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Interdisciplinary Management of Diabetic Eye Disease: A Global Approach to Care

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Abstract
Diabetic eye disease is a leading cause of acquired blindness in the United States. Most cases of blindness secondary to diabetes mellitus are preventable. In addition to exercise, proper diet, and aggressive glycemic control, patients with diabetes mellitus should be educated to adhere to established guidelines for an annual dilated retinal evaluation. The ideal model of care for patients with diabetic eye disease is an interdisciplinary, team-oriented approach with the patient as the central member of the healthcare team. The primary purpose of this paper is to present an interdisciplinary approach to management of the ocular complications of diabetes mellitus and to educate clinicians about diabetic eye disease.

Introduction

Diabetes Mellitus (DM) is a group of metabolic diseases characterized by hyperglycemia resulting from defects of insulin secretion and/or increased cellular resistance to insulin. Chronic hyperglycemia and other metabolic disturbances lead to long-term tissue and organ damage involving the eyes, kidneys, nervous, and vascular systems.[1] Classified as a "chronic disease epidemic" by the Centers for Disease Control and Prevention, the prevalence of DM has increased dramatically over the past forty years.[2] This trend is especially significant among minority populations and high-risk ethnic groups (Native Americans, Hispanic Americans, African Americans, and Asian Americans).[3]

Type 1 DM accounts for approximately 10% of all patients with DM in the United States. Although it can occur at any age, Type 1 DM is more common in those under 30 and replaces the term "insulin dependent diabetes mellitus." Type 1 DM is a disease of fat, carbohydrate, and protein catabolism caused by a lack of circulating insulin. Patients require exogenous insulin to reverse this metabolic abnormality, prevent ketosis, and decrease hyperglucagonemia. Type 1 DM is thought to be an autoimmune disease with specific organ involvement; it results from destruction of insulin-producing beta cells in the pancreas.[4,5] Examination of islet tissue obtained from pancreatic biopsy from patients with recent onset Type 1 DM confirmed the presence of this "insulitis."[6]

Current thinking on the pathogenesis of Type 1 DM involves an interaction of genetic and infectious or environmental factors that trigger an immune mediated reaction. The appearance of autoantibodies constitutes a response against altered beta cell antigens or molecules in beta cells that resemble viral proteins.[7]

Environmental agents that have been theorized to trigger this sequence include viruses, toxic chemicals, early exposure to cow's milk, and cytotoxins.[7,8] Recent evidence also suggests a role for vitamin D in the pathogenesis and prevention of Type 1 DM.[8]

Type 2 DM is the most common form of DM worldwide; its incidence increases with age, especially after age 40. The condition can vary from predominant insulin resistance with relative insulin deficiency to a predominant insulin-excretory defect with insulin resistance.[9] Caloric excess (usually consisting of a high-fat diet) accompanied by inadequate caloric

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expenditure (lack of physical activity) in a susceptible genotype is presumed to be the cause of Type 2 DM.[10] Type 2 DM in children is on the rise, especially in the high-risk ethnic groups. Most of these children are between 10 and 19 years old, have infrequent or mild diabetic ketoacidosis, are obese, and have a strong family history of DM.[11]

DM cannot be optimally managed in isolation. The benefits of an integrated, interdisciplinary team model for management of chronic illness are well documented. For example, chronic disease management systems (CDMS) have been implemented for patients living with (or at risk for developing) conditions such as asthma, congestive heart failure, and hypertension. Application of this model, however, is still relatively new to DM. Successful adoption of this team concept requires a paradigm shift in how providers, including allied health care professionals, and patients view their respective roles in the process. Recognition of the complexity of DM treatment has prompted both the American Association of Diabetes Educators (AADE) and the American Diabetes Association (ADA) to endorse team management as the optimal model for care.[12]

The ADA recommends that all adults aged 45 years and older be screened for DM.[1] Patients who at higher risk should be screened at younger ages and more frequently. In general, high-risk individuals are:

- Obese: > 120% desirable body weight or body mass index >27 kg/m^2
- A first degree relative of someone with DM
- Members of high-risk ethnic groups (African-American, Hispanic, Native American)
- Women diagnosed with gestational diabetes or with babies weighing more than 9 pounds
- Hypertensive (blood pressure >140/90)
- Those with HDL cholesterol less than 35 mg/dl and/or triglyceride level greater than 250 mg/dl
- Those who have had impaired fasting glucose or impaired glucose tolerance on previous testing[4]

People with diabetic retinopathy (DR) represent a large segment of the population with vision impairment.[13] In the 1990s, blindness secondary to DR in the United States was estimated to cost 500 million dollars annually.[14] According to the Centers for Disease Control, DM was the sixth leading cause of blindness in the United States in 2000.[2] By 2004, it was the leading cause of blindness for persons younger than 75.[11] Both the prevalence and the economic burden of the disease are on the rise.

