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Risk Factors for Proliferative Diabetic Retinopathy in African Americans with Type 2 Diabetes

Alan Penman[1,2], **Heather Hancock**[3], **Evangelia Papavasileiou**[4], **Maurice James**[5], **Omolola Idowu**[3], **Daniel M. Riche**[1], **Marlene Fernandez**[6], **Stacey Brauner**[4], **Sataria O. Smith**[3], **Suzanne Hoadley**[3], **Cole Richardson**[3], **Vanessa Vazquez**[7], **Cheryl Chi**[7], **Christopher Andreoli**[8], **Deeba Husain**[4], **Ching J. Chen**[3], and **Lucia Sobrin**[4]

[1]Department of Medicine, University of Mississippi Medical Center, Jackson, MS

[2]Center of Biostatistics and Bioinformatics, University of Mississippi Medical Center, Jackson, MS

[3]Department of Ophthalmology, University of Mississippi Medical Center, Jackson, MS

[4]Department of Ophthalmology, Harvard Medical School, Massachusetts Eye and Ear Infirmary, Boston, MA

[5]Ophthalmology, St Dominic's Hospital, Jackson, MS

[6]Center for Human Genetic Research, Massachusetts General Hospital, Boston, MA

[7]Department of Ophthalmology, Boston Medical Center, Boston, MA

[8]Visual Services Department, Harvard Vanguard Medical Associates, Boston, MA

Abstract

Purpose—To assess personal and demographic risk factors for proliferative diabetic retinopathy in African Americans with type 2 diabetes.

Methods—In this prospective, non-interventional, cross-sectional case-control study, 380 African Americans with type 2 diabetes were enrolled. Participants were recruited prospectively and had to have either; 1) absence of diabetic retinopathy after ≥10 years of type 2 diabetes or, 2) presence of proliferative diabetic retinopathy when enrolled. Dilated, 7-field fundus photographs were graded using the Early Treatment Diabetic Retinopathy Study scale. Covariates including hemoglobin A_{1C} (HbA_{1C}), blood pressure, height, weight and waist circumference were collected prospectively. Multivariate regression models adjusted for age, sex and site were constructed to assess associations between risk factors and proliferative diabetic retinopathy.

Results—Proliferative diabetic retinopathy was associated with longer duration of diabetes (odds ratio, OR, 1.62, P<0.001), higher systolic blood pressure (OR 1.65, P<0.001) and insulin use (OR 6.65, P<0.001) in the multivariate regression analysis. HbA_{1C} was associated with proliferative

Corresponding Author: Lucia Sobrin, MD, MPH, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, MA 02114, Tel 617-573-4279, Fax 617-573-3011, Lucia_sobrin@meei.harvard.edu.

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diabetic retinopathy in the univariate analysis (OR 1.31, P=0.002) but was no longer significant in the multivariate analysis.

Conclusions—In this case-control study of African Americans with type 2 diabetes, duration of diabetes, systolic hypertension and insulin use were strong risk factors for the development of proliferative diabetic retinopathy. Interestingly, HbA_{1C} did not confer additional risk in this cohort.

Keywords

African Americans; Diabetic retinopathy; Proliferative diabetic retinopathy; Risk factors; Type 2 diabetes

Introduction

Proliferative diabetic retinopathy (PDR) is a leading cause of new cases of blindness among adults aged 20–74 years in the United States.[1] In the US, African Americans have a high prevalence of type 2 diabetes and seem to be at high risk for microvascular complications, including diabetic retinopathy (DR).[2] Some studies have found that African Americans have a higher risk of developing DR compared with Caucasians after adjusting for clinical risk factors.[3–7] For example, the prevalence of moderate non-proliferative DR or worse was higher for African American veterans than for Caucasian veterans in the Veterans Affairs Diabetes Trial of type 2 diabetes, and could not be accounted for by traditional risk factors.[5]

Risk factors for developing any DR have previously been well delineated in many studies.[3, 5, 8–17] Longer diabetes duration, hyperglycemia and hypertension are the most consistent risk factors for DR.[18] Of studies that have reported DR risk factors in various ethnicities, few studies have reported risk factors for PDR in selected subsets of these large cohorts.[14, 16, 19] Risk factors for progression to PDR in particular are of importance given the high risk of profound vision loss in these patients, and there is evidence to suggest that risk factors for advanced DR stages may differ in some respects from risk factors for any DR.[20]

The number of studies that have examined DR risk factors in African Americans is more limited (Table 1). Among these studies, most have not reported risk factors for PDR specifically. Those that have were studies performed in either exclusive type 1 diabetes populations or mixed type 1 and type 2 diabetes populations.[17, 19, 21] To the best of our knowledge, risk factors for developing PDR specifically (as opposed to any retinopathy) in an exclusively African American population with type 2 diabetes have not been described. In this report, we explore this issue in a case-control study to determine the personal and demographic risk factors for PDR in African Americans with type 2 diabetes.

