

**Eye Care Skills: Presentations for Physicians
and Other Health Care Professionals Version 3.0**

Diabetes and Eye Disease

Speaker Notes

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 **AMERICAN ACADEMY
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The Eye M.D. Association

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A GUIDE TO PRESENTING Diabetes and Eye Disease

Diabetes and Eye Disease is a slide-script program for primary care physicians and other medical professionals who care for patients with diabetes.

Although diabetic retinopathy, a serious vision-threatening complication of diabetes, is the leading cause of new blindness in adults aged 20-74, it is estimated that blindness can be prevented in most of these cases. For this reason, the early diagnosis and referral of patients with diabetic retinopathy is essential.

The goal of this program is to enhance primary care physicians' awareness and knowledge of diabetic retinopathy as well as their diagnostic and management skills. The program provides information on the epidemiology, pathophysiology, diagnosis, and management of diabetic retinopathy. Specific referral guidelines are given, and a section on therapy is included to allow the health practitioner to more effectively describe laser surgery and vitrectomy to the patient. The management principles outlined in this slide-script are based in large measure on NIH-sponsored multicenter prospective trials.

Approximate Running Time

45-75 minutes

Suggested Audience

- Medical students
- Family physicians
- Internists
- Diabetologists (adult and pediatric)
- First-year ophthalmology residents
- American Association of Diabetes Educators (member organization)
- Physicians' assistants
- Nurses and nurse practitioners

INTRODUCTION

SLIDE

1

The goal of this presentation is to strengthen the role of the primary care physician and other medical professionals in preventing vision loss from diabetic retinopathy. At the end of this presentation, the participant should be able to identify systemic risk factors of diabetic retinopathy, distinguish among the various stages of retinal disease, describe treatment strategies and screening guidelines for diabetic patients, and recognize the importance of communication between primary care physicians and ophthalmologists.

SLIDE

2

Diabetes mellitus is a ubiquitous disease of enormous proportion. Worldwide, 135 million people are affected, a number expected to increase to 300 million by 2025. Ninety percent of these patients have type 2 (non-insulin-dependent) diabetes. The disease has reached epidemic numbers in many developing nations.

SLIDE

3

Diabetes affects nearly 18 million Americans, at least one third of whom have not yet been diagnosed. Each year, 800,000 new cases of type 2 diabetes are diagnosed in the United States. Minority communities are particularly at risk. African-Americans, Latinos, and native Americans are roughly twice as likely as Caucasians to be affected.

Introduction

DIABETES AND EYE DISEASE: LEARNING OBJECTIVES

- Identify systemic risk factors
- Differentiate clinical stages
- Describe treatment strategies and screening guidelines
- Recognize importance of team approach

Introduction

DIABETES MELLITUS: EPIDEMIOLOGY

- 135 million people with diabetes worldwide (90% type 2)
- 300 million people with diabetes projected by 2025

Introduction

DIABETES MELLITUS: EPIDEMIOLOGY

- 18 million Americans affected
- 800,000 new cases/year (type 2)
- 2x greater risk: African-Americans, Latinos, Native Americans

SLIDE

4

The retinal complications of diabetes, called *diabetic retinopathy*, are the leading cause of blindness in working-age Americans. The American Diabetes Association reports that 12,000 to 24,000 people with diabetes lose their sight each year.

SLIDE

5

Patients who have retinopathy and who are under the care of an ophthalmologist should adhere to a follow-up examination schedule determined by the ophthalmologist in cooperation with the primary care physician. A team approach to achieving tight glycemic control, timely screening, and early treatment will maximize the patient's ocular and systemic prognosis.

Introduction

DIABETIC RETINOPATHY

- Retinal complications of diabetes
- Leading cause of blindness in working-age Americans

Introduction

Primary care physician
+
Ophthalmologist
↓
Systemic control,
timely screening,
and early treatment

SYSTEMIC CONTROLS

Glucose

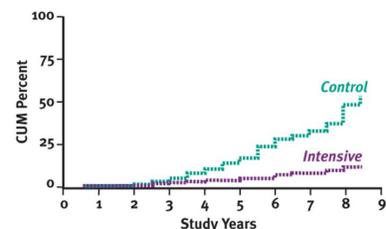
SLIDE

6

The Diabetes Control and Complications Trial (DCCT), a prospective clinical trial sponsored by the NIH, recruited 1400 patients with insulin-dependent (type 1) diabetes in the United States and Canada. Patients with no baseline retinopathy and patients with mild to moderate nonproliferative diabetic retinopathy (NPDR) were randomized between two treatment groups—conventional or intensive therapy. The DCCT demonstrated that intensive glucose control reduces the rate of development and progression of diabetic retinopathy in type 1 patients with and without baseline retinopathy.

Systemic Controls

DCCT: NO BASELINE RETINOPATHY



SLIDE

7

Intensive glucose control also reduces the development and progression of diabetic retinopathy in patients with mild to moderate retinopathy at baseline.

SLIDE

8

Patients in the intensive treatment group achieved glycemic control with insulin pumps or three or more daily insulin injections and frequent home blood glucose monitoring. Those in the conventional group received no more than two daily insulin shots and less frequent glucose monitoring. Both groups were observed for an average of 6.5 years. Patients with no baseline retinopathy who used intensive therapy showed a 27% reduction in developing retinopathy and a 76% reduction in developing more progressive retinopathy.

