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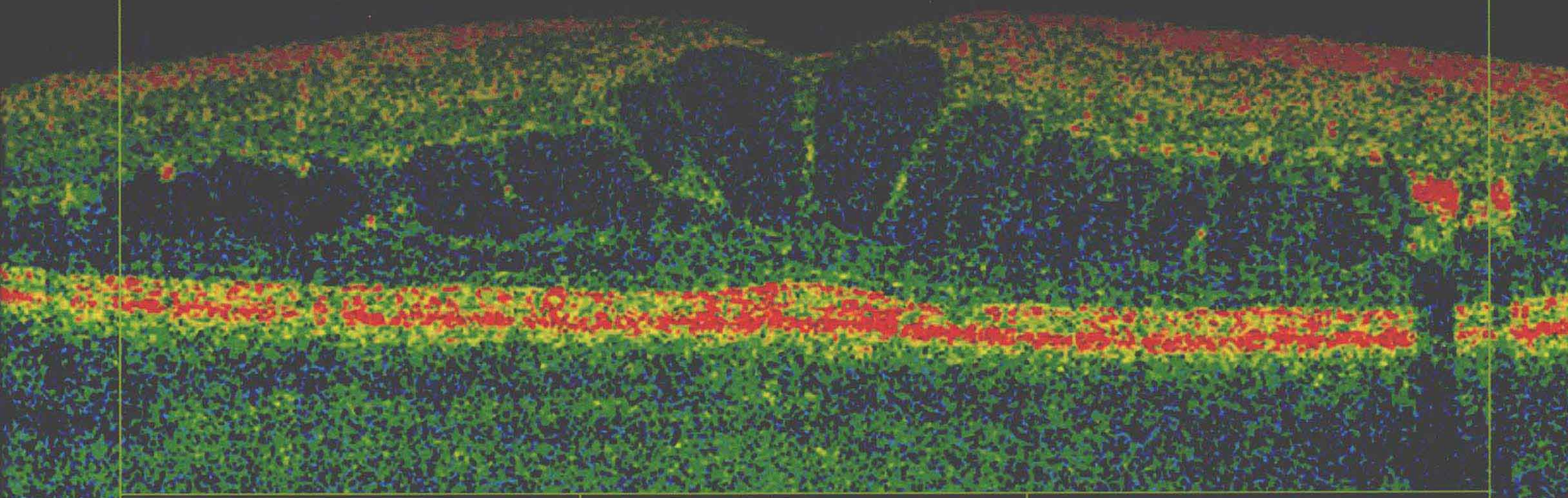
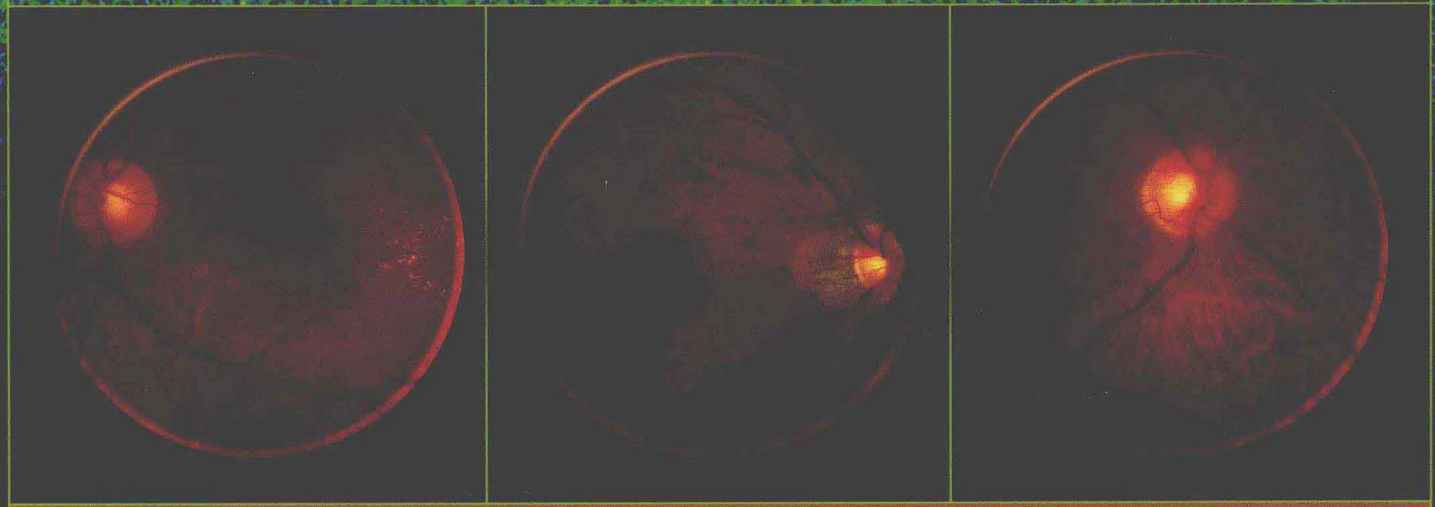
Diabetic Retinopathy

