DIABETIC RETINOPATHY

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Diabetic Retinopathy is well described by Duke Elder and Debree (1967) as: "One of the major tragedies of Ophthalmology of our generation; always common and rapidly becoming still more common, affecting the young as well as the aged; predictable but not preventable, chronic and progressive in its course and leading to blindness in a distressing number of cases."

Diabetic Retinopathy is responsible for about 10% of new cases of blindness at all ages and almost 20% of new cases of blindness between 45 and 75 years. Because it is assocated with a much higher mortality than most other causes of blindness, these proportions are halved if persons who are blind from diabetic retinopathy are compared to the total existing population of blind persons. (Kahn & Heller, 1974). In a survery of the causes and incidence of blindness in the Maltese Islands carried out in 1958, it was found that Diabetic Retinopathy was the cause of 17% of cases of blindness (Damato 1958). Diabetes is rapidly becoming a major Public Health problem in many nations in the world. There is every reason to believe that within the next five to ten years, it will be the leading cause of new adult blindness in many countries in Europe and the U.S.A. (Kupper, 1971). If Diabetes is diagnosed at the age of 20, the risk of blindness at the age of 30 is only 0.1%. It increases to 3.5% at the age of 50 (35 times). For the general population, the risk of blindness from all causes at

age 30 is 0.09%
at age 50 is 0.15 i.e. 1.6 times.

At the age of 50, the diabetic is about 23 times more likely to be blind than his non-diabetic counterpart. (Goldberg, 1971).

Visual disability in Diabetes can be caused by Cataract, Refractive changes, retinopathy, and rubeosis iridis ending in secondary glaucoma.

The preponderance of evidence suggests that a chemical aberration of the carbohydrate metabolism is the common aetiological forerunner of diabetic retinopathy. The intervening pathogenesis responsible for the conversion of carbohydrate intolerance to diabetic retinopathy remains obscure.

Certain conclusions regarding the natural history of this serious retinal disease can be drawn. 2% of all diabetics become blind from Retinopathy alone. This is 10% greater than that of blindness from all causes in the general population. Duration of Diabetes appears to be the primary factor affecting the frequency of retinopathy.

Diagnosis before 30:

after 5 to 9 years	10%
after 15 years	50%
after 25 years	80 to 90%

Ageing makes the retinal vasculature more vulnerable to the diabetic process and makes the older patient more likely than the younger one to develop retinopathy within a given period of time (Goldger, 1971). Caird and Knowles in 1969 summarized evidence showing that the frequency of retinopathy may be reduced and the age of onset raised if control of diabetes is particularly strict during the first five years following the discovery of the disease. However, once most retinal lesions are established, metabolic control has little if any influence on the retinopathy.

This point is controversial. Irregularities of the retinal veins (dilatations, tortuosity, beading) may be reversible with ap-

propriate therapeutic management such as diet and insulin. Permanent lesions such as neovascular and fibrovascular proliferations appear to be minimally influenced by the systematic effects of dietary management and metabolic control.

Regression has been observed in approximatively 10% of cases having neovascular tissue but no vitreous contraction. Instead of following the course of vitreous contraction and associated haemorrhages, neovascular tissue simply regresses and atrophies, so that major extravasations are rare. The factors responsible for this phenomenon are currently unknown. They are obviously important in devising a successful treatment for diabetic retinopathy (Ashton, 1963).

The initial vascular changes in Diabetes occur in the capillary bed. There is a thickening of the capillary membrane which is associated with increased permeability. Its pathological significance, however, is not known. The electron microscope has contributed important information on changes in the basement membrane of the capillaries and other ultra-microscopic structures.

The initial vascular change in the retinal circulation of the diabetic is a venous engorgement which may last for several years. The first sign of retinopathy is the appearance of small punctate haemorrhages usually around the posterior pole on the temporal side. Pathological changes occur in the basement membrane of the capillaries in one suffering from vascular decompensation as a result of venous obstruction. This is a fundamental condition. Whether it results from decompensation of the vascular circulation and partial anoxia, or as a direct result of toxaemia due to diabetes. is not known.

The basement membrane undergoes thickening and vascularization, especially that between the mural cells and the outer border of the capillary. The mural cells (intramural pericyte) undergoes eosinophilic degeneration. This area of the capillary wall is weakened and gives way under intracapillary blood pressure, leading to the sacculated outpouching seen ophthal moscopically as minute pin-point haemorrhages or capillary aneurisms. Serum and blood cells exude through weakened capillary walls and lipid substances are deposited in the retina. Since the circulation is poor, these substances remain as retinal exudates and haemorrhages. Still another and more malignant phase of diabetic retinopathy is the formation of new vessels usually at the disc margin and also in the retina proper.

This is an attempt to re-establish the circulation in area of venous obstruction and compensate for the anoxia of the retinal tissue (Kornweig, 1971).

Photocoagulation is useful where localized areas of newly formed blood vessels can be seen and treated. The study of the results of such treatment suggests the interesting and exciting hypothesis that the progress of diabetic neovascular proliferating retinopathy can be altered by reducing the amount of functioning retina.

The retinochoroidal metabolic or haemodynamic balance can be altered by producing numerous harmless, nonfunctioning choroid retinal scars.

When photocoagulation is applied before the stage of vessel proliferation, the symptoms of diabetic retinopatly are almost always reversible. By treatment in the early stage, late sequelae with proliferations, hemmorhages and retinal detachment can be prevented. If however, vessel proliferation and fibrous changes are already present at the time treatment is started, photocoagulation is too late to stop the progress of the disease (Waubke 1971).