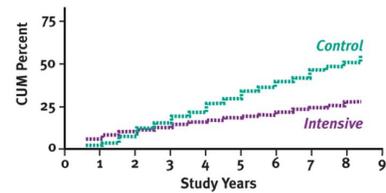
SLIDE

9

Patients with baseline (mild to moderate NPDR) retinopathy who received intensive treatment showed a 54% reduction in the progression of retinopathy, a 47% reduction in the development of severe NPDR or proliferative diabetic retinopathy (PDR), and a 59% reduction in the need for laser surgery. It should be noted that during the early stages of intensive management, pre-existing retinopathy may worsen.

Systemic Controls

DCCT: MILD TO MODERATE RETINOPATHY



Systemic Controls

DCCT: INTENSIVE GLUCOSE CONTROL, NO BASELINE RETINOPATHY

- 27% reduction in developing retinopathy
- 76% reduction in risk of developing progressive retinopathy

Systemic Controls

DCCT: INTENSIVE GLUCOSE CONTROL, MILD TO MODERATE NPDR

- 54% reduction in progression of retinopathy
- 47% reduction in development of severe NPDR or PDR
- 59% reduction in need for laser surgery
- Pre-existing retinopathy may worsen in early stages of treatment

Hypertension

SLIDE

10

This study is an extension of the DCCT that showed that initial glycemic control has long term benefit. After completion of the DCCT the intensive and standard control groups were followed for an additional 2 to 3 years. Even though the A1C median leveled out there was a statistically decreased risk of macular edema and vitreous hemorrhage and progression of retinopathy in the initial tight-control group.

SLIDE

11

The United Kingdom Prospective Diabetes Study (UKPDS) was a prospective, randomized trial of patients with type 2 diabetes. Intensive glucose control with insulin or oral hypoglycemic agents was compared to conventional therapy in which patients were initially treated with diet alone. In addition, intensive blood pressure control was compared to conventional management. Reduction of both glucose and blood pressure with intensive control was found to decrease the rate of progression of diabetic retinopathy.

SLIDE

12

The UKPDS demonstrated that reducing hemoglobin A_{1C} from 7.9 to 7.0 with intensive therapy was associated with a 25% decrease in microvascular complications (as exemplified by a corresponding decrease in the need for retinal laser surgery) and that reducing blood pressure to less than 150/85 mm Hg was associated with a 34% reduction in the progression of retinopathy.

Systemic Controls

EDIC

- 8.2 % vs 7.9 %
- ↓ ME
- ↓ PPDR, PDR
- ↓ VH
- ↓ laser

Epidemiology of Diabetes Interventions and Complications

Systemic Controls

UKPDS: TYPE 2 DIABETES

- Increased glucose and BP control decreases progression of retinopathy

Systemic Controls

UKPDS: RESULTS

- Hemoglobin A_{1C} reduced from 7.9 to 7.0 = 25% decrease in microvascular complications
- BP reduced to <150/85 mm Hg = 34% decrease in retinopathy progression

SLIDE

13

The UKPDS found that control of hypertension in type 2 diabetes is just as important as glucose control in delaying the onset or progression of diabetic retinopathy. Angiotensin-converting enzyme (ACE) inhibitors and beta blockers were equally effective in lowering blood pressure and decreasing the risk of progression of retinopathy.

SLIDE

14

To help realize the benefits demonstrated by the DCCT and the UKPDS, all physicians should familiarize themselves and their patients with diabetic ocular complications, the benefits of maintaining strict glycemic and blood pressure control, and the need for appropriate examination schedules.

Systemic Controls

UKPDS: HYPERTENSION CONTROL

- As important as glucose control in lowering rate of progression of diabetic retinopathy
- ACE inhibitor or beta blocker decreases microvascular complications

Systemic Controls

DCCT/UKPDS LESSONS

- Professional and patient education
- Good glucose and BP control
- Regular examination

Additional Systemic Controls

SLIDE

15

Diabetic nephropathy and proteinuria have been associated with more advanced retinopathy and macular edema. Optimizing the kidney status may be beneficial to the retina. ACE inhibitors such as lisinopril are known to be of benefit to the diabetic kidney. The EUCLID study (European Controlled Trial of Lisinopril in Insulin-dependent Diabetes) found that lisinopril decreases the progression of retinopathy in normotensive patients with type 1 diabetes.

Systemic Controls

ADDITIONAL SYSTEMIC CONTROLS

- Proteinuria is a risk factor for macular edema
- Lisinopril may benefit the diabetic kidney and retina even in normotensive patients

SLIDE

16

Data suggest that elevated total cholesterol is directly associated with the presence or development of hard exudates, an adverse feature of macular edema. It remains to be seen if lowering serum cholesterol will decrease hard exudates and improve the visual prognosis of diabetic macular edema.

Systemic Controls

High cholesterol may be associated with increased macular exudates and vision loss.