The Essentials





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Diabetic Retinopathy: *The Essentials*

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Diabetic Retinopathy: *The Essentials*

PREFACE

In 2008, the World Health Organization estimated that more than 180 million people worldwide had diabetes and that by 2030, this number would double. Thus, diabetes has become epidemic in the world at large. China and India both have increasing rates of diabetes in their populations. Thus, diabetic retinopathy has become a more common problem in physicians' offices. In California, where our patients come from all over the globe, we have seen diabetic retinopathy in all stages of severity. In fact, many of the patients present with decreased vision and diabetic retinopathy but do not yet know that they have diabetes since they have never had the appropriate laboratory testing.

I wrote this book for the ophthalmology resident-in-training, the general ophthalmologist or optometrist, the family practitioner, the diabetologist, and the general internist. I wanted to answer their questions about diabetic retinopathy.

The first three chapters deal with the background of diabetes and diabetic retinopathy. These chapters serve to bridge our background as retinal specialists to our colleagues'. Chapter 4 describes our diagnostic testing and what we, as retinal specialists, use for the examination of a diabetic patient. Chapter 5 specifically goes through our ophthalmic examination using the slit lamp. Chapter 6 summarizes the new treatment modalities of this past decade including the anti-VEGF treatments. Chapters 7 through 9 have clinical correlations with photographs and case reports. These chapters would be useful for all clinicians, especially ophthalmologists and optometrists. The photographs would be interesting for our internal medicine and family practice colleagues. Chapter 10 is a summary of surgical techniques for diabetic retinopathy and vitreous hemorrhages. This might be useful for our internal medicine colleagues. Chapter 11 is for our colleagues who see pregnant diabetic patients. Chapter 12 is about complications of medical and surgical treatment. Chapter 13 is about diabetes and neovascular glaucoma, a common complication of diabetes. Chapter 14 is about informed consent and how it applies to all of us as clinicians. In this chapter, cross-cultural concerns are discussed. Chapter 15 gives guidelines for nurses and office assistants. In this chapter, specific telephone scripts addressing cross-cultural considerations are given. Appendix A is entirely devoted to the visual image of diabetic retinopathy as can be seen in a modern retina-vitreous practice. There are scans using Optical Coherent Tomography to show the retina in cross section. Appendix B lists medical equipment companies and supplies. This appendix also provides web addresses for patient information. This is useful to all clinicians who want to provide reading materials for their patients. Appendix C is a short diabetic cookbook which can be shared with our patients.

I hope that this book proves to be useful. I look forward to your emails if you have additional questions.

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I am especially indebted to my teachers in ophthalmology and retina. Beginning with Dr. Myles Behrens, who taught me the importance of research and inquiry when I was a fourth-year medical student at Columbia University College of Physicians and Surgeons, with whom I wrote my first paper, a case report, "Hyphema in the Newborn." Dr. D. Jackson Coleman, Dr. Harvey Lincoff, and Dr. Stanley Chang were outstanding role models and teachers to me. Dr. Coleman taught me ultrasound along with the art of medicine. It was with Dr. Lincoff that I found my first retinal hole. Dr. Chang and his fellows taught by example: in the operating rooms of Cornell University Medical Center, the techniques of vitreous surgery leading to the complex reattachment of diabetic traction detachments and proliferative vitreoretinopathy. To Dr. Charles Schepens, Dr. J. Wallace McMeel, Dr. Hal Freeman, Dr. Clement Trempe, Dr. Ronald Pruett, Dr. Felipe Tolentino, Dr. Tatsuo Hirose, Dr. John Weiter, Dr. Sheldon Buzney, and Dr. Alexandre Jalkh, I say earnest and sincere thanks for the transformative role that they played in my professional life. In my fellowship days at the Massachusetts Eye and Ear Infirmary, I had the opportunity to learn from Dr. Evangelos Gragoudas and his fluorescein conferences and from Dr. Claes Dohlman in his grand rounds for the residents and fellows. My formative years in Boston were made memorable by my time at the Joslin Diabetes Center, under the skillful and watchful guidance of Dr. Lloyd M. Aiello and Dr. Lawrence I. Rand. The Joslin Diabetes Center and its approach to diabetes have informed my practice patterns to this day. I thank Dr. Joseph Googe, Dr. Lory Snady-McCoy, Dr. Aaron Appiah, Dr. Sal Melgen, Dr. Jerry Sebag, and Dr. Susan Elner, my co-fellows at the Schepens Retina Associates; and Dr. David Lee, Dr. Joan Miller, Dr. Joan O'Brien, Dr. Robert Bhisitkul, and Dr. Lucy Young, my fellow residents and fellows at the Massachusetts Eye and Ear Infirmary, for their friendship for the past 20 years. A special word of thanks goes to Dr. Jay Duker who has supported my professional collaboration with Tufts University School of Medicine.

