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Fundus picture in advanced Diabetic Retinopathy, a case report and literature

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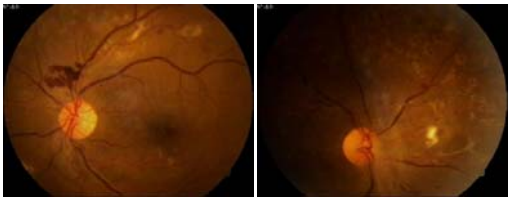
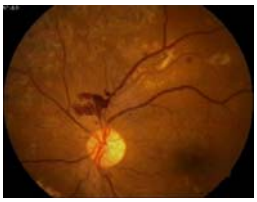
Abstract
A 55yr old female presented with defective vision since 1 yr.

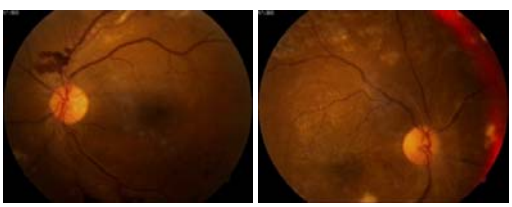
Keywords: Fundus picture in advanced Diabetic Retinopathy.



Introduction
A 55yr old female presented with defective vision since 1yr. she is diabetic and hypertensive since 6 yrs.



Case report: A 55yr old female presented with defective vision since 1yr. **past history**: she is diabetic and hypertensive since 6yrs, and on regular medication. No history of CAD/CVA/Pulmonary Koch's/Epilepsy**. Personal history**: she takes mixed diet. Bowels and micturition were normal. Appetite and sleep were normal**. Family history**: nil significant. **General examination**: patient moderately built, moderately nourished. There is no pallor, cyanosis, icterus, clubbing, pedal edema, lymphadenopathy. BP:150/100mmhg, PR:82/min, CVS:S1S1+, RS: Clear. **Ocular examination**: Vision: Both eyes:6/24 with pinhole 6/24, **Fundal examination**: Both eyes: media: hazy due to lenticular sclerosis, OD:normal, Vessels: Artereolar sclerotic changes present, dot and blot haemorrhages, microaneurysms, hard exudates, superficial haemorrhages and cotton wool spots present, subhyaloidhaemorrhage present, FR: dull. Intra Ocular pressure: Normal. Random Blood Sugar:200mg/dl.CT Brain: normal.



Discussion

Diabetic Retinopathy

A frequent cause of blindness in the United States, diabetic retinopathy is the leading cause in patients aged 20-64 years.

The terminology used for types of diabetes is changing and may be confusing. The American

Diabetes Association (ADA) now uses the term *immune-mediated diabetes* instead of *type* 1 *diabetes*,

Which has traditionally been known as *insulin-dependent diabetes mellitus (IDDM).* The

Change in usage is not universal, however. The ADA has also abandoned the use of *non- insulindependent*

Diabetes mellitus (NIDDM) for *type* 2 *diabetes*, but, again, this change is not yet widespread.

The prevalence of all types of diabetic retinopathy in the diabetic population increases with

The duration of diabetes and patient age. Diabetic retinopathy is rarely found in children younger than 10 years of age, regardless of the duration of diabetes.

The risk of developing retinopathy increases after puberty.(2)

