETIC RETINOPATHY

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According to Yanoff, 1982, diabetic retinopathy may be classified according to the fundus picture into:

*** Specific constellation of vascular finding.**

- I. Loss of capillary pericytes.
- II. Capillary microaneurysms.
- III. Thickening of retinal capillary basement membrane.
- IV. Arterial-venular connections ("shunts").

*** Exudative Retinopathy.**

- I. "Hard" or "Waxy" exudate.
- II. "Soft" or "cotton wool" spots.
- III. Microcystoid degeneration of the retinal macula.

*** Hemorrhagic Retinopathy.**

- I. Dot and blot haemorrhages.
- II. Splinter (flame-shaped) haemorrhages.
- II. Globular haemorrhages.
- IV. Confluent haemorrhages.
- V. Massive haemorrhages.
- VI. Larger haemorrhages.

*** Pre-proliferative Retinopathy.**

- I. Increased retinal haemorrhages.
- II. Cotton wool spots.
- III. Venous dilatation.
- IV. Venous beading.

Proliferative Retinopathy.

- I. The neovascularization, which is initially intra-retinal, generally, breaks through the internal limiting membrane and lies between it and the vitreous.
- II. Pure neovascularization eventually is accompanied by a fibrous component, is then called retinitis proliferans.

*** Vitreous.**

- I. Vitreous detachment.
- II. Haemorrhage into vitreous compartment.

*** Optic Nerve.**

- I. Neovascularization.
- II. Ischemic (Nonarteritic) optic neuropathy.

*** Central retinal vein occlusion.**

The nonproliferative changes are the first to occur and they may or may not develop into the proliferative phase (Bresnick 1980).

A classification of the clinical forms of diabetic retinopathy which seems to cover all the recognised variation and to relate them to one another in a pathological process is as follows (Ballantyne and Michaelson 1962).

1. **Microlesions:**

Microaneurysms alone or accompanied by minute haemorrhages and punctate exudates.

2. **Macrolesions:**

This is the common form: dot and blot haemorrhages; waxy exudates discrete or confluent, sometimes circinate but never in the form of macular star.

3. **Vascular changes:**

Retinal haemorrhages, irregular tortuosity of the veins and formation of knobs, loops, coils and leashes, sheathing of the veins, newly formed intra-retinal and pre-retinal plexus, vascular tufts in the vitreous and vascular thrombosis.

4. **Destructive changes:**

Intra-ocular haemorrhages, retinitis proliferans, detachment of the retina and vitreous and secondary glaucoma.

5. **Mixed forms:**

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

CHAPTER III

PATHOGENESIS

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PATHOGENESIS

1. **Non Proliferative Phase:**

The variety of fundus changes that occur in diabetic retinopathy are due to the multiplicity of pathological abnormalities, that may exist at all levels of the retinal vascular bed. The obstruction of retinal capillaries and small arterioles begin relatively early in diabetic retinopathy, and is the fundamental aspect of the non proliferative phase of the disease. Microaneurysms formation and many of the venous abnormalities of diabetic retinopathy may be a response to ischemia of the surrounding retina. (Bresnick 1980).

Abnormally increased permeability of the retinal capillaries is another fundamental aspect. The abnormal permeability represent a break down of the normal blood retinal barrier. (Cunha-Vaz 1966).

Capillary Microaneurysms:

The wall of the young aneurysm consists of an endothelial lining and a relatively thin basement membrane. Through a period of months to years, the lesion

may gradually "hyalinize" and appear as a yellow or white spot, hyalinization occur due to gradual thickening of the wall of the aneurysm by the deposition of PAS-positive material. The deposition of lipid in the walls of some aneurysms can also be demonstrated histologically. (Bresnick 1977).

It is possible to document the appearance, growth, hyalinization and ultimate disappearance of individual microaneurysms by serial fundus photography. The disappearance of the aneurysm is due to either progressive hyalinization or to occlusion of the vessel supplies the capillary bed from which aneurysm arise. Rate of appearance and disappearance has been used as a measure of the "Activity" of the retinopathy. (Kohner 1970).

Retinal Haemorrhages:

Pathologic retinal capillaries and retinal microaneurysms are the most common sources of these hemorrhages. The severity of retinal haemorrhages parallels that of the other retinal lesions of non proliferative diabetic retinopathy. (Bresnick 1980).