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CONTENTS

CHAPTER 1

Epidemiology

SCOPE OF THE PROBLEM

In 2007, the Center for Disease Control in the United States estimated that diabetes affects approximately 23.6 million children and adults or 8% of the US population. An estimated one third is unaware that they have the disease. An additional 57 million people who are prediabetic will become diabetic if they do not change their eating habits.[1] Diabetes is the fifth most common cause of death in America. In one study, 18% of the hospital admissions were found to have unrecognized and probable diabetes by the criteria of elevated HbA1c (>6.1).[2]

From 1990 to 2005, the prevalence of diabetes increased from 4.5 to 26.4 per 1,000 people, a sixfold increase. Researchers postulated that this increase was due to the increase in obesity during 1990–2005.[3] One study shows that currently 4% of obese white adolescents have diabetes and an additional 21% have abnormal glucose tolerance.[4]

In 2007, the economic cost of diabetes was estimated to be $174 billion.[1] Health care and medical expenditures related to diabetes totaled $116 billion (Table 1-1). Of that amount, $27 billion was for diabetes care, $31 billion for extra general medical costs, and $58 billion for chronic diabetic complications. People with diabetes had medical expenses that were 2.3 times higher than those for people without diabetes. Diagnosed diabetic patients accounted for 5.8% of the total US population. Inpatient hospital care accounted for $58.3 billion. For physician office visits, $9.9 billion was spent in 2007. Diabetes accounted for 24.3 million days of hospitalizations in 2007, whereas in 2002, that figure was 16.9 million days. In 2007, average cost for a hospital inpatient day due to diabetes was $1853 and $2281 due to diabetes-related chronic complications including neurological, peripheral-vascular, cardiovascular, renal, metabolic, and ophthalmic complications.

The indirect costs were estimated to $58 billion in 2007.[1] In 2007, diabetes accounted for 15 million work days of absence, 120 million work days with reduced performance, 6 million reduced productivity days for those not in the workforce, and an additional 107 million work days lost due to unemployment disability attributed to diabetes. Diabetes costs 445,000 cases of unemployment disability in 2007. The value of lost productivity due to premature death related to diabetes was $26.9 billion.

The increase in cost due to diabetes is related to (a) the growth of diabetes prevalence, (b) medical costs rising faster than general inflation, and (c) improvements made in the methods and data sources influencing cost estimates. The national burden of diabetes likely exceeds the $174 billion estimate because it omits the social cost of intangibles such as pain and suffering, care provided by nonpaid caregivers, excess medical costs associated with undiagnosed diabetes, and diabetes-attributed costs for health care expenditures categories not studied.[1]

Thus, the indirect cost of $58 billion dollars is from increased absenteeism, reduced productivity, disease-related unemployment disability, and loss of workplace productivity.[1] This $58 billion cost in 2007 eclipsed the $16 billion for the same problems in 2002. The reason for the increase is due to the obesity epidemic among teens. One out of every five health care dollars is spent caring for a diabetic patient, whereas 1 in 19 health care dollars is spent for the treatment of diabetes and its complications.[5]

TABLE 1.1 Cost of Diabetes ($174 Billion)

Medical cost $116 billion
$27 billion for diabetes care
$58 billion for chronic diabetes-related complications
$31 billion for related medical costs

Indirect cost $58 billion
15 million work days absent
120 million work days with reduced performance
6 million reduced productivity for those not in the workforce
107 million work days lost to unemployment disability
445,000 cases of unemployment disability

LIFETIME RISK FOR DIABETES MELLITUS IN THE UNITED STATES

Data from the National Health Interview Survey (1984–2000) show that the estimated lifetime risk of developing diabetes for individuals born in 2000 is 32.8% for males and 38.5% for females. Females have the higher residual lifetime risks for all age groups. The highest estimated lifetime risk for diabetes is among Hispanics (males 45.4% and females 52.5%). Individuals diagnosed as having diabetes have a large reduction in life expectancy. If a person is diagnosed at age 40, men will lose 11.6 life years and 18.6 quality-adjusted life years and women will lose 14.3 life years and 22.0 quality-adjusted life years. Thus, primary prevention of diabetes and its complications is an important public health priority.[6]