Materials and Methods

Participants and retinopathy assessment

The Institutional Review Boards of the University of Mississippi Medical Center, Massachusetts Eye and Ear Infirmary, and Boston Medical Center approved this study, and all participants gave written informed consent after explanation of the nature and possible

consequences of the study. All procedures conformed to the tenets of the Declaration of Helsinki.

Participants for the African American Proliferative Diabetic Retinopathy Study were recruited between 2011 and 2013 from 4 clinical sites: University of Mississippi Medical Center, Massachusetts Eye and Ear Infirmary, Boston Medical Center and Harvard Vanguard Medical Associates. All participants self-identified as African American and had a known diagnosis of type 2 diabetes by 2003 American Diabetes Association criteria[22] and/or by being on anti-diabetic medication. To be enrolled in the study, participants had to meet either the case or control definition. Cases were patients with PDR either in 1 or both eyes. Controls were patients with no DR in either eye and at least 10 years of diagnosed diabetes. Dilated, digital 7-standard field fundus photography of both eyes was obtained using a Topcon TRC 50 DX camera (Topcon, Tokyo, Japan). Photographs were graded for degree of DR by 2 independent, masked ophthalmologist-investigators. Level of retinopathy was scored using the Early Treatment Diabetic Retinopathy Study (ETDRS) adaptation of the modified Airlie House classification.[23] Intergrader reliability was determined by percent agreement and weighted kappa. For ETDRS level, the percent agreement and weighted kappa were 96.5% and 0.90, respectively. This indicated substantial intergrader reliability.[24] Disagreements were arbitrated by a third masked ophthalmologist-investigator. If there was still disagreement after arbitration, the final grade was determined by joint review of the ophthalmologist-investigators.

Covariate data

The covariates examined in this study were age, sex, site of recruitment, duration of diabetes, hemoglobin A_{1C} (HbA_{1C}), systolic blood pressure (BP), diastolic BP, body mass index (BMI), waist circumference, total cholesterol, low density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol, triglycerides, smoking status, and insulin use. These covariates have been implicated as risk factors for PDR in studies of type 2 diabetes patients of varied ethnicities.[14, 16, 19, 25] Covariate data were collected by uniform methods at the study visit or from medical records. Using a standardized questionnaire, duration of diabetes, history of smoking, insulin use, and oral hypoglycemic agent use were recorded. Duration of diabetes was verified by review of medical records. For each participant, a blood sample was sent for HbA_{1c} measurement at a centralized laboratory. Blood was also collected for genetic analyses to be completed at a later date. Resting, seated BP was measured 3 times and the mean was used in the analyses. Standing height, weight, and waist circumference were recorded at the study visit. BMI was calculated as weight in kilograms divided by height in meters squared. 1 set of fasting lipid levels (total, LDL, and HDL cholesterol and triglycerides) closest to the study visit but within 1 year of the study visit were recorded from medical records.

Statistical analyses

Sample size calculations—Sample size for this study was determined *a priori* based on preliminary data regarding the number of patients seen at the participating clinics who would meet the case and control definitions and review of the medical literature regarding the expected means and standard deviations for the risk factors examined. With 70 controls

and 300 cases, we would have between 93% and 100% power to detect the expected differences for the most consistent risk factors; duration of diabetes, HbA_{1c}, and BP.

Outcome and covariate definitions—Retinopathy status was determined based on the eye with the higher ETDRS level. Absence of retinopathy was defined as ETDRS level <14. PDR was determined as ETDRS level ≥60. If 1 eye was ungradable, the score for the other eye was used.

For statistical analyses, duration of diabetes, HbA_{1c}, mean systolic BP, mean diastolic BP, BMI, waist circumference, serum fasting total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides were evaluated as continuous variables for maximal power. Tobacco use was evaluated as a dichotomous variable; ever (past or current) smoker vs never smoker. Insulin use was also evaluated as a dichotomous variable; taking insulin vs not taking insulin.

Univariate and multivariate analyses—To examine the variables that have previously been associated with PDR and determine their association with PDR in the African American Proliferative Diabetic Retinopathy Study sample, we used the t-test for continuous variables and chi-square test for dichotomous variables. For all analyses, we used the subset of participants with complete information for the covariate of interest in that particular analysis to maximize the generalizability and power of the analysis.