Diabetic Retinopathy and Cardiovascular Disease

SLIDE

17

Findings of the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR) show special interrelationships between proliferative diabetic retinopathy and cardiovascular disease. The presence of PDR was observed to be an important risk indicator for the development of myocardial infarction, stroke, and amputation. These findings were noted after controlling for age and duration of diabetes. In addition, patients with PDR were at higher risk of developing diabetic nephropathy.

Systemic Controls

WESDR: DIABETIC RETINOPATHY AND CARDIOVASCULAR DISEASE

- PDR a risk indicator for MI, stroke, amputation
- PDR elevates risk of developing nephropathy

PATHOGENESIS

SLIDE

18

Elevated glucose levels may result in various metabolic abnormalities, leading to an increased production of multiple growth factors, especially vascular endothelial growth factor (VEGF). VEGF causes increased capillary permeability and abnormal vasoproliferation.

Pathogenesis

DIABETIC RETINOPATHY: PATHOGENESIS

Increased glucose



VEGF



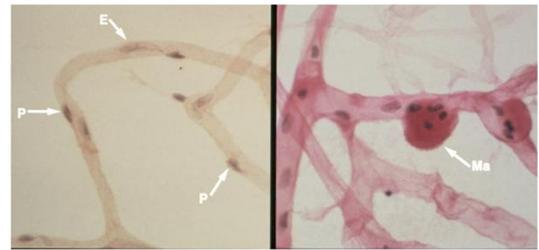
Increased capillary permeability/
abnormal vasoproliferation

SLIDE

19

The trypsin digest—a light microscopic study of retinal blood vessels—on the right highlights an early change of diabetic retinopathy: loss of pericytes, the supportive cells of the retinal vasculature, which are more numerous in the normal digest on the left. Endothelial cells are lost later. These abnormalities are followed by the formation of microaneurysms, leaky outpouchings of the capillary walls that represent the first clinically detectable evidence of diabetic retinopathy. Other sequelae include abnormal permeability, capillary nonperfusion, and neovascularization.

Pathogenesis



Normal

Diabetic retinopathy

CLINICAL STAGES OF RETINOPATHY

SLIDE

20

The effects of diabetes on the retina are manifested in progressive stages that are defined by ophthalmoscopic criteria. In the first stage, termed *nonproliferative diabetic retinopathy* (NPDR), features that reflect retinal capillary damage and breakdown in the blood-ocular barrier are noted. In the second stage, called *preproliferative diabetic retinopathy* (also referred to as *severe NPDR*), ischemic retinopathy is more prominent. In the third stage, proliferative diabetic retinopathy (PDR), progressive retinal ischemia promotes the growth of fragile new blood vessels, or neovascularization.

Clinical Stages of Retinopathy

DIABETIC RETINOPATHY: CLINICAL STAGES

- Nonproliferative diabetic retinopathy (NPDR)
- Preproliferative diabetic retinopathy
- Proliferative diabetic retinopathy (PDR)

Nonproliferative Diabetic Retinopathy

SLIDE

21

Ophthalmoscopy reveals microaneurysms, hard exudates, and intraretinal hemorrhages with mild to moderate NPDR. Each of these abnormalities may occur in isolation or in combination. Patients may be asymptomatic at this stage, even as their disease is causing severe retinal damage.

Clinical Stages of Retinopathy

MILD TO MODERATE NPDR

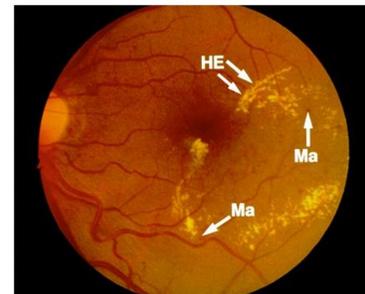
- Microaneurysms
- Hard exudates
- Intraretinal hemorrhages
- Patients may be asymptomatic.

SLIDE

22

Microaneurysms are usually the earliest visible manifestation of diabetic retinopathy. They appear as tiny red dots scattered in the retina posteriorly (single arrows). They may be surrounded by a ring of yellow lipid, or hard, exudates (double arrow). Exudates are the result of vascular leakage, but the specific leaking microaneurysm may not be visible ophthalmoscopically.

Clinical Stages of Retinopathy



Microaneurysms

SLIDE

23

Intraretinal hemorrhages appear either as red dots or blots, sometimes indistinguishable from microaneurysms, or as flame-shaped hemorrhages.

Clinical Stages of Retinopathy



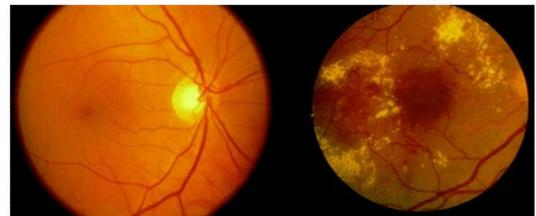
Intraretinal hemorrhages

SLIDE

24

Vascular leakage, fluid, and/or exudate in the macula is referred to as diabetic macular edema (DME). Compare the healthy macula on the left with the edematous macula, caused by leaking microaneurysms, on the right. When diabetic macular edema involves or threatens the fovea, it is termed clinically significant macular edema (CSME). DME is the most frequent cause of moderate visual loss that may occur at any stage of retinopathy, although it is more common and severe in the later stages. It also may be associated with normal vision and may be difficult to appreciate ophthalmoscopically.