The way in which photocoagulation acts in diabetic angiopathy of the retina is unknown. It has been suggested that changes in the intravascular pressure or an improvement in the oxygen supply to the remaining undestroyed retina may play an important role:

1 — Improvement in the disturbed permeability. The regression of impaired permeability indicates that after photo-coagulation, a functioning blood tissue barrier may be re-established.

2 — Occurrence of a new capillary pattern. The angiogram before light coagulation shows typical findings in capillary

dilatation, micro-aneurysms, haemorr hages, and leakage of dye.

The angiogram taken a year after coagulation shows a pattern of rather large capillaries extending quite regularly over the whole posterior fundus. Some of the microaneurysms are still visible but the leakage o fthe dye has been considerable reduced.

This new pattern is not a temporary effect of the treatment but a long lasting change in the whole capillary network.

This is not found in untreated eyes. The newly formed capillary pattern which develops after photocoagulation seems to demonstrate a new adaptation in both the haemodynamics and metabolism of the retina (Waubke 1971).

In April 1974, an assessment of cases suffering from Diabetic Retinopathy with a view to treatment by light coagulation, was carried out at the O.P.D. along with Dr Duncan and Dr. Cullen of Edinburgh University. Three hundred and thirty one cases were examined: 226 females and 105 males. The fundi of both eyes of every patient were examined under full pupillary dilatation. Visual acuity was also recorded. According to the changes found in the fundi, cases were divided into 5 groups (Table 1):

1 — Exudative
2 — Haemorhagic
3 — Mixed (Haemorhagic and Exudative)
4 — Background retinopathy. Early microaneurysms and scattered exudates.
5 — Proliferative retinopathy.

In the "exudative group" (Table 2) extensive lipoid deposits were found on the macula and around it all over the fundi. Most of the cases were in the 60-69 age group. There were five cases in the 40-49 age group. There was a preponderance of female patients over males. Visual acuity was found to be considerably impaired, and varied from 6/12 to counting fingers.

Haemorrhages varying in size were the main findings in the group 3 (Table 3). There were 3 cases in the 40-49 age group. The largest number of cases was in the 50-59 age group. Visual acuity was better than in the exudative group. A considerable number of these cases were considered suitable for light coagulation.

"In the mixed group", (Table 4) more or less equal changes of the haemorrhagic and exudative varieties were found. The most numerous age group was the 60-69 age group. Vision varied from 6/12 to 6/60. There were some cases suitable for light coagulation. As usual, there were more females than males.

Table 1
Diabetic Retinopathy in Malta

	Females	Males
Exudative	58	21
Haemorrhagic	17	12
Mixed	18	4
Proliferative	60	29
Background	73	39

Table 2
Exudative Retinopathy

Age	Females	Males
40-49	1	4
50-59	7	5
60-69	34	8
70-79	16	4

Table 3
Haemorrhagic Retinopathy

Age	Females	Males
40-49	2	1
50-59	6	6
60-69	5	2
70-79	3	3
80	1	-

Table 4
Mixed Retinopathy

Age	Females	Males
40-49	-	-
50-59	3	2
60-69	10	2
70-79	5	-
80-89	-	-

"Background group", (Table 5) this is by far the most numerous group. There were many more females than males. The main changes are minute micro-aneurysms, pinpoint haemorrhages, minute lipoid deposits and macular oedema. These changes are usually found at the posterior pole. Other areas however showed changes. Vision varied from 6/6 to 6/60.

Most cases are found in the 60-69 age group. There was one case in the 20-29 age group, and another one in the 80-89 age group.

"Proliferative diabetic retinopathy" (Table 6). In this group, there were extensive changes varying from large retinal haemorrhages, lipoid deposits, fibrotic changes, retinal detachment and glaucoma. Vision was very much impaired. As many as 78 cases could be considered as practically blind. The was one case in the 20-29 age group and 2 cases in the 30-39 age group. The numerous age group was the 60-69 age group. Fifty six cases were considered to benefit from treatment by light coagulation. Another group of forty patients required constant watching for possible treatment by light coagulation. There were no indications for pituitary ablation.

The aim of treatment by light coagulation is to seal off the new vessels in order to prevent or at least delay the bleeding sequelae of neovascularization, leading to recurrent vitreous haemorrhages, fibrous tissue formation, retinal detachment and haemorrhagic glaucoma (Guinan 1968). It is known that, in some cases, new vessels regress spontaneously without these severe effects. But this is the exception (Beetham, 1963; Bobree, 1964). Sometimes, too, the new vessels remain almost stationary for long periods. In general, the usual course is one of proliferation. Beetham has shown that the average deterioration time from slight to extreme proliferaive retinopathy is 5 years.

It is mainly in the early stages that treatment by light coagulation seems to show obvious improvement both anatomically and functionally. One may claim that blocking of the progress of the disease and even improvement could be achieved by photocoagulation. Therapeutic results can best be interpreted by considering the morphological details.

Here, comparative fundus photography is the most useful method. Since one can analyse microstructure changes over a long period of time.

Table 5

Background Retinopathy

Age	Females	Males
20-29	1	-
30-39	-	-
40-49	8	5
50-59	11	7
60-69	41	20
70-79	11	7
80-89	1	-

Table 6

Proliferative Diabetic Retinopathy

Age	Females	Males
20-29	-	1
30-39	1	1
40-49	3	2
50-59	21	6
60-69	27	18
70-79	8	1

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