Severe vision loss from DR is often preventable with timely detection and treatment. One study has shown that in 90% of the cases, blindness secondary to DR is preventable.[15] Unfortunately, studies of managed care organizations have shown that fundus examination for diabetic retinopathy has been the quality of care indicator with the lowest compliance measure.[16]

The primary purpose of this paper is to present an interdisciplinary approach to management of the ocular complications of DM and to educate clinicians about diabetic eye disease. In addition, we review those risk factors that are most predicative of DR and outline the clinical features, diagnosis, and treatment of those sight-threatening elements of DR. By identifying risk factors and early signs of DR, clinicians can provide early monitoring and/or treatment to those patients in need, within the team-oriented model of care.

A Global Approach to DM Management

Optimal DM care cannot be achieved in isolation. One professional or profession cannot provide comprehensive service to diabetic patients that will appropriately meet their diverse needs. For example, optometric physicians, though skilled primary eye care providers, cannot adequately care for patients with diabetic podiatric problems.

An interdisciplinary team approach to DM management is essential. A recent American Public Health Association statement recommended comprehensive (interdisciplinary) care for all persons with DM, including high-risk populations of African-Americans, Hispanic Americans, and Native Americans. The Diabetes Control and Complications Trial (DCCT) provided evidence that a model of interdisciplinary team care resulted in improved metabolic control and better overall outcomes.[17] A team-oriented model is maximally effective when it becomes an interdisciplinary commitment on the part of several individuals. We advocate an interdisciplinary concept of care using a patient empowerment-based model.

An effective DM management team should have a coordinator. It is common for the primary care physician (PCP) to oversee the entire team. Without a coordinator, the care can be fragmented and cost-ineffective. The Indiana Chronic Disease Management Program (ICDMP) implements Nurse Care Managers who work with the patient's PCP to deliver a consistent message to patients regarding management of their chronic disease. Nurse Care Managers also provide one-on-one assessments and education to patients during an intervention period and subsequent reinforcement phase. The coordinator may also be a Certified Diabetes Educator, or health care professional with expertise in DM education that has met eligibility requirements and successfully completed a certification examination.

Chronic disease management has long been accomplished through the use of a compliance-based model. Whether the treatment is medical, surgical, or rehabilitative, patients were simply told what to do, and were expected to comply with the management plan. Using this model, the decision-making power was thought to rest solely with the health professionals. The compliance model, however, does not take into account one crucial factor: people make their own choices.[18] These choices are based upon an individual's values, goals, needs, fears, and problems as human beings living with DM and other

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diseases.

Anderson and colleagues describe a DM management program, based on patient empowerment, which was tested on patients at the University of Michigan. In a randomized controlled trial, measurable improvements in attitude and self-efficacy, as well as a significant (0.71%) reduction in glycosylated hemoglobin resulted from this chronic disease management program.[19]

In the empowerment-based model, people accept the fact that DM is part of their life, and they adapt to live well despite this challenge. This model gives people the opportunity to explore healthy coping strategies, drawing upon the expertise of each member of the health care team. The power is split between the health professionals and the patient, who is no longer a passive member of the team, but a primary decision-maker.[20] Patient empowerment requires effective patient education. This can be accomplished via several avenues. Each member of the healthcare team can provide in-office education and supplement this with appropriate written materials. The case manager or chronic care coordinator can facilitate the overall patient education component. Various DM wellness programs are available through hospitals and outpatient clinics.

Table 1 lists several potential members of the interdisciplinary DM management team, with the patient as the central member. Other professionals and sub-specialists may be added to the team on a case-by-case basis.

Table 1:Composition of an Interdisciplinary Diabetes Mellitus Management Team

Optometric physicians
Ophthalmologists/Retinologists
Primary care medical physicians
Other sub-specialists (i.e. Endocrinologists, Geriatric Medicine, Neurologists, Rehabilitative medicine)
Physician assistants, Nursing professionals
Occupational therapists
Physical therapists
Pharmacists, Certified diabetes educators (CDE)
Dentists
Dietitians, Nutritionists
Podiatrists
Behavioral scientists

Economic Impact of Early Detection and Treatment of DR

There is overwhelming evidence of the efficacy of treatment of DR. In Iceland, all persons with type 1 DM have been screened for DR through a central system since 1980. Using this system, only one case of blindness secondary to DR has been reported since the 1990s.[21] The results of this screening program and others like it supports the view that identification (of DR) and timely intervention can be successful on a large-scale basis, at least in type 1 DM. The cost of undiagnosed DM is higher than the expense associated with managing the pathological endpoints. These already account for 1 of every 7 health care dollars spent in the United States, a number that is expected to rise dramatically in the coming years.[22,23] Blindness alone has been associated with increased length of hospital stay, nursing home placement, and hip fracture.[24-27] These facts indicate a need for timely detection and management of DR.