Variables that differed significantly in the t-test and chi-square analyses ($P<0.05$) were considered for inclusion in the logistic regression models. All logistic regression models included age, sex and study site *a priori*. Variables were considered sequentially with measures of diabetic severity considered first. Collinearity and confounding were assessed by changes in the effect estimates and standard errors of the existing variables in the model. Collinear variables were excluded from the final models. Confounders were retained if they changed the effect estimate for an existing variable by 10% or more. All analyses were performed using Stata/IC 12.1 (StataCorp, College Station, TX, USA).

Results

There were 358 participants enrolled in this study; 68 participants had no diabetic retinopathy (ETDRS grade <14) despite at least 10 years of type 2 diabetes and qualified as controls, and 290 participants had PDR (ETDRS grade ≥60) and were designated cases. These numbers were very close to the *a priori* enrollment goals for the study. Of the 290 participants with PDR, 212 (73%) had PDR in both eyes, 43 (15%) had PDR in 1 eye and the contralateral eye was ungradable, and 35 (12%) had PDR in 1 eye with a lesser degree of retinopathy in the contralateral eye.

Table 2 shows characteristics of participants with PDR compared to those with no DR. Participants with PDR were more likely to have a longer duration of diabetes ($P<0.001$), higher HbA_{1c} ($P=0.001$), higher systolic BP ($P<0.001$), and be using insulin ($P<0.001$). After adjusting for age, sex and site, these associations remained significant for the individual risk factors (Table 3). In the full multivariate logistic regression model, duration

of diabetes, systolic BP, and insulin use remained independent risk factors for PDR (all P<0.001), while HbA_{1c} was no longer statistically significant (Table 3). The odds of having PDR were 62% higher for every 5 years of diabetes duration and 65% higher for every 10mmHg systolic BP. Insulin use was associated with a >6-fold risk of PDR.

Discussion

In this study of African Americans with type 2 diabetes, insulin use, longer duration of diabetes, and systolic hypertension were found to be associated with the development of PDR. Participants using insulin had much higher odds of having PDR compared with those not on insulin. This association has also been found in other ethnicities and may be due to the fact that participants using insulin have more severe diabetes and poorer glycemic control.[16, 25] Although the association with insulin use did remain significant after adjustment for HbA_{1c}, it should be noted that HbA_{1c} was based on a 1-time draw at the time of fundus photography, and average levels over an extended period of the participants' diabetes were not available.

The findings that longer duration of diabetes and higher systolic BP are associated with PDR are consistent with previous studies in various ethnicities and of both diabetes types.[14, 16–19, 25–27] African Americans in our cohort had a 65% increased risk of developing PDR for every 10mmHg increase in systolic BP. These findings underscore the need for patient education among African Americans with type 2 diabetes about the importance of BP control in preventing blindness from advanced DR. Prospective randomized controlled trials in patients with type 2 diabetes have shown the benefit of BP control on progression of retinopathy in general.[28, 29] Although these trials have been executed in predominantly Caucasian populations, 1 study enrolled 30% non-white patients which included African Americans along with other non-white ethnic minorities.[29] A previous study conducted in African Americans with type 1 diabetes also found elevated BP, along with glycemic control and renal disease, to be associated with PDR.[30] We did not assess renal disease in the current study. We did not find age, sex, diastolic BP, serum lipid levels, BMI, waist circumference or smoking status to be independently associated with PDR in African Americans with type 2 diabetes. While there are some reports of these factors being associated with PDR in other populations,[16, 19, 25, 31, 32] these have not been consistently found to impact development of PDR overall.

Randomized clinical trials in patients with type 2 diabetes have consistently shown that glycemic control is crucial to reducing the risk of DR progression.[28, 29] Poorer glycemic control, as measured by a higher HbA_{1c} level, was associated with PDR in our univariate analyses but did not reach statistical significance in the multivariate model. This same finding has been observed in a similar PDR study in a Latino population.[25] The association between HbA_{1c} and DR may weaken with more advanced forms of retinopathy. One explanation for this phenomenon is that the diagnosis of PDR can be the impetus for tighter regulation of blood sugar control. Patients with PDR may be motivated to achieve better HbA_{1c} levels after receiving this diagnosis and their HbA_{1c} after PDR diagnosis may not be reflective of their glycemic control for most of the duration of their diabetes. There is conflicting evidence as to whether retinopathy begins to appear at lower HbA_{1c} levels in

African Americans.[33, 34] These population-based studies on ethnic differences in HbA_{1c} have focused on prevalence of any DR and not on advanced DR. It is not known whether these potential ethnic-specific differences might affect the relationship between HbA_{1c} and PDR in African Americans.