Clinical Stages of Retinopathy



Healthy macula

Edematous macula

SLIDE

25

The prevalence of diabetic macular edema is strongly associated with the duration of diabetes, according to the Wisconsin Epidemiologic Study of Diabetic Retinopathy (Klein et al, 1989b). Macular edema was present in about 5% of patients who had diabetes for 5 years or less. However, the prevalence of edema rose to about 15% among patients who had had diabetes for 15 years or more. The prevalence was slightly lower for diabetic patients not using insulin. The study also found that higher glycosylated hemoglobin levels were associated with a higher prevalence of macular edema (Klein et al, 1988).

Clinical Stages of Retinopathy

DIABETIC MACULAR EDEMA

- Diabetes ≤ 5 yrs = 5% prevalence
- Diabetes ≥ 15 yrs = 15% prevalence

SLIDE

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NPDR may also be associated with cotton-wool spots, superficial white lesions with feathery edges due to infarction of the nerve fiber layer of the retina causing axoplasmic stasis.

Clinical Stages of Retinopathy



Cotton-wool spots

Preproliferative Diabetic Retinopathy

SLIDE
27

In preproliferative diabetic retinopathy (PPDR), more ominous ischemic lesions occur. These include irregular dilations of retinal veins, called venous beading (shown on the slide by the arrows), intraretinal microvascular abnormalities or capillary shunt vessels (shown on the slide by the arrowheads), and extensive retinal hemorrhages. Once these signs have appeared, approximately 50% of patients will develop proliferative retinopathy within 1 year. It may be beneficial, particularly for patients with type 2 diabetes, to begin panretinal laser therapy at this stage of disease to prevent long-term visual loss.

Clinical Stages of Retinopathy



Venous beading and capillary shunt vessels

Proliferative Diabetic Retinopathy

SLIDE
28

Proliferative diabetic retinopathy (PDR) is the major cause of blindness in diabetes. The ophthalmoscopic features include new retinal or optic disc blood vessels (neovascularization) that may be complicated by vitreous hemorrhage (the most common cause of blindness) and traction retinal detachment. Any of the nonproliferative findings, including macular edema, may also be present.

Clinical Stages of Retinopathy

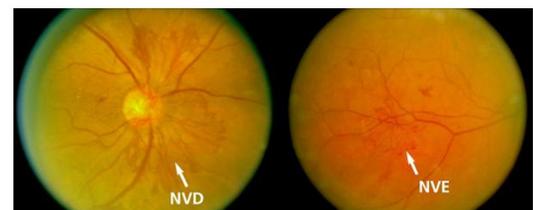
PDR: CLINICAL SIGNS

- Neovascularization
- Vitreous hemorrhage and traction
- NPDR features, including macular edema

SLIDE
29

The delicate new vessels that grow on the surface of the retina resemble a tangle of hair or a fishnet. Neovascularization originating around the optic nerve head is referred to as new vessels at the disc (NVD), shown on the left. If originating elsewhere on the retina, the neovascularization is called new vessels elsewhere (NVE) shown on the right.

Clinical Stages of Retinopathy



New vessels at the disc

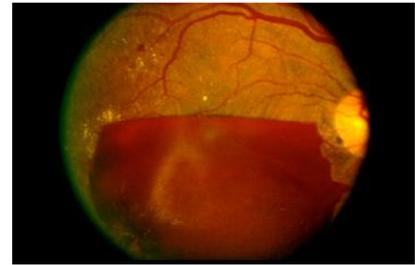
New vessels elsewhere

SLIDE

30

As these fragile new vessels grow, they may bleed into the vitreous cavity, a condition called vitreous hemorrhage.

Clinical Stages of Retinopathy



Vitreous hemorrhage

SLIDE

31

Vitreous hemorrhage may be mild, perceived by the patient as dark spots or floaters. Alternatively, vitreous hemorrhage may be more severe and may fill the vitreous compartment with blood, decreasing the patient's visual acuity to light perception only. Patients with these symptoms should be examined immediately by an ophthalmologist to rule out other causes of blindness such as retinal detachment and to assess for laser therapy.

Clinical Stages of Retinopathy

VITREOUS HEMORRHAGE: SYMPTOMS

- Floaters
- Severe visual loss
- Requires immediate ophthalmologic consultation

SLIDE

32

Fibrosis may accompany neovascularization. Fibrous elements may contract, causing distortion, tearing, or detachment of the retina and subsequent visual loss. Severe distortion of the retinal architecture due to fibrovascular proliferation is noted in this photograph.

Clinical Stages of Retinopathy



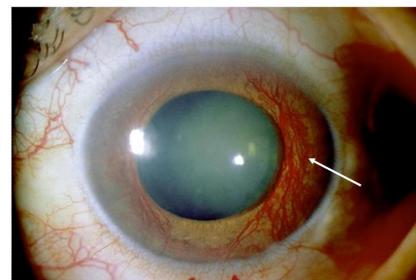
Severely distorted retinal architecture

SLIDE

33

Neovascularization in diabetes is not always confined to the retina. Retinal ischemia may induce new vessel growth on the iris surface, shown by the arrow. Iris neovascularization, or rubeosis iridis, may lead to peripheral iris adhesions blocking the normal drainage of aqueous fluid from the eye, potentially causing acute angle-closure glaucoma.