But do these team-oriented, chronic disease management systems save money and improve patient outcomes? In a report of simultaneous short-term savings and quality improvement associated with a health maintenance organization (HMO)-sponsored disease management program, the authors evaluated an interdisciplinary program for patients with DM. This study compared health care costs for patients who fulfilled Health Employer Data and Information Set (HEDIS) criteria for DM and were in an HMO-sponsored disease management program with costs for those not in the disease management program. In this HMO, an opt-in DM

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disease management program appeared to be associated with a significant reduction in health care costs and other measures of health care use. There was also a simultaneous improvement in HEDIS measures of quality care and patient outcomes. The percentage of patients that underwent HbA1c testing as well as lipid, eye, and kidney screening were 96.6, 91.1, 79.1, and 68.5%, respectively among program patients, compared with 83.8, 77.6, 64.9, and 39.3%, respectively among non-program patients. These data suggest that patient education, clinical guidelines with provider teaming, and financial performance need not be mutually exclusive.[28]

We as members of the provider team must address the various barriers to care that patients living with DM face. In a recent study, people with DM were more likely to have had an eye test within the last 2 years if they had seen a healthcare provider or had one of various health checks, including checks not related to DM, within the same time frame. These results suggest that people who take an interest in their general health may also be more aware of the importance of eye examinations to avoid vision loss. Eye health promotion activities, therefore, need to broaden their reach to approach from outside the health sector, targeting people with DM who normally do not receive health checks. The importance of dilated eye examinations for people with DM needs to be further promoted by all providers.[29]

There must exist an environment of collegiality among team members that is mutually respectful, trustful, and non-competitive. Information needs to flow between members of the health care team. The primary care provider (PCP) needs to know what is happening with his/her patient's eyes, and too often eye care providers fail to complete the communication link back to the PCP. Likewise, specific information from the PCP or team coordinator about the patient's DM history and current level of glycemic control is invaluable to the eye care provider. We recommend that each member of the team concentrate on their own area of expertise, but communicate their results and recommendations to the others clearly, and at times, with sufficient tact. We must keep our eyes on the ultimate goal: a well adjusted, empowered, optimally functioning person who has overcome the challenges that DM poses.

Complications of Diabetes Mellitus

DM has several systemic complications. These include heart disease, kidney failure and circulatory problems, potentially leading to amputation and blindness. Additionally, neuropathies can produce functional difficulties for the patient. Common systemic symptoms may include polyuria, polydipsia, polyphagia, unexplained weight changes, dry mouth, pruritus leg cramps or pains, impotence, delayed healing of bruises or wounds, and recurrent infections of the skin, genitalia or urinary tract. Systemic complaints are more common in patients with type 1 DM. Patients with type 2 DM are frequently asymptomatic. Table 2 outlines DM-related systemic complications.[9]

Table 2: Systemic Complications of DM

Cardiovascular Disease Atherosclerosis Stroke Myocardial Infarction Hypertension	***Kidney Disease*** Kidney failure requiring transplantation or dialysis
Nerve Disease Amputations Pain Loss of Sensation Muscle Weakness	***Circulatory*** Gum Disease Reduced Wound Healing

Common ocular symptoms of undiagnosed DM include a recent onset of blurred or fluctuating vision, diplopia (double vision), and ocular dryness. A loss of fine detail in central vision is typically one of the first and most common symptoms of DR. Night vision problems, flashes, and floaters are other, less common symptoms of DR. The disease can eventually lead to retinal detachment, glaucoma, and blindness at its most severe.

The symptom of fluctuating vision may be caused by refractive error shifts. With elevated levels of glucose, the glycolytic pathway backs up, forcing some of the glucose to shift into the polyol metabolic pathway. In this pathway, aldose reductase converts glucose to sorbitol. Sorbitol can act osmotically to shift fluid into cells. As glucose levels drop, sorbitol levels also fall and fluid shifts out of cells. These osmotic shifts change the shape of the eye's crystalline lens, thus altering a patient's refractive error, resulting in blur and visual fluctuation.[30]

Eventually, most people with DM will develop some degree of DR.[31] DR is common in both Type 1 and Type 2 DM, thereby affecting a broader range of people than many other conditions.

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DR also has a wide spectrum of severity and may not progress uniformly, implying that people with DR may have varying degrees of residual vision, regardless of age and other factors. The exact cause of microvascular complications is not known. The Diabetes Control and Complications Trial (DCCT) showed that control of hyperglycemia decreased microvascular complications. DR is a small vessel disease that affects the capillaries before the larger vessels are affected. Pericytes provide structural support for the endothelial cells. An early finding in DM is the loss of pericytes, which may cause leakage and dysfunction of endothelial cells. Out-pouchings of the capillaries, called microaneurysms, are frequently the earliest clinically detectable sign of DR. DR results from an alteration in retinal blood flow that degrades performance of the retina.[32,33] Over time, non-perfusion of retinal capillaries weakens the capillary walls, resulting in bulging, leaking, or scarring of blood vessels. With tissue ischemia, angiogenic factors are released, causing new blood vessel formation (neovascularization) as well as leakage from normal vessels.[34] Table 3 outlines the many ocular manifestations of DM.[35]