MEDICAL CARE FOR DIABETIC PATIENTS

In the 1989 National Health Interview Survey, 84,572 adults 18 years and older were studied. From this group, a subgroup of diabetic patients, 2,405 respondents, were queried about their diabetes care. More than 90% of the diabetic adults had one physician for the usual care of their diabetes, but 32% made fewer than four visits to the physician each year. Most physician visits by diabetic patients were not made to diabetes specialists. The visit rate to nonprimary care physicians such as other health care professionals such as ophthalmologists, podiatrists, nutritionists, was low. About half of the insulin-treated diabetic subjects used multiple daily insulin injections. Forty percent of patients with insulin-dependent diabetes and 26% with non–insulin-dependent diabetes mellitus (NIDDM) took insulin, and 5% of NIDDM patients not taking insulin monitored their blood glucose daily. Diabetes patient education classes were attended by 35% of diabetic adults. Thus, there is a need for specialists who treat diabetic patients.[7]

RACE AND ETHNIC DIFFERENCES

The association between impaired glucose tolerance (IGT) and the risk of Type 2 diabetes has been documented in a wide range of racial and ethnic groups (Table 1-2). In the San Antonio Heart Study, the relative risk of developing Type 2 diabetes conferred by IGT ranges from 4.3 to 7 depending upon race and gender.[8]

Native Americans

The 2005 Indian Health Service User population database indicated that 14.2% of American Indians and Alaskan Natives (AI/AN) aged 20 or older were diabetic. Rates varied from 6.0% in Alaska Native adults to 29.3% among American Indian adults in

TABLE 1.2 Clinical Interpretations of Plasma Glucose Concentrations

Fasting (mg/dL)
<100 within reference range
100–125 impaired fasting glucose/prediabetes mellitus
≥126 overt diabetes mellitus

2-hour post challenge load (75 g oral glucose tolerance test)
<140 within reference range
140–199 IGT
≥200 overt diabetes mellitus

Source: Diabetic Retinopathy Study Research Group. DRS report no. 3. Four risk factors for severe visual loss in diabetic retinopathy. *Arch Ophthalmol.* 1979;97:654–655.

Southern Arizona. In this study, diabetes occurred in 6.6% non-Hispanic whites, 7.5% Asian Americans, 10.4% Hispanics, and 11.8% non-Hispanic blacks.[9] In another study, American and Alaskan Native children (<15 years), adolescents (15–19 years), and young adults (20–34 years) had a 71% increase in diagnosis of diabetes. Diabetes prevalence increased by 46% from 1990 to 1998 in the above three groups.[10]

African Americans

In 1993, 1.3 million African Americans were known to have diabetes. This figure is almost three times the number of African Americans with diabetes in 1963. The actual number of diabetic African Americans is probably higher since there are many undiagnosed with the disease than reported. It is thought that for every African American diagnosed with diabetes, there is another African American yet to be diagnosed or who does not know that he or she has the disease. From 1980 to 2005, the age-adjusted prevalence of diagnosed diabetes doubled among black males and increased 69% among black females. However, of all groups observed, black females had the highest overall prevalence.[11]

After the 2000 census, it is known that 11% or 2.7 million of African Americans, aged 20 or older, have diabetes. One third of these do not even know that they have the disease. African Americans are 1.6 times more likely than white Americans to get diabetes.

One out of every four African American women is diabetic if they are 55 years or older. Twenty-five percent of blacks between the ages of 65 and 74 have diabetes.[12] Of note, African Americans are twice as likely to develop diabetic retinopathy than their white counterparts.[13]

In 1993, for the age group of 65 to 74 years, 17.4% of blacks were diabetic versus 9.5% of white Americans.[13] African Americans had a greater incidence of Type 2 diabetes. In fact, prevalence of Type 1 diabetes in white American children aged 15 and younger was nearly twice as that in African American children of the same age.[13] At age 45 and older, the prevalence of diabetes was 1.4 to 2.3 times as frequent in blacks as in whites. Within the age group of 65 to 74 years, 17.4% of blacks had diagnosed diabetes versus 9.5% of whites.

While gestational diabetes, which affects 2% to 5% of all pregnant women, usually resolves after childbirth, African American women have a higher rate of gestational diabetes. An Illinois study has shown an 80% higher incidence of gestational diabetes in black women as compared to their white counterparts. Experts estimate that about half of the women with gestational diabetes develop Type 2 diabetes within 20 years of pregnancy, regardless of race.[13]

African Americans may have a hemoglobin variant. Thus, the HbA1c of African Americans may not be accurate, falsely low or high, thus, it is important to check with the clinical laboratory to correlate the hemoglobin A1c with the patient's hemoglobin variant. African Americans have hemoglobin variants such as Hemoglobin S, Hemoglobin C, or Hemoglobin E.[14]

Latino Americans

Two in five Hispanics born in the year 2000 face a risk for diabetes. Compared to whites, Hispanics are more than two times as likely to have diabetes. From 1997 to 2005, the age-adjusted prevalence among Hispanics increased 16% among males and 21% among females.