Clinical Stages of Retinopathy



New vessel growth

SLIDE

34

Insulin-using diabetic patients diagnosed before age 30 have a rare risk of developing PDR within the first 5 years after diagnosis. However, after 15 years' duration of DM, the prevalence rises to 25%, and to 55 % after 20 years.

Clinical Stages of Retinopathy

INSULIN USERS Dx <AGE 30

Duration (yrs)	PDR Prevalence
5	negligible
10	25%
15	55%

SLIDE

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In diabetic patients diagnosed after age 30, the prevalence of PDR is 20% in insulin-using patients 20 years after diagnosis, but is less common in non-insulin users.

Clinical Stages of Retinopathy

INSULIN USERS Dx >AGE 30

Duration (yrs)	PDR Prevalence
20	20%

PDR less common among noninsulin users

SLIDE

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In summary, the clinical stages of diabetic retinopathy include nonproliferative diabetic retinopathy (NPDR), in which patients may be asymptomatic; preproliferative diabetic retinopathy (PPDR), during which laser therapy may help preserve vision; and proliferative diabetic retinopathy (PDR), a major cause of severe visual loss.

Clinical Stages of Retinopathy

REVIEW OF CLINICAL STAGES

- NPDR: Patients may be asymptomatic
- PPDR: Laser therapy at this stage may help prevent long-term visual loss
- PDR: Major cause of severe visual loss

DIAGNOSIS

SLIDE

37

Careful and detailed ophthalmoscopic examination through dilated pupils is an important technique used to accurately grade diabetic retinopathy and to assess the need for laser photocoagulation surgery. Although most of the abnormalities are concentrated in the posterior retina, they may be difficult to appreciate with a direct ophthalmoscope, which precludes stereoscopic, wide-field viewing.

SLIDE

38

Detailed grading of retinopathy and accurate recognition of macular edema are aided by the magnified, stereoscopic view of the slit lamp biomicroscope. The wide-field view of the indirect ophthalmoscope allows visualization of the far retinal periphery, which is essential for a complete examination.

SLIDE

39

Fundus photography (left) and fluorescein angiography (right) can document areas of vascular leakage, nonperfusion, and neovascularization. Fluorescein, a yellow dye that fluoresces under cobalt-blue light, is injected intravenously. A series of photographs is taken as the dye courses through the retinal vasculature. Fluorescein angiography is useful in guiding laser surgery for macular edema. Note the leaky microaneurysms (arrows) in this patient with CSME.

Diagnosis



Ophthalmoscopic examination through dilated pupils

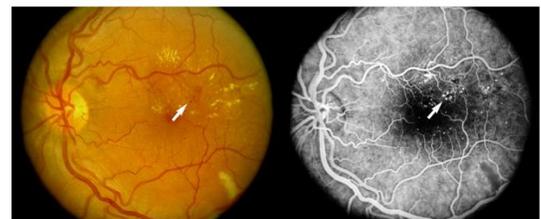
Diagnosis



Slit-lamp biomicroscopy

Indirect ophthalmoscopy

Diagnosis



Fundus photography

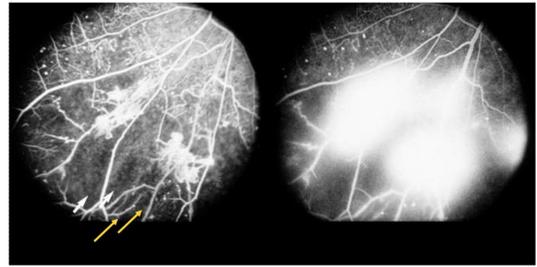
Fluorescein angiography

SLIDE

40

Fluorescein angiography is also very sensitive in detecting fronds of neovascularization that leak profusely during the course of the angiogram. Note the dark, hypofluorescent patches (arrows) associated with the neovascular fronds, indicative of ischemia.

Diagnosis



Dark, hypofluorescent patches indicative of ischemia

TREATMENT

SLIDE

41

The principal method used in treating diabetic retinopathy is laser photocoagulation surgery. Topical anesthesia is given; retrobulbar anesthesia may also be necessary. During the procedure, the fully alert patient sits at a device similar to a slit lamp; a contact lens is placed on the eye, allowing a magnified, stereoscopic view of the area to be treated.

Treatment



Laser photocoagulation surgery

SLIDE

42

Photocoagulation is usually accomplished with the argon laser, which delivers green light that is well absorbed by the retinal pigment epithelium (RPE) beneath the retina. A white laser burn of the retina and RPE is produced by the absorption of the heat energy, as demonstrated in this photograph of acute panretinal laser photocoagulation burns.

Treatment



Acute panretinal laser photocoagulation burns

Laser Photocoagulation for Macular Edema

SLIDE

43

The photocoagulation technique used to treat CSME, known as focal macular laser, differs from that used to treat proliferative retinopathy. Focal macular laser uses a limited distribution of laser spots, delicately placed within the bed of retinal edema, and may include the direct treatment of associated leaking microaneurysms using yellow wavelength. As the edema is gradually resorbed, visual acuity may stabilize or even improve.