Table 3: Ocular Complications of DM

Functional vision problems	***Extraocular muscle anomalies***	***External ocular anomalies***
• Color vision deficiencies • Refractive error changes • Accommodative dysfunction • Visual field defects • Reduced contrast sensitivity	• Mononeuropathies involving third, fourth, or sixth cranial nerves	• Sluggish pupillary reflexes • Bulbar conjunctival microaneurysms • Tear film deficiencies, resulting in dry eye syndrome
Cornea • Reduced sensitivity • Reduced wound-healing ability • Increased frequency of abrasions or recurrent erosion syndrome. (See Figure 1)	**Iris** • Depigmentation • Rubeosis iridis • Neovascular glaucoma	**Lens** • Higher prevalence of cataracts (See Figure 2)
Vitreous • Hemorrhage in proliferative retinopathy	**Retina** • Nonproliferative retinopathy (NPDR) • Proliferative retinopathy (PDR) (See Figures 3 and 4) • Macular edema (CSME) (See Figure 5) • Tractional retinal detachment	**Optic Nerve** • Papillopathy • Ischemic optic neuropathy • Higher incidence of open angle glaucoma • Neovascular glaucoma

Risk Factors for Diabetic Retinopathy

DR affects 40-45% of the approximately 18 million Americans who have DM.[31] Risk factors for the development or progression of DR include increasing duration of disease (DM), hypertension, hyperglycemia, puberty, pregnancy, renal failure, hyperlipidemia, HIV infection, smoking (disputed), and genetics.[36] However, evaluations of HEDIS data show that only 35% to 50% of known diabetics undergo an annual retinal evaluation.[37]

Table 4 summarizes the results of the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR).[38,39] WESDR demonstrated how the increasing duration of DM in both Type 1 and Type 2 patients increases the incidence of ocular manifestations.[40,41] In WESDR, higher frequencies of proliferative diabetic retinopathy (PDR) were present in younger-onset men as compared with younger-onset women, but there was no significant difference in the 10-year incidence or progression of DR.[42]

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Table 4: WESDR Study: Duration of Diabetes Mellitus

Type of DM	Duration of DM	Ocular Manifestations
Type 1	5 years	Possible ocular manifestations
	>10 years	60% have some retinopathy
	>15 years	Virtually all patients have some degree of retinopathy. 25% progress to proliferative diabetic retinopathy.
	>20years	50% progress to proliferative retinopathy.
Type 2	At diagnosis	20% have retinopathy
	>4 years	4% progress to proliferative retinopathy.
	>15 years	60-80% have some retinopathy. Up to 20% progress to proliferative retinopathy.

Figure 1: Corneal abrasion in a patient with DM

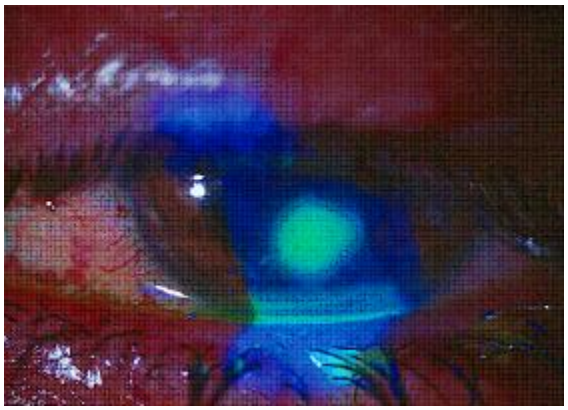


Figure 2: Dense cataract in a patient with DM

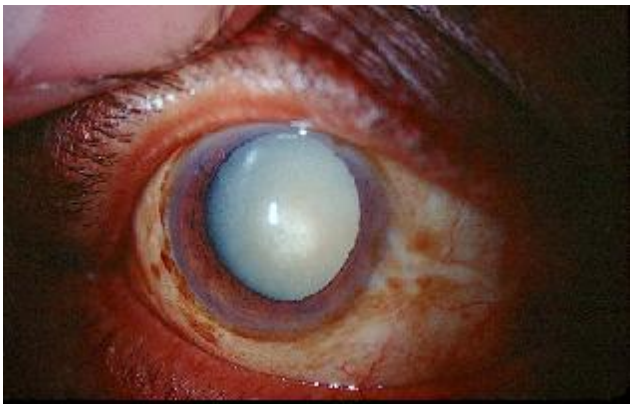


Figure 3: Proliferative retinopathy with large pre-retinal hemorrhage (Photo courtesy of Alan Kabat, OD)