In one study, the Hispanic population, which is the second largest and fastest growing minority in the United States, shares genetic markers with Americans, the Spanish, and Africans. The high frequency of Native American–derived genes in the contemporary Hispanic population predicts a higher frequency of NIDDM. The genetic markers, taken from 1,000 randomly selected Mexican Americans from Starr County, Texas, are used as a representative sample of the Mexican American population. For Mexican Americans, 31% of the contemporary gene pool is estimated to be Native American derived whereas 61% and 8% are Spanish and African derived, respectively. In Puerto Rico, the percentage of contributions of Spanish, Native American, and African mixture to the population are 15%, 18%, and 37%, respectively. In Cuba, the parallel estimates are 62%, 18%, and 20%. The high frequency of Native American-derived genes in contemporary Hispanic population predicts a higher frequency of NIDDM under the assumption that NIDDM may have genetic markers.[15]

Two million Latinos, aged 20 or older, have Type 1 or Type 2 diabetes. Latinos are 1.5 times more likely than non-Hispanic whites to have Type 2 diabetes, but Latinos are 2 times more likely than non-Hispanic whites to have any type of diabetes, Type 1 or Type 2. Specifically, Mexican Americans are 1.7 times more likely and Puerto Ricans are 1.8 times more likely to develop Type 2 diabetes than whites. Twenty-five percent to thirty percent of Hispanics older than age 50 have diabetes. Latinos are the fastest growing minority group in the United States. However, they have the lowest rates of insurance coverage and without regular and proper health, health care, and health care follow-up, diabetes can progress to blindness. Nearly half of the Latino children born in 2000 are likely to develop diabetes in their lifetime.[16]

Asian Americans

Diabetes is rising faster in Asian Americans than Caucasians. The rate among Asian Americans is 10% to 15% versus 6% to 8% in Caucasians.[9] These rates are similar to that found in Hong Kong.[16] The International Diabetes Federation predicts that diabetic rate in Asia are expected to rise to 160 million by 2025. India and China would account for 120 million by 2025.[16]

Top five countries with largest number of people affected by diabetes in 2003:

India—35.5 million
China—23.8 million
United States—16 million (by 2008, 21 to 23 million)
Russia—9.7 million
Japan—6.7 million

Ninety to ninety-five percent of Asians with diabetes have Type 2 diabetes. The rate of diabetes in Chinese Americans is notably higher than the rate of Chinese population in rural China. Thus, there are environmental factors that play a role in the development of diabetes in Chinese Americans in the United States. Indo-Asian women in America have the highest gestational diabetes rate in the country, with a prevalence of 56.1 per 100,000.[17] Native Hawaiians are 2.5 times likely to have diabetes than their white counterparts.[18] When Asians immigrate to the United States, their risk of developing diabetes increases significantly. This is most probably due to a change in lifestyle, diet, and exercise.[19]

Native Americans

AI/AN are people who have origins in any of the original peoples of North and South America including Central America and who maintain a tribal affiliation or community attachment. According to the 2000 US Census, those who identify as AI/AN constitute 0.9% of the US population or 2.5 million individuals. The greatest concentration of AI/AN populations are in the West, Southwest, and Midwest, especially in Alaska, Arizona,

TABLE 1.3 Risk Factors for Prediabetes and Diabetes Mellitus

Family history
Cardiovascular disease
Overweight or obese state
Sedentary lifestyle
Latino/Hispanic, non-Hispanic black, Asian American, Native American, or Pacific Islander ethnicity
Previously identified IGT or impaired fasting glucose
Hypertension
Increased levels of triglycerides, low concentrations of high-density lipoprotein cholesterol or both
History of gestational diabetes
History of delivery of infant with a birth weight greater than 9 lb
Polycystic ovary syndrome

Montana, New Mexico, Oklahoma, and South Dakota. There are 569 federally recognized AI/AN tribes, plus an unknown number of tribes that are not federally recognized. Each tribe has its own culture, beliefs, and practices. Among people younger than age 20, American Indians aged 10 to 19 years have the highest prevalence of Type 2 diabetes.[20] Gestational diabetes occurs more frequently among American Indians who are thought to have a high incidence of obesity during pregnancy. The risk factors for gestational diabetes include obesity and women with a family history of diabetes (Table 1-3).