Risk factors:
Duration of diabetes is the most important risk factor. In patients diagnosed with diabetes before the age of 30 years, the incidence of DR after 10 years is 50%, and after 30 years 90%. DR rarely develops within 5 years of the onset of diabetes or before puberty, but about 5% of type 2 diabetics have DR at presentation. It appears that duration is a stronger predictor for proliferative disease than for maculopathy **Poor control of diabetes.** It has been shown that tight blood glucose control, particularly when instituted early, can prevent or delay the development or progression of DR. However, a sudden improvement in control may be associated with progression of retinopathy in the near term. Type 1 diabetic patients appear to obtain greater benefit from good control than those with type 2. Raised HbA1c is associated with an increased risk of proliferative disease.
Pregnancy is sometimes associated with rapid progression of DR. Predicating factors include greater pre-pregnancy severity of retinopathy, poor pre-pregnancy control of diabetes, control exerted too rapidly during the early stages of pregnancy, and the development of pre-eclampsia and fluid imbalance. The risk of progression is related to the severity of DR in the first trimester. If substantial DR is present, frequency of review should reflect the individual risk, and can be up to monthly. Diabetic macular oedema usually resolves spontaneously after pregnancy and need not be treated if it develops in later pregnancy.
Hypertension, which is very common in patients with type 2 diabetes, should be rigorously controlled (<140/80). Tight control appears to be particularly beneficial in type 2 diabetics with maculopathy. Cardiovascular disease and previous stroke are also predictive.
Nephropathy, if severe, is associated with worsening of DR. Conversely, treatment of renal disease (e.g. renal transplantation) may be associated with improvement of retinopathy and a better response to photocoagulation.
Other risk factors include hyperlipidaemia, smoking, cataract surgery, obesity and anaemia.(3)

Pathogenesis

The exact cause of diabetic microvascular disease is unknown. It is believed that exposure to hyperglycemia over an extended period results in a number of biochemical and physiologic changes that ultimately cause endothelial damage. Specific retinal capillary changes include selective loss of pericytes and basement membrane thickening, which favor capillary occlusion and retinal non perfusion, as well as decompensation of the endothelial barrier function, which allows serum leakage and retinal edema to occur.

A large number of hematologic and biochemical abnormalities have been correlated with the prevalence and severity of retinopathy:

increased platelet adhesiveness

increased erythrocyte aggregation

abnormal serum lipids defectivefibrinolysis abnormal levels of growth hormone up regulation of vascular endothelial growth factor (VEGF) abnormalities in serum and whole blood viscosity

However, the precise role of these abnormalities-individually or in combination-in the pathogenesis of retinopathy is not well defined.(2)

Conditions Associated With Potential Visual Loss From Diabetic Retinopathy

Potential visual loss in patients with diabetic retinopathy can be associated with the following conditions:

Macular edema (capillary leakage)

Macular ischemia (capillary occlusion)

Sequelae from ischemia-induced neovascularization.

Classification: The classification used in the Early Treatment Diabetic Retinopathy Study (the modified Airlie House classification) is widely used internationally. An abbreviated version is set out, in conjunction with management guidelines. The following descriptive categories are also in widespread use in clinical practice:

1	**Background diabetic retinopathy (BDR)** is characterized by microaneurysms, dot and blot haemorrhages and exudates. Generally the earlier signs of DR, although persisting as more advanced lesions appear.
2	**Diabetic maculopathy** strictly refers to the presence of any retinopathy at the macula, but commonly reserved for significant changes, particularly vision-threatening oedema and ischaemia.
3	**Preproliferative diabetic retinopathy (PPDR)** manifests cotton wool spots, venous changes, intraretinal microvascular anomalies (IRMA) and often deep retinal haemorrhages. PPDR indicates progressive retinal ischaemia, with a heightened risk of progression to retinal neovascularization.
4	**PDR** is characterized by neovascularization on or within one disc diameter of the disc (NVD) and/or new vessels elsewhere (NVE) in the fundus.
5	**Advanced diabetic eye disease** is characterized by tractional retinal detachment, significant persistent vitreous haemorrhage and neovascular glaucoma.(3)

Classification of Diabetic Retinopathy and Disease Progression

Diabetic retinopathy is classified into an early stage, *nonproliferative diabetic retinopathy*

(NPDR), and a more advanced stage, *proliferative diabetic retinopathy (PDR).*This latterstage is a manifestation ofischemia-induced neovascularization from diabetes. The progressionfrom mild stages of the disease to advanced proliferative changes occurs in a predictable stepwise fashion. The pace of the progression varies among patients. NPDR, alsoknown as *background diabetic retinopathy*, is further graded into mild, moderate, severe, or very severe. PDR is described as early, high-risk, or advanced.