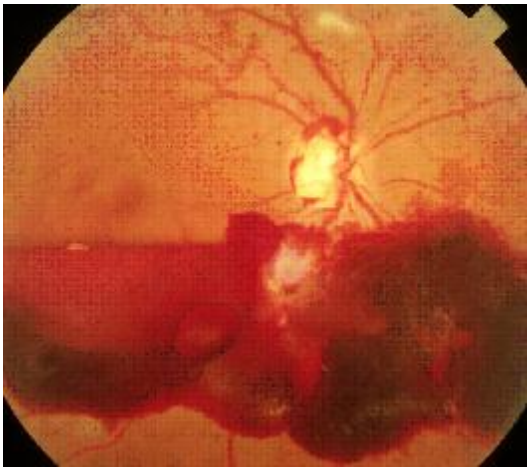


Figure 4: Proliferative retinopathy with vitreous involvement

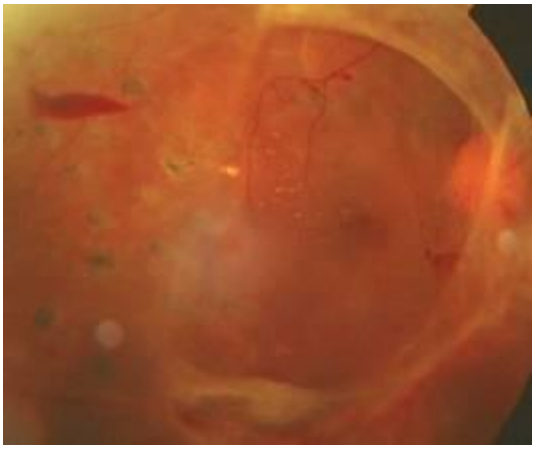
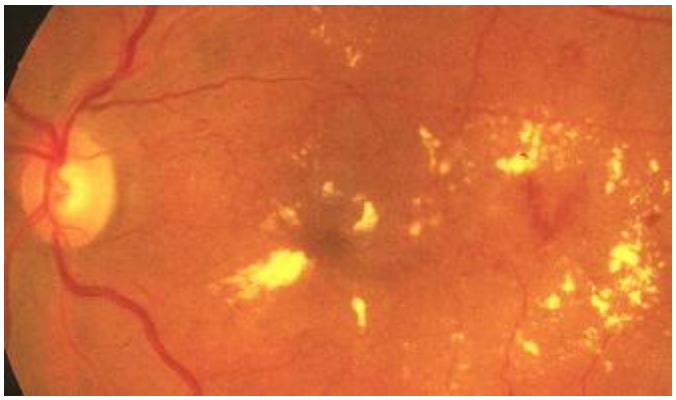
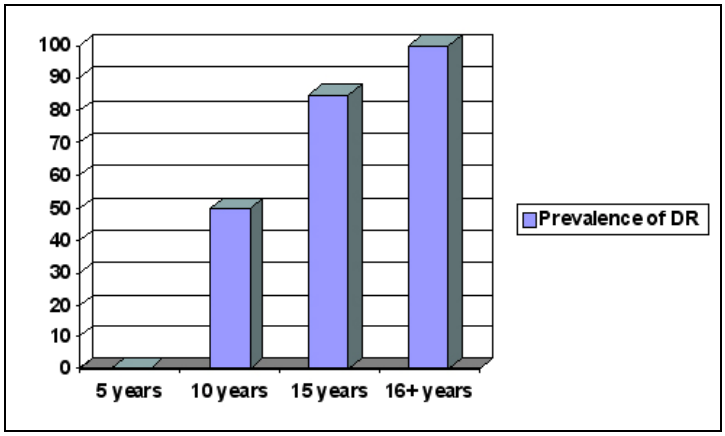


Figure 5: Macular edema with a "circinate ring" of hard exudates in a patient with DM



We recently reported the results of a study of risk factors for DR.[43] The factors that we studied were age, race, gender, smoking history, age at initial diagnosis of DM, duration of DM, and whether insulin was used for glycemic control. Subjects with DM, aged 29-79 years, underwent a health history survey and ophthalmic examination, including a dilated fundus evaluation. The Early Treatment of Diabetic Retinopathy Study (ETDRS) grading system was used to classify each fundus.[44-45] We found that duration of DM significantly predicted the presence of retinopathy (P<.001). Specifically, as duration of DM increased, the odds of developing DR increased by 30%. Figure 6 shows the increasing prevalence of DR with increasing duration of DM for patients in our study. Our results are consistent with those of WESDR.[36,38-43,46]

Figure 6: Prevalence of Diabetic Retinopathy as a Function of Duration of DM



Clinical Features and Classification of Diabetic Retinopathy

The clinical features of non-proliferative diabetic retinopathy (NPDR) include dot, blot, and flame-shaped hemorrhages, microaneurysms, intraretinal microvascular abnormalities (IRMA), venous beading, hard exudates, "cotton wool-like" infarct, and macular edema. For an eye to be classified as proliferative diabetic retinopathy (PDR), it must have one or more of the following: neovascularization of the optic disc (NVD), neovascularization elsewhere (NVE), and a vitreous or preretinal hemorrhage associated with NVE.[47]

As mentioned previously, microaneurysms are out-pouchings of retinal capillaries. IRMA are dilated and tortuous capillaries and are good indicators of progressive DR. Venous beading is a focal irregularity in the caliber of retinal veins and is a strong predictor for the development of neovascularization. Cotton-wool spots represent focal infarcts of the retinal nerve fiber layer. Ruptured microaneurysms, leaking capillaries, and IRMA may result in intraretinal hemorrhages. The ophthalmoscopic appearance of these hemorrhages is consistent with the retinal level in which they occur. Hemorrhages in the retinal nerve fiber layer have a flame-shaped appearance and coincide with the structure of the nerve fiber layer. Hemorrhages deeper in the retina, assume a pinpoint, blot, or dot shape and are more characteristic of DR.