TEEN OBESITY

In 2002–2003, SEARCH for Diabetes in Youth Study reported that 15,000 US children under the age of 20 years are diagnosed annually with Type 1 diabetes and 37,000 are newly diagnosed with Type 2 diabetes. While Type 2 diabetes is rare in children younger than 10 years of age, regardless of race and ethnicity, it becomes increasingly common in minority groups older than 10 years of age:

14.9% of new diabetes in Caucasians (non-Hispanic white)
46.1% Hispanic Youth
57.8% African Americans
69.7% in Asian and Pacific Islanders
86.2% in American Indian Youth

Results from the 2005–2006 National Health and Nutrition Examination Survey, using measured heights and weights, indicate that an estimated 16% to 17% of children and adolescents aged 2 to 19 had a BMI greater than or equal to 95th percentile of the age-and sex-specific BMI, about double the number of two decades ago.[21] Obesity in youth contributes to the increasing numbers of young people with Type 2 diabetes.[22]

GENETICS

Researchers suggest that African Americans and recent African immigrants to America have inherited a "thrifty" gene from their African ancestors. The presence of this gene has enabled Africans during "feast and famine" cycles to use food energy more efficiently when food was scarce. Today, with fewer "feast and famine" cycles, the thrifty gene that developed for survival may instead make weight control more difficult. This genetic predisposition with IGT occurs together with the genetic tendency of high blood pressure.[23]

EPIDEMIOLOGY OF DIABETIC RETINOPATHY

Diabetic retinopathy is the leading cause of blindness in working-age Americans, between 21 and 64 years of age. Diabetic retinopathy affects up to 16 million Americans, contributing to 14% of new blindness cases each year. It is estimated that 85% of the diabetics

TABLE 1.4 Eye Examination Schedule

Time of onset of DM	Recommended time of first exam	Routine minimum follow-up
≤30 y of age	5 y after onset	Annually
>30 y	At time of dx	Annually
Before pregnancy	Before or soon after conception	At least q 3 months

do not know that they have diabetic retinopathy. Twenty thousand people become legally blind each year from diabetes.

When diabetic retinopathy is appropriately handled, the 5-year risk of blindness for patients with proliferative diabetic retinopathy is reduced by 90% and the risk of visual loss from macular edema is reduced by 50%.[24] However, only 50% of diabetic patients receive regular dilated fundus examinations and many patients become legally blind without treatment.[25,26] The value of screening eye examinations has been known[27] (Table 1-4). The follow-up of patients with diabetic retinopathy depends on their presenting status of retinopathy (Table 1-5).

Diabetic retinopathy, a potential cause of irreversible vision loss, can cause a decrease in workplace productivity and a loss of income and productivity. Unfortunately, many diabetic patients are not aware of the possibility of early diagnosis and intervention which could save a lifetime of visual impairment.

Approximately 500,000 people in America have macular edema, which can be a harbinger of future acceleration of diabetic retinopathy. Seven hundred thousand Americans have proliferative diabetic retinopathy, and 65,000 new cases of proliferative diabetic retinopathy occur each year.

In one study evaluating diabetic retinopathy using a nonmydriatic fundus camera, the authors find that prevalence of any diabetic retinopathy in people with diagnosed diabetes is 46% higher in non-Hispanic blacks and 84% higher in Mexican Americans, compared with non-Hispanic whites. Blacks and Mexican Americans have higher rates of moderate and severe retinopathy and high levels of the risk factors of retinopathy. However, blacks have lower diabetic retinopathy prevalence among those with undiagnosed diabetes. Retinopathy in those with diagnosed diabetes is associated with diabetes severity, for example, duration of diabetes, severity, HbA1c, treatment with insulin and oral agents, and systolic blood pressure. Despite adjustment of these factors, the risk of retinopathy in Mexican Americans is twice that of non-Hispanic whites. Non-Hispanic blacks are not at a higher risk for retinopathy. The excess risk in Mexican Americans is unexplained by these researchers.[28]

NONPROLIFERATIVE DIABETIC RETINOPATHY: BACKGROUND DIABETIC RETINOPATHY

Nonproliferative Diabetic Retinopathy (NPDR) is a common yet underdiagnosed form of diabetic retinopathy. It is also known in the nonophthalmic, medical literature as "background diabetic retinopathy." The earliest form of NPDR is the appearance of microaneurysms or dot blot hemorrhages. These are outpouchings of the vessel walls and can be

TABLE 1.5 Follow-up Schedule

Recommended follow-up schedule	Follow-up in months
NDR or microaneurysms only	12
Mild/mod NPDR without DME	6–12
Mild/mod NPDR, + macular edema but not clinically significant	4–6
Mild/mod NPDR, + CSME	3–4
Severe/very severe NPDR	3–4

TABLE 1.6 Signs and Symptoms of Diabetic Retinopathy

Symptoms	Signs
Floaters, black lines Cobwebs	Vitreous hemorrhage
Loss of vision	Vitreous hemorrhage or retinal detachment
Loss of near vision	Macular edema
Transient loss of vision	TIA, emboli, vitreous hemorrhage, hypoglycemia, or hyperglycemia

50 μm in diameter or 100 μm in diameter. These are best seen upon slit lamp biomicroscopy with a 90 D or 60 D lens. Or, they can be photographed with a fundus camera. These abnormalities usually do not involve the macula and thus, there are no visual symptoms (Table 1-6).

CLINICAL CORRELATIONS

Patient complaints: Routine exam or mild visual loss on near vision or Amsler grid changes. Some patients might notice a smudge in central vision at near. Patients may present with the chief complaint of "I see a smudge when I read my book or when I use the computer."

Or suddenly, "I feel that I see better and do not need my glasses," denoting new diabetic cataracts from a recent diagnosis of diabetes.

Caveats: Many patients do not seek ophthalmic care or optometric care unless they experience a loss of vision. There may be a lack of health literacy and health education about diabetes. These patients do not understand the need for a dilated funduscopic examination yearly if they have diabetes.

FIGURE 1-1. Background diabetic retinopathy.

Case Study (Fig. 1-1)

9–23–08: EG, is a 43-year-old Filipino male who presented with a new diagnosis with Type 2 diabetes and was referred to our office for a baseline evaluation.

Laboratory evaluation: HbA1c = 6.5%, Fasting blood sugar = 128 mg/dL, Cholesterol = 255 mg/dL (nL = 240), Triglycerides = 251 mg/dL (nL = 150).

V 20/15
20/15
Slit lamp examination
Cornea: Clear OU
Anterior chamber: D-deep and quiet OU
Iris: Normal, no rubeosis OU
Lens: Clear OU
Applanation tonometry = 18/18 mm Hg (2:30 PM)
Gonioscopy: No rubeosis OU for 360 degrees, angles open for 360 degrees OU.

Dilated funduscopic examination:

Right eye: Small hard exudates found in the macular region, 1:00 from the foveal avascular zone.

Left eye: No microaneurysms seen in the macular region. However, with Goldmann three-mirror examination, there are a few microaneurysms seen in the far equatorial region.

TABLE 1.7 "Moderate-to-Severe" Nonproliferative Retinopathy

Cotton-wool spots
Intraretinal hemorrhages: mild to moderate in four quadrants
Venous beading
IRMA

MODERATE NONPROLIFERATIVE DIABETIC RETINOPATHY

The ophthalmologist should look for venous beading and intraretinal microvascular abnormalities (IRMA) in the equator to the periphery. Cotton-wool spots are also present in these cases. These can be missed by the nonophthalmologist because of the difficulty in examining the fundus in some patients or the lack of seven standard photographic fields (Table 1-7).

Cotton-wool spots or areas of retinal ischemia can occur outside of the arcades, outside of the macula. Once cotton-wool spots are seen, the patient is in the moderate to severe category of NPDR. With increasing number of the cotton-wool spots and increasing involvement of the number of photographic fields, the patient may fall into the severe category of NPDR. If no photographic fields are available, the patient can be categorized by examining the optic nerve area, macula, temporal to the macula, superotemporal area, inferotemporal area, superonasal area, and inferonasal area to assess the involvement of the number of cotton-wool spots, venous beading, and venous abnormalities.

CLINICAL CORRELATIONS

Patient complaints: Some decrease in vision is noted on physical examination, usually 20/25 visual acuity or worse vision. Patients have more vague and more frequent complaints of vision loss.

Caveats: Is there loss to medical follow-up because of inadequate health literacy or denial? The patient may have cross-cultural needs in that his or her diabetic diet has to be tailored to his or her preferred diet patterns.

Case Study (Fig. 1-2)

4–10–09: DC, a 61-year-old unemployed male engineer, presents to our office for decreased vision for six months. He was last seen by a physician 10 years earlier and was lost to follow-up. He was referred by a local optometrist who noted decreased vision. At the time, he brought no laboratory records with him.

Laboratory evaluation: HbA1c = 12%, Fasting blood sugar = 256 mg/dL, Cholesterol 400 mg/dL (nL = 200), Triglycerides 399 mg/dL (nL = 150).

V 20/30
20/60
Slit lamp examination
Cornea: Clear OU
Anterior chamber: Deep and quiet OU
Iris: No rubeosis OU

FIGURE 1-2. Macular edema and nonproliferative diabetic retinopathy.

Lens: Trace nuclear sclerosis OU
Applanation tonometry = 20/20 mm Hg (9:00 AM)
Gonioscopy: No rubeosis in the angle for 360 degrees.

Dilated funduscopic examination:
Right eye: Clinically significant macular edema seen on slit lamp biomicroscopy, large dot blot hemorrhages, and cotton-wool spots seen.
Left eye: Clinically significant macular edema seen on slit lamp biomicroscopy, cotton-wool spots, and large dot blot hemorrhages.
Fluorescein angiography: Macular edema OU

Comment
In summary, the patient, DC, has poorly controlled diabetes and he needs endocrinological follow-up and ophthalmic follow-up. His diagnosis is severe NPDR.

MACULAR EDEMA

Macular edema is one of the insidious manifestations of diabetic retinopathy which can be as subtle as one or two hard exudates in the macula to full-fledged hard exudate ring encompassing part of the macula and the fovea centralis region. Or it can present as a diffuse elevation of the inner retina filled with small cysts seen on slit lamp biomicroscopy. These diagnoses are easily made with optical coherence tomography (OCT), where the architecture of the retina and macula is well delineated.

Macular edema, which can exist with NPDR or proliferative diabetic retinopathy, involves the macula with swelling within the 10 layers of the retina. Macular edema has been defined by the landmark study, the Early Treatment Diabetic Retinopathy Study.[29] This study has defined for the retina-vitreous community the standard of care for the treatment of macular edema and diabetic retinopathy. For the past 20 years, early treatment of diabetic retinopathy denotes early laser when the clinician discovered macular edema seen on slit lamp biomicroscopy; now it has evolved to include macular edema as diagnosed by fluorescein angiography and or by ocular coherence tomography. Fast Fourier transform analysis of OCT, or spectral domain OCT, has changed the way that many retinal specialists have viewed macular edema. Thus, these new diagnostic tools have made it easier for clinicians to diagnose macular edema and initiate early treatment and maintain good vision for the patients.

CLINICAL CORRELATIONS

Patient complaints: Decrease in vision is noted on physical examination. Usually, 20/25 to 20/40 visual acuity or worse vision. The patient is symptomatic at near: reading books, doing close work, and decreased vision at the computer are the most common complaints. The patient may see a crooked line where the newspaper print may be distorted.

Caveats: Poor internal medicine or endocrinologic follow-up. Alcohol intake can add to the patient's daily calorie intake. The patient is a physician and he may be in denial of his disease.

Case Study (Fig. 1-3)

6–5–2005: Dr. AP is a 66-year-old Filipino male, practicing anesthesiologist, who presents with poor vision and a history of poor glycemic control. He denies hypertension. He has had insulin-dependent diabetes for the past 8 years. According to the patient, his HbA1c = 9%.

V 20/40
20/100
Slit lamp examination
Cornea: Clear OU
Anterior chamber: Deep and quiet
Iris: No rubeosis OU
Lens: 2 + nuclear sclerosis OU
Applanation tonometry = 18/18 mm Hg (3 PM)
Gonioscopy: No rubeosis 360 OU
Pigmented trabecular meshwork OU but open to ciliary body OU

FIGURE 1-3. Macular edema and severe nonproliferative diabetic retinopathy.

Dilated fundus examination:
Right eye: Clinically significant macular edema, dot blot hemorrhages
Left eye: Clinically significant macular edema, dot blot hemorrhages

Comment:
Of note, this patient is noncompliant, is a physician and colleague. His management hinges on better glycemic control, laser for his clinically significant macular edema, and long-term ophthalmologic follow-up. The patient and his family need to be part of this sensitive discussion that glycemic control is important. The nurse in the office should provide American Diabetes Association pamphlets to the family. These noncompliant patients who need precise vision to maintain their livelihood are difficult to manage over the long term. It is best to involve an endocrinologist in addition to the internist who will reinforce the need for glycemic control.

PROLIFERATIVE DIABETIC RETINOPATHY

Approximately 700,000 Americans have proliferative diabetic retinopathy and 500,000 have macular edema. Approximately 65,000 new cases of proliferative diabetic retinopathy occur each year.

Proliferative diabetic retinopathy is the most severe form of diabetic eye disease affecting the retina. There is growth of new vessels from areas of the retina that are hypoxic from damage from hyperglycemia and cell death. The growth of new vessels lends itself to the name of "proliferation" of vascular growth. These new abnormal vessels bleed easily. With the bleeding, there is associated loss of vision, which may be abrupt or gradual. Treatment is laser, called panretinal photocoagulation or in severe cases, vitrectomy. In addition, in this severe form of diabetic retinopathy, traction retinal detachment can occur and in these instances, vitrectomy is done very quickly after diagnosis.

Testing involves ultrasound if there is no view of the retina landmarks, such as optic nerve or the retinal vessels. In addition, ERG and VER testing can be performed to evaluate test function and for prognosis for vision postoperatively. For proliferative diabetic retinopathy where there are new fronds of vessels but the vision is still preserved since there is no frank vitreous hemorrhage, laser photocoagulation is performed.