This investigation's strengths include DR phenotyping with dilated, 7-field photography. The case and control criteria were strictly defined to enrich the study for participants at the 2 extremes of the disease spectrum and minimize misclassification bias. Most covariates were measured using standardized protocols at the same time point as fundus photography. The study fell only slightly short of its recruitment goals, and the power to detect the associations for the most consistent risk factors (diabetes duration, HbA_{1c}, and systolic BP) still ranged between 93% and 100% for the recruited cohort. The case to control ratio was 4:1. The power of this study is equivalent to that of a 220-participant study with a 1:1 case to control ratio.

There are some limitations to our study. Although most covariates were collected prospectively at the study visit and had few missing data points, we collected serum lipid measurements retrospectively and there was a higher rate of missing data for these variables. This could limit the power to detect associations with serum lipids and creates a potential selection bias, although comparison of participants missing lipid measurements compared with those with lipid measurements did not reveal any significant differences in duration of diabetes, HbA_{1c} or systolic BP. We did not have data on time to requiring insulin which might be an important covariate with shorter time to requiring insulin indicating longer, undiagnosed type 2 diabetes duration. The cross-sectional design of our study does not allow us to judge the causal or temporal relationships of the associations found. Caution must be taken in comparing risk factor results in our study to previous studies as these differences might be accounted for in part by differences in retinal photography protocols and/or definitions of retinopathy. Our sample is clinic-based rather than population-based and this limits the generalizability of the results to the overall African American population.

In summary, our study shows that longer duration of diabetes, increased systolic BP and insulin use are strong risk factors for PDR in an African American population with type 2 diabetes. To the best of our knowledge, this is the first study reporting the risk factors specifically for the development of PDR in an exclusively African American population with type 2 diabetes. These data may be useful to future investigations, particularly because African Americans have the highest prevalence of diabetes as a percentage of the adult population in the US and this prevalence is projected to increase through to 2050.[35]

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References

1. National Institute of Diabetes and Digestive and Kidney Diseases. National Diabetes Statistics. Bethesda, MD: US Department of Health and Human Services, National Institute of Health; 2011. Publication No 11-3892 2011
2. Zhang X, Cotch MF, Ryskulova A, Primo SA, Nair P, Chou CF, Geiss LS, Barker LE, Elliott AF, Crews JE, Saaddine JB. Vision health disparities in the United States by race/ethnicity, education, and economic status: findings from two nationally representative surveys. Am J Ophthalmol. 2012; 154(6 Suppl):S53–62. e51. [PubMed: 23158224]
3. Klein R, Marino EK, Kuller LH, Polak JF, Tracy RP, Gottdiener JS, Burke GL, Hubbard LD, Boineau R. The relation of atherosclerotic cardiovascular disease to retinopathy in people with diabetes in the Cardiovascular Health Study. Br J Ophthalmol. 2002; 86(1):84–90. [PubMed: 11801510]
4. Leske MC, Wu SY, Hennis A, Nemesure B, Schachat AP, Hyman L, Yang L. Nine-year incidence of diabetic retinopathy in the Barbados Eye Studies. Arch Ophthalmol. 2006; 124(2):250–255. [PubMed: 16476895]
5. Emanuele N, Sacks J, Klein R, Reda D, Anderson R, Duckworth W, Abraira C. Ethnicity, race, and baseline retinopathy correlates in the veterans affairs diabetes trial. Diabetes Care. 2005; 28(8): 1954–1958. [PubMed: 16043738]
6. Harris EL, Sherman SH, Georgopoulos A. Black-white differences in risk of developing retinopathy among individuals with type 2 diabetes. Diabetes Care. 1999; 22(5):779–783. [PubMed: 10332681]
7. Emanuele N, Moritz T, Klein R, Davis MD, Glander K, Khanna A, Thottapurathu L, Bahn G, Duckworth W, Abraira C. Ethnicity, race, and clinically significant macular edema in the Veterans Affairs Diabetes Trial (VADT). Diabetes Res Clin Pract. 2009; 86:104–110. [PubMed: 19720420]
8. Harris MI, Klein R, Cowie CC, Rowland M, Byrd-Holt DD. Is the risk of diabetic retinopathy greater in non-Hispanic blacks and Mexican Americans than in non-Hispanic whites with type 2 diabetes? A U.S. population study. Diabetes Care. 1998; 21(8):1230–1235. [PubMed: 9702425]
9. Zhang X, Saaddine JB, Chou CF, Cotch MF, Cheng YJ, Geiss LS, Gregg EW, Albright AL, Klein BE, Klein R. Prevalence of diabetic retinopathy in the United States, 2005–2008. Jama. 2010; 304(6):649–656. [PubMed: 20699456]
10. Wong TY, Klein R, Islam FM, Cotch MF, Folsom AR, Klein BE, Sharrett AR, Shea S. Diabetic retinopathy in a multi-ethnic cohort in the United States. Am J Ophthalmol. 2006; 141(3):446–455. [PubMed: 16490489]
11. Klein R, Sharrett AR, Klein BE, Moss SE, Folsom AR, Wong TY, Brancati FL, Hubbard LD, Couper D. The association of atherosclerosis, vascular risk factors, and retinopathy in adults with diabetes : the atherosclerosis risk in communities study. Ophthalmology. 2002; 109(7):1225–1234. [PubMed: 12093643]
12. Klein R, Klein BE, Moss SE, Linton KL. The Beaver Dam Eye Study. Retinopathy in adults with newly discovered and previously diagnosed diabetes mellitus. Ophthalmology. 1992; 99(1):58–62. [PubMed: 1741141]
13. Klein R, Klein BE, Moss SE, Cruickshanks KJ. The Wisconsin Epidemiologic Study of Diabetic Retinopathy: XVII. The 14-year incidence and progression of diabetic retinopathy and associated risk factors in type 1 diabetes. Ophthalmology. 1998; 105(10):1801–1815. [PubMed: 9787347]
14. Porta M, Sjoelie AK, Chaturvedi N, Stevens L, Rottiers R, Veglio M, Fuller JH. Risk factors for progression to proliferative diabetic retinopathy in the EURODIAB Prospective Complications Study. Diabetologia. 2001; 44(12):2203–2209. [PubMed: 11793022]
15. Zhang L, Krzentowski G, Albert A, Lefebvre PJ. Risk of developing retinopathy in Diabetes Control and Complications Trial type 1 diabetic patients with good or poor metabolic control. Diabetes Care. 2001; 24(7):1275–1279. [PubMed: 11423515]
16. West SK, Munoz B, Klein R, Broman AT, Sanchez R, Rodriguez J, Snyder R. Risk factors for Type II diabetes and diabetic retinopathy in a mexican-american population: Proyecto VER. Am J Ophthalmol. 2002; 134(3):390–398. [PubMed: 12208251]
17. Roy MS. Diabetic retinopathy in African Americans with type 1 diabetes: The New Jersey 725: II. Risk factors. Arch Ophthalmol. 2000; 118(1):105–115. [PubMed: 10636423]