Leakage from peri-foveal vessels causes macular edema, which, if left untreated, may result in permanent vision loss. This edema can occur at any stage of retinopathy. The classification of clinically significant macular edema (CSME) from the ETDRS grading system requires the presence of one or more of the following: thickening of the retina within 500 microns (1/3 optic disc diameter) from the center of the center of the macula, hard exudates within 500 microns from the center of the macula with associated thickening of the adjacent retina, and a zone or zones of retinal thickening greater than 1 optic disc diameter (DD) in size, any portion of which is within 1 DD from the center of the macula.[44,45]

Diagnosis and Treatment of Diabetic Retinopathy

Treatment for NPDR (Without CSME)

NPDR is significant because of its rate of progression into PDR. Thus, the stage of NPDR at the initial diagnosis dictates the follow-up schedule for the patient. Mild NPDR has a 5% risk of progressing to PDR in 1 year and a 15% risk of progression to high-risk PDR within 5 years. Moderate NPDR has a 12-27% risk of progressing to PDR in 1 year and a 33 % risk of progressing to high-risk PDR within 5 years. Severe NPDR has a 52% risk of progressing to PDR in 1 year and a 60% risk of progressing to high-risk PDR within 5 years.[9,44]

Management of NPDR centers on stabilizing and arresting the progression of DR. The DCCT showed that intensive DM control, involving multiple daily blood sugar measurements, nutritional counseling, medical evaluations every 3 months, and glycosylated hemoglobin evaluation every 3 months, decreased the development and progression of retinopathy. Using an

interdisciplinary approach, the ADA recommends close blood pressure and cholesterol monitoring, as well as smoking avoidance, exercise, and weight control. DM patients without retinopathy or with mild NPDR should be monitored with dilated fundoscopy on an annual basis. Those with moderate-to-severe NPDR should be monitored with more frequent dilated fundoscopy. Instruction on DM self-management by a Certified Diabetes Educator is a mainstay of interdisciplinary management.

Treatment for PDR (Without CSME)

In cases of PDR and CSME, fluorescein angiography is needed to detect treatment landmarks and patterns of leakage. Injecting a fluorescein dye into the arm and subsequently photographing the retina through a dilated pupil using a special filter accomplishes this test.

Treatment of PDR usually involves laser surgery to seal leaking vessels and prevent the development of aberrant, new blood vessels. These new, weaker blood vessels can rupture, scar, and cause death of retinal tissue. Early treatment of PDR with photocoagulation surgery reduces the risk of severe vision loss by at least 50-60 percent. The various structures of the eye respond to different wavelengths of laser light. This necessitates different types of laser treatment, depending on the location of the disease process within the eye. In some cases of PDR, a vitrectomy procedure (removal of the vitreous body) is performed. Indications for vitrectomy surgery include vitreous hemorrhage that blocks the view of the retina, dense premacular hemorrhage, complicated retinal detachment, and severe neovascular proliferation that is non-responsive to laser treatment.[48,49]

Treatment of CSME

Optical coherence tomography (OCT) is an in-vivo imaging technology that displays different layers of retinal structure clearly and processes the images objectively. OCT can be helpful in establishing the diagnosis of CSME in a non-invasive manner. In OCT, CSME is represented by an increase in retinal thickening due to intraretinal fluid leakage.

The goal of laser treatment for CSME is not to improve vision, but to try to slow visual loss as a result of chronic edema and resultant tissue damage. Focal or grid laser photocoagulation exerts its beneficial effect on macular edema by producing coagulation necrosis. Patients with macular edema that is not clinically significant should be followed every 3 to 6 months.

Conclusions

The longer a patient has been living with DM, the more likely it is that he/she will develop DR.[43,50] DR is treatable. In addition to exercise, proper diet, and aggressive glycemic control, patients with DM should be educated to adhere to guidelines established by the American Optometric Association for an annual dilated fundus evaluation. By detecting diabetic retinopathy earlier, many of its potentially sight- threatening complications could be reduced or prevented.

In interdisciplinary care, providers share a common professional identity and purpose. Egos are put aside and roles of the team members remain flexible. Interdisciplinary DM management can result in fewer long-term diabetic complications. Allied health care providers have a significant role in caring for patients with DM. Owing to their diverse and expanding roles in health care delivery systems, allied health professionals are in a unique position to work with eye care practitioners and other team members to improve the quality of life and health of their patients living with diabetes. By properly screening for symptoms such as visual blur and for ocular complications such as retinopathy, allied health professionals can facilitate appropriate diagnostic testing and treatment to reduce the likelihood of significant vision loss.

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References

1. American Diabetes Association. Screening for diabetes, Diabetes Care 1998;21(suppl 1):s 20-22.
2. Centers for Disease Control and Prevention, Department of Health and Human Services. (2000) National diabetes fact sheet: general information and national estimates on diabetes in the United States, 2000. Atlanta, GA: United States Department of Health and Human Services.
3. Mokdad AH, Ford ES, Bowman BA, et al. Diabetes trends in the U.S.: 1990-1998. Diabetes Care. 2000;23(9):1278-83.
4. American Diabetes Association. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care. 1997;20: 1183-97.
5. Devendra D, Liu E, Eisenbarth GS. Type 1 diabetes: recent developments. BMJ328: 750 –754, 2004.
6. Imagawa A, Hanafusa T, Itoh N, Waguri M, Yamamoto K, Miyagawa J, et al. Immunological abnormalities in islets at diagnosis paralleled further deterioration of glycaemic control in patients with recent-onset type I (insulin-dependent) diabetes mellitus. Diabetologia 1999;42: 574-8.
7. Atkinson MA, Eisenbarth GS. Type 1A diabetes: new perspectives on disease pathogenesis and treatment. Lancet 2001;358: 221-9.
8. Mathieu C, Gysemans C, Giulietti A, Bouillon R. Vitamin D and

diabetes. Diabetologia. Jul 2005;48(7):1247-57.
9. American Optometric Association. Clinical Practice Guidelines for Diabetic Patients. http://www.aoa.org/documents.
10. Wannamethee SG, Shaper AG, Alberti KG. Physical activity, metabolic factors, and the incidence of coronary heart disease and type 2 diabetes. Arch Intern Med. Jul 24 2000;160(14):2108-16.
11. The Eye Disease Prevalence Research Group. National Institutes of Health. Vision Loss will increase as Americans age. http://www.nei.nih.gov/news/pressre;eases/041204.asp. Last accessed September 28, 2004.
12. Bayless M, Martin C: The team approach to intensive diabetes management. Diabetes Spectrum, 11(1): 33-37, 1998.
13. Vertesi, A., Lever, J.A., Molloy, D. W., Sanderson, B., Tuttle, I., Pokoradi, L., & Vision Problems in the US. Prevalence of adult vision impairment and age-related eye disease in America. Prevent Blindness America 2002:26-29.
14. Chiang YP, Bassi LJ, Javitt JC. Federal budgetary cost of blindness. Milbank Q 1992;70:319-40.
15. American Academy of Ophthalmology Retina Panel, Preferred Practice Patterns Committee. Diabetic retinopathy. http://www.guideline.gov/summary/summary.aspx?doc_id=4350. Last accessed October 4, 2004.
16. Diabetes Physician Recognition Program. http://www.ncqa.org/dprp/dprpfaq.htm#Diabetes%20Physician%20Recognition%20Program%20Measures%20for%20Adult%20Patients. Last accessed October 1, 2004.
17. Eastman RC, Seibert CW, Harris M, Gorden P: Implications of the Diabetes Control and Complications trial. J Clinical Endocrinol metab 77:1105-1107, 1993.
18. Feste C, Anderson A. Empowerment: from philosophy to practice. Patient Education Counseling. 1995; 26: 139-144.
19. Anderson RM, Funnell MM, Butler PM, Arnold MS, Fitzgerald JT, Feste C. Patient empowerment: results of a randomized controlled trial. Diabetes Care. 1995; 18: 943-949.
20. Feste C. A practical look at patient empowerment. Diabetes Care. 1992; 15: 922-925.
21. Stefansson E, Bek T, Porta M, et al. Screening and Prevention of diabetic blindness. Acta Ophthalmol Scand 1997;223(suppl):1-76.
22. Hogan P, Dall T, Nikolov P. American Diabetes Association. Economic costs of diabetes in the US in 2002. Diabetes Care 2003;26(3):917-32.
23. Boyle JP, Honeycutt AA, Narayan KM, Hoerger TJ, Geiss LS, Chen H, et al. Projection of diabetes burden through 2050: impact of changing demography and disease prevalence in the US Diabetes Care 2001;24:1936-40.
24. Taylor TN, Chrischilles A. Economic evaluations of interventions in endocrinology. Endocrinol Metab Clin North Am 1997;26:67-87.
25. Morse AR, Yatzkan E, Berberich B, et al. Acute care hospital utilization by patients with visual impairment. Arch Ophthalmol 1999;117(7):943-9.
26. Aditya BS, Sharma JC, Allen SC, et al. Predictors of a nursing home placement from a non-acute geriatric hospital. Clin Rehabil 2003; 17(1): 108-13.
27. Ivers RQ, Norton R, Cumming RG, et al. Visual impairment and risk of hip fracture. Am J Epidemiol 2000;152(7): 633-9.
28. Sidorov J, Shull R, Tomcavage J, Girolami S, Lawton N, Harris R. Does diabetes disease management save money and improve outcomes? A report of simultaneous short-term savings and quality improvement associated with a health maintenance organization-sponsored disease management program among patients fulfilling health employer data and information set criteria. Diabetes Care 2002; 25: 684–9.
29. Muller A, Lamoureux E, Bullen C, Keeffe JE. Factors associated with regular eye examinations in people with diabetes: results from the Victorian Population Health Survey. Optom Vis Sci. 2006 Feb;83(2):96-101.
30. Bron AJ, Sparrow J, Brown NA, Harding JJ, Blakytny R. The lens in diabetes. Eye. 1993;7 (Pt 2):260-75.
31. Prevent Blindness America. (2003) Diabetes is the leading cause of new cases of blindness in American adults. Retrieved March 13, 2004 from http://www.preventblindness.org/news/releases/diabetes_2003.pdf.
32. Bursell SE, Clermont Ac, Kingley BT, et al. Retinal Blood flow changes in patients with Insulin-Dependent diabetes mellitus and no diabetic retinopathy. Invest Ophthalmol. Vis Sci. 1996;37(5): 886-97
33. Fong D, Bursell SE, Clermont Ac, et al. Von Willebrand factor and retinal circulation in early-stage retinopathy of type 1 diabetes. Diabetes Care 2000:23:1694-8
34. Hammes HP, Lin J, Renner O, Shani M, Lundqvist A, Betsholtz C, Brownlee M, Deutsch U. Pericytes and the Pathogenesis of Diabetic Retinopathy. Diabetes 2002; 51:3107-12.
35. Aiello LM, Cavallerano JD. Ocular complications of diabetes mellitus. In: Kahn CR, Weir GC, eds. Joslin's diabetes mellitus, 13th ed. Philadelphia: Lea & Febiger; 1994:771-93.
36. Klein R, Klein BEK, Moss SE, Cruickshanks KJ. The Wisconsin Epidemiologic Study of Diabetic Retinopathy, XIV: ten-year incidence and progression of diabetic retinopathy. Arch Ophthalmol 1994;112:1217-1228.
37. Benjamin SM,Rolka DB, Valdez R, et al. Estimated number of adults with prediabetes in the U.S. 2000. Diabetes Care 2003;26(2): 349-54.
38. Klein R, Klein BEK, Moss SE, et al. The Wisconsin epidemiologic study of diabetic retinopathy. III. Prevalence and risk of diabetic retinopathy when age at diagnosis is 30 or more years. Arch Ophthalmol 1984;102:527-32.

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39. 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 1984;102:520-6.
40. Klein R, Klein BEK, Moss SE, et al. The Wisconsin epidemiologic study of diabetic retinopathy. IX. Four-year incidence and progression of diabetic retinopathy when age at diagnosis is less than 30 years. Arch Ophthalmol 1989; 107:237-43.
41. Klein R, Klein BEK, Moss SE, et al. The Wisconsin epidemiologic study of diabetic retinopathy. X. Four-year incidence and progression of diabetic retinopathy when age at diagnosis is 30 years or more. Arch Ophthalmol 1989; 107:244-9.
42. Klein R, Klein BEK, Moss SE, et al. Prevalence of diabetes mellitus in southern Wisconsin. Am J Epidemiol 1984;119:54-61.
43. Bade A, Pizzimenti JJ, Oliver PR. Risk factors for diabetic retinopathy. Optom Vis Sci 2004; 81 (suppl.): 226.
44. Early Treatment Diabetic Retinopathy Study Research Group. Grading diabetic retinopathy from stereoscopic color fundus photographs: An extension of the modified Airlie House classification. ETDRS Report No.10 Ophthalmology 1991;98:786-806.
45. Early Treatment Diabetic Retinopathy Study Research Group. Fundus photographic risk factors for progression of diabetic retinopathy. ETDRS Report No 12. Ophthalmology 1991;98:823-40.
46. Klein R, Klein BEK, Moss SE, Cruickshanks K. The Wisconsin Epidemiologic Study of Diabetic Retinopathy XVII. Ophthalmology 1998;105:1801-1815
47. University of Maryland School of Medicine, Department of Epidemiology and Preventive Medicine (1980). Early treatment diabetic retinopathy study. Manual of Operations, pp. 1-15. Baltimore: ETDRS Coordinating Center.
48. American Optometric Association, Guidelines for Care of the Patient with Diabetes 2002; pp. 12.
49. Early Treatment Diabetic Retinopathy Study Research Group. Classification of diabetic retinopathy from fluorescein angiograms. ETDRS Report No. 11. Ophthalmology 1991;98:807-22
50. Kostraba JN, Dorman JS, Orchard TJ, et al. Contribution of diabetes duration before puberty to development of microvascular complications in IDDM subjects. Diabetes Care 1989;12:686-93.

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