Treatment

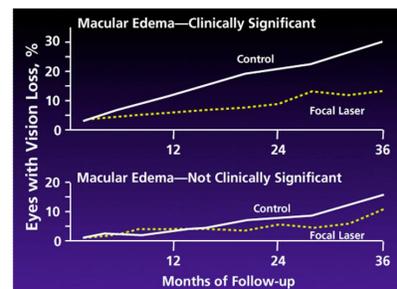


SLIDE

44

According to the report of the Early Treatment Diabetic Retinopathy Study (1985), sponsored by the National Eye Institute, the incidence of visual loss from CSME was reduced by focal macular laser from 16% to 7% after 2 years and from 24% to 12% after 3 years (top graph). However, the benefit of focal macular laser was substantially attenuated in patients with macular edema that was not clinically significant (bottom graph).

Treatment



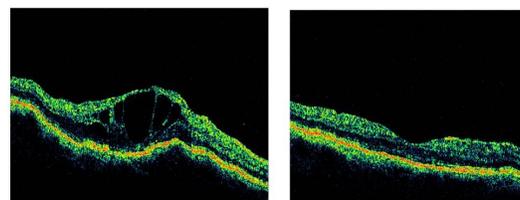
SLIDE

45

Optical coherence tomography (OCT), is a more novel imaging technology that uses a laser source of light to create an optical cross section of the macula similar to ultrasonography. It is a useful technique in the diagnosis and monitoring of diabetic macular edema, especially cystoid macular edema (CME). CME is often recalcitrant to laser therapy. Some degree of success in treating CME has been achieved with intravitreal steroid injection and more recently intravitreal antiVEGF injection (eg, Avstin therapy). This slide shows an example of DME and CME which resolved status post intravitreal Kenalog injection.

Treatment

MACULAR EDEMA TREATMENT WITH TRIAMCINOLONE INJECTION



OCT before

OCT after

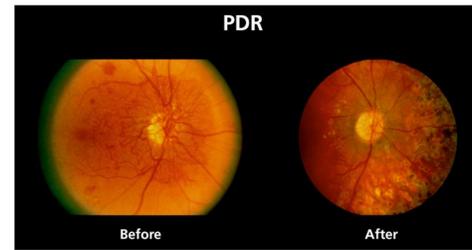
Laser Photocoagulation for Proliferative Retinopathy

SLIDE

46

Panretinal photocoagulation involves the widespread, peripheral administration of laser, causing extensive peripheral damage while preserving the posterior pole and macula. In a manner not completely understood, this technique causes the proliferating vessels to regress. Research indicates that the ischemic retina elaborates vasoproliferative factors, the most important of which appears to be VEGF. Photocoagulative damage of the ischemic retina eliminates the source of VEGF and other growth factors, causing vascular proliferations to regress.

Treatment



SLIDE

47

Performed as an outpatient procedure, a complete panretinal photocoagulation treatment consists of about 1000 to 2000 burns or more, usually delivered in one to three sessions. A single session usually takes less than 30 minutes. Most patients are given only topical anesthesia; those who have pain may require retrobulbar anesthesia.

Treatment

PANRETINAL PHOTOCOAGULATION (PRP)

- Outpatient procedure
- Approximately 1000 to 2000 burns per session
- 1 to 3 sessions

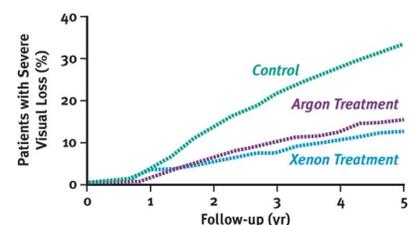
SLIDE

48

How effective is panretinal photocoagulation? The Diabetic Retinopathy Study, sponsored by the National Eye Institute, investigated the use of this technique. 50% or greater reduction in vision loss was noted with PRP. Those patients with high risk characteristics sustained the greatest benefit: the 2-year risk of severe visual loss, defined as visual acuity of 5/200 or worse, decreased from 26% to 11%; after 5 years there was a reduction from 50% to 26%.

Treatment

PRP: EFFECTIVENESS



SLIDE

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Expected consequences of panretinal photocoagulation include partial loss of side vision and night vision. This loss results from the destruction of portions of the retinal periphery, where the rods are most concentrated. Occasionally, central vision may be decreased by more than one or two lines on the Snellen visual acuity chart.

Treatment

PRP: SIDE EFFECTS

- Decreased night vision
- Decreased peripheral vision

Vitrectomy

SLIDE

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For patients whose retinal complications are not amenable to photocoagulation surgery, vitrectomy (removal of the vitreous) is a treatment option to evacuate vitreous hemorrhage, repair retinal detachment, and allow treatment with panretinal photocoagulation.