Nonproliferative diabetic retinopathy

Retinal microvascular changes that occur in NPDR are limited to the confines of the retina and do not extend beyond the internal limiting membrane (ILM). Characteristic

findings in NPDR include microaneurysms, areas of capillary nonperfusion, nerve fiber layer (NFL) infarcts, lRMAs, dot-and-blot intraretinal hemorrhages, retinal edema, hard exudates, arteriolar abnormalities, and dilation and beading of retinal veins.

NPDR can affect visual function through 2 mechanisms:

Increasedintraretinal vascular permeability, resulting in macular edema variable degrees of intraretinal capillary closure, resulting in macular ischemia. (2)

Diabetic Macular Edema

Retinal edema threatening or involving the macula is an important visual consequence of abnormal retinal vascular permeability in diabetic retinopathy. The diagnosis of diabetic macular edema (DME) is best made by slit-lamp biomicroscopy of the posterior pole using a contact lens.

Important observations include location of retinal thickening relative to the fovea presence and location of exudates presence of cystoid macular edema.

Fluorescein angiography is useful in demonstrating the breakdown of the blood- retinal barrier by showing retinal capillary leakage. However, angiography should not be used to evaluate for the presence of macular edema, as this should be primarily a slit-lamp biomicroscopic diagnosis. Simple leakage on the angiogram may not always be associated with retinal thickening in the macular area.

DME may manifest as focal or diffuse retinal thickening with or without exudates.

Although an overlap of categories often occurs, 2 general categories of macular edema focal and diffuse- have been described. *Focal macular edema* is characterized by areas of focalfluorescein leakage from specific capillary lesions. It may be associated with rings of hard exudate derived from plasma lipoproteins that appear to emanate from microaneurysms. Resorption of fluid components results in precipitation of lipid residues, usually in the outer and inner plexiform layers but occasionally beneath the sensory retina itself. These residues are the white to yellow deposits described as *hard exudates. Diffuse* macular edema is characterized by widespread retinal capillary abnormalities associated with diffuse leakage from the extensive breakdown of the blood- retinal barrier and, often, with cystoid macular edema. (2)

CSME: Clinically significant macular edema is defined as oedema or hard exudates present within 500micrometres of the foveal centre, or oedema of more than one disc area in extent, any part of the disc is within one DD of the foveal centre. The leakage can be focal or diffuse, resulting in focal or diffuse maculopathy.

Ischemic maculopathy is the result of ischemic changes at the macula. A mixed histological picture of ischemia and oedema at the macular area is fairly common. Patientmay complain of decreasein vision, but could also have normal vision. Clinically CSME is best recognized using slit-lamp biomicroscopy using +78D or +60D lens, as a loss of foveal reflex and thickening at or around the macula. Treatment is by photocoagulation using lasers with long wavelength, such as krypton and diode. (1).

Treatment of DME

Treatment strategies for DME encompass lifestyle modification, exercise, and smoking cessation, as well as better control of blood sugar, blood pressure, blood lipids, and body mass index. A simple pedometer and scales may be more powerful than a laser, medications, or surgery in the long term.

Laser treatment of DME

Many of the current treatment paradigms for managing DME are derived from the Early

Treatment Diabetic Retinopathy Study (ETDRS), a prospective, randomized clinical trial evaluating photocoagulation of patients with diabetes with less than high-risk PDR in both eyes (Clinical Trial S- I). The primary outcome measurement inthe ETDRS was moderate visual loss (MVL), comparing baseline with follow-up visual acuities. MVL was defined as a doubling of the visual angle (eg, a decrease from *20/20* to *20/40* or from *20/50* to*20/ 100),* a drop of IS or more letters on ETDRS visual acuity charts, or a drop of3 or more lines of Snellen equivalent. (2)

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2. American Academy of Ophthalmology, 2011-2012, 110, 112, 113.
3. Clinical ophthalmology Jack J Kanski, 7th Edition, 534, 535, 536.