18. Yau JW, Rogers SL, Kawasaki R, Lamoureux EL, Kowalski JW, Bek T, Chen SJ, Dekker JM, Fletcher A, Grauslund J, Haffner S, Hamman RF, Ikram MK, Kayama T, Klein BE, Klein R, Krishnaiah S, Mayurasakorn K, O'Hare JP, Orchard TJ, Porta M, Rema M, Roy MS, Sharma T, Shaw J, Taylor H, Tielsch JM, Varma R, Wang JJ, Wang N, West S, Xu L, Yasuda M, Zhang X, Mitchell P, Wong TY. Global prevalence and major risk factors of diabetic retinopathy. Diabetes Care. 2012; 35(3):556–564. [PubMed: 22301125]
19. Davis MD, Fisher MR, Gangnon RE, Barton F, Aiello LM, Chew EY, Ferris FL 3rd, Knatterud GL. Risk factors for high-risk proliferative diabetic retinopathy and severe visual loss: Early Treatment Diabetic Retinopathy Study Report #18. Invest Ophthalmol Vis Sci. 1998; 39(2):233–252. [PubMed: 9477980]
20. Klein R, Klein BE, Moss SE. Epidemiology of proliferative diabetic retinopathy. Diabetes Care. 1992; 15(12):1875–1891. [PubMed: 1464243]
21. Arfken CL, Reno PL, Santiago JV, Klein R. Development of proliferative diabetic retinopathy in African-Americans and whites with type 1 diabetes. Diabetes Care. 1998; 21(5):792–795. [PubMed: 9589242]
22. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care. 2003; 26(Suppl):S5–20. [PubMed: 12502614]
23. 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 number 10. Ophthalmology. 1991; 98(5 Suppl):786–806. [PubMed: 2062513]
24. Landis JR, Koch GG. The measurement of observer agreement for categorical data. Biometrics. 1977; 33(1):159–174. [PubMed: 843571]
25. Nittala MG, Keane PA, Zhang K, Sadda SR. Risk Factors for Proliferative Diabetic Retinopathy in a