Treatment

VITRECTOMY

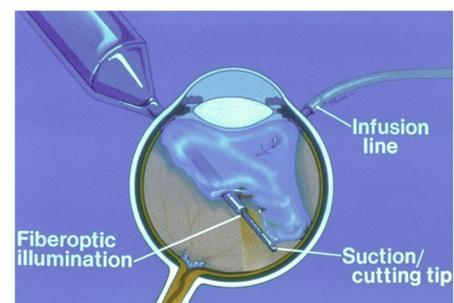
- Remove vitreous hemorrhage
- Repair retinal detachment
- Allow treatment with PRP

SLIDE

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Vitrectomy is a complex procedure in which the vitreous is simultaneously removed and replaced by an infusion of a modified saline solution. An intraocular fiberoptic light source allows visualization. If necessary, tractional fibrous bands are dissected and released. Intraocular laser photocoagulation, called endophotocoagulation, may also be performed at the time of surgery to expedite regression of new retinal vessels.

Treatment

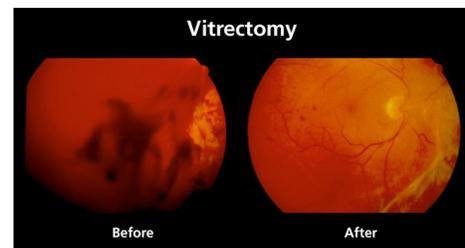


SLIDE

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The figure on the left shows an eye with a dense vitreous hemorrhage and light-perception vision. Vision is restored with removal of the blood by vitrectomy (figure on the right). The 1988 report of the Diabetic Retinopathy Vitrectomy Study documented a better visual outcome in patients with type 1 diabetes with PDR and vitreous hemorrhage who received early vitrectomy. Vitrectomy, however, is not a substitute for panretinal photocoagulation, which is essential in decreasing the ischemic drive.

Treatment



Treatment Options: Summary

SLIDE

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In summary, treatment options for diabetic eye disease include laser photocoagulation surgery for clinically significant macular edema and proliferative diabetic retinopathy, and vitrectomy for vitreous hemorrhage and retinal detachment.

Treatment

TREATMENT OPTIONS: SUMMARY

- Laser photocoagulation surgery
 - Focal macular laser for CSME
 - Panretinal photocoagulation for PDR
- Vitrectomy
 - May be necessary for vitreous hemorrhage or retinal detachment

SLIDE

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Inhibitors of angiogenesis, such as anti-VEGF therapy, are being used for the treatment of some retinal vascular disorders. In the future they may be found useful as an adjuvant treatment to laser therapy in patients with diabetic macular edema or proliferative diabetic retinopathy.

Treatment

FUTURE THERAPIES

- Anti-VEGF agents decrease capillary permeability and angiogenesis
- May prove useful as adjuvant treatment to laser therapy for diabetic retinopathies

SCREENING GUIDELINES

SLIDE

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Based on current knowledge of the natural history of diabetic retinopathy and the efficacy of treatment, the following screening guidelines are suggested:

1. Patients with type 1 diabetes should be evaluated annually by an ophthalmologist, beginning 5 years after diagnosis and not before age 12. Earlier evaluation is not necessary because vision-threatening retinopathy tends not to occur before puberty and is rare before 5 years' duration of the disease.

SLIDE

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2. Patients with type 2 diabetes should be evaluated by an ophthalmologist at the time of the initial diagnosis. These patients must be evaluated sooner than earlier-onset patients because the condition has generally been present before the time that it is first diagnosed. A small number of such patients already have macular edema or proliferative retinopathy at the time of diagnosis. After the initial screening examination, patients should be examined yearly or more frequently if the severity of the retinopathy warrants.

Screening Guidelines

SCREENING GUIDELINES: PATIENTS WITH TYPE 1 DIABETES

- Annual ophthalmologic exams starting 5 years after diagnosis and not before puberty

Screening Guidelines

PATIENTS WITH TYPE 2 DIABETES

- Annual ophthalmologic exams starting at time of Dx

Hormonal and blood flow changes during pregnancy may aggravate glucose control and retinopathy. Ideally, women with diabetes who are planning a pregnancy should be examined before conception to obtain a baseline assessment of the retina and to discuss the risks of developing or exacerbating retinopathy. First trimester screening by an ophthalmologist is the typical protocol. The subsequent interval for examination will depend on the grade of baseline retinopathy. Patients with gestational diabetes do not need to be examined.

DIABETES AND PREGNANCY

- Ophthalmologic exam before conception
- Ophthalmologic exam during first trimester
- Follow-up depends on baseline grade

CONCLUSION

Improving patient's access to and compliance with ocular examination is a priority for continued progress in the treatment of diabetic retinopathy. The WESDR showed that more than one third (36%) of patients with diabetes had missed their yearly dilated examination, and more than 60% of patients with high-risk proliferative diabetic retinopathy still had not received laser surgery 14 years later.

WESDR: PATIENTS' ACCESS AND COMPLIANCE

- 36% missed annual ocular exam
- 60% missed laser surgery

Timely ocular examination can decrease the risk of blindness from 50% to just 5% in those who develop proliferative diabetic retinopathy. Moreover, the financial benefits of improved access to examinations are enormous. Each 1% increase in screening of the diabetic population saves approximately \$1.7 million. It has been estimated that annual savings would reach \$167 million with 100% screening.

GOALS FOR SUCCESS

- Timely screening reduces risk of blindness from 50% to 5%
- 100% screening estimated to save \$167 million annually

SLIDE

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Optimal systemic control of glycosylated hemoglobin, blood pressure, kidney status, and serum lipid profile is crucial in improving the systemic and ocular prognoses of diabetic patients.

SLIDE

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The proper management of diabetic retinopathy requires a multidisciplinary approach. Ideally, the primary care physician, nutritionist, endocrinologist, nephrologist, and ophthalmologist should all work together to ensure the diabetic patient's access to eye care, optimize systemic control, and, ultimately, help reduce the risk of blindness.

Conclusion

GOALS FOR SUCCESS

Better systemic control of:

- Hemoglobin A_{1c}
- BP
- Kidney status
- Serum lipids

Conclusion

REDUCING THE RISK OF BLINDNESS

- Team approach: primary care physician, ophthalmologist, nutritionist, endocrinologist, nephrologist
- Access to eye care
- Systemic control

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APPENDIX 1

Recommended Eye Examination Schedule for Patients with Diabetes

Age at Onset of Diabetes Mellitus	Recommended Time of First Exam	Recommended Follow-up*
0-29 [†]	5 years after onset ¹	Yearly ¹
30 and older [†]	At time of diagnosis ²	Yearly ²
Prior to pregnancy	Prior to conception or early in the first trimester ^{3,4}	No retinopathy to nonsevere NPDR: every 3–12 months. Other stages of diabetic retinopathy: every 1–3 months.

* Abnormal findings may dictate more frequent follow-up examinations.

[†] As indicated in Wisconsin Epidemiologic Study of Diabetic Retinopathy, these are operational definitions of type 1 and type 2 diabetes based on age (age <30 years at diagnosis, type 1; age ≥30 years at diagnosis, type 2) and not pathogenetic classification.

These recommendations are based on level I evidence (randomized clinical trials). Table excerpted from: *Diabetic Retinopathy*. Preferred Practice Pattern. San Francisco: American Academy of Ophthalmology; 1998.

Notes

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3. Klein R, Klein BEK, Klein R. Effect of pregnancy on progression of diabetic retinopathy. *Diabetes Care* 1990;13:34-40.
4. Chew EY, Mills JL, Metzger BE, et al. Metabolic control and progression or retinopathy. The Diabetes in Early Pregnancy Study. *Diabetes Care* 1995; 18:631-637.

APPENDIX 2

Resources

Selected Academy Resources

For Practitioners

Basic and Clinical Science Course, Section 12: *Retina and Vitreous* (updated annually).

Bradford CA, ed: *Basic Ophthalmology for Medical Students and Primary Care Residents*. 8th ed. 2004. Material on diabetes and diabetic retinopathy appears in chapter 8.

Diabetic Retinopathy (Preferred Practice Pattern). 2003. A compact summary of treatment guidelines for ophthalmologists; useful as a reference for the nonophthalmologist physician.

DRSR Group, ETDRSR Group: *Management of Diabetic Retinopathy for the Primary Care Physician* (Videotape). 1999, 20 minutes. Covers the pathogenesis and stages of diabetic retinopathy and discusses treatment, follow-up, and visual prognosis.

ETDRSR Group: *Photocoagulation for Diabetic Macular Edema* (Videotape). 1999, 30 minutes. Reviews the findings on 4000 patients followed over a 5-year period and demonstrates photocoagulation techniques. Includes a section covering the DCCT.

Flynn HW Jr, Smiddy WE: *Diabetes and Ocular Disease*. Ophthalmology Monograph 14, 2000.

Ophthalmic Technology Assessment: Single Field Fundus Photography for Diabetic Retinopathy Screening. 2004. Six-page assessment of new and existing procedures, drugs, and diagnostic and screening tests for safety and clinical effectiveness.

Trobe JD: *The Physician's Guide to Eye Care*. 2nd ed. 2001. Material on diabetes and diabetic retinopathy appears in chapters 1, 2, 6 and 9.

For Patients and the General Public

Diabetic Retinopathy Brochure. An easy-to-understand brochure available in English and Spanish

Diabetic Retinopathy. Patient Education Booklet. Explains how diabetes affects vision, methods of diagnosis and treatment, and the importance of regular ophthalmic exams.

Diabetic Retinopathy (Videotape). 1999, 10 minutes. Discusses the stages of diabetic retinopathy and methods of treatment.

EyeCare America Diabetes Project. Referral program for those 65 and older; for more information, patients may call 800/222-EYES.

Fluorescein Angiography (Eye Fact Sheet). A single page that explains the purpose and procedure of fluorescein angiography.

Laser Surgery of the Eye. A brochure that helps patients understand laser surgery; available in English and Spanish.

Additional Resources

Publications

Friberg TR, Rosenstock J, Sanborn G, et al: The effect of long-term near normal glycemic control on mild diabetic retinopathy. *Ophthalmology* 1985;92:1051-1058.

Klein R, Klein BEK, Moss SE, et al: The Wisconsin epidemiologic study of diabetic retinopathy, II: prevalence and risk of diabetic retinopathy when age at diagnosis is less than 30 years. *Arch Ophthalmol* 1984a;102:520-526.

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Online Resources

American Academy of Ophthalmology
www.aao.org

American Diabetes Association
www.diabetes.org

Diabetic Retinopathy Studies
<http://www.nei.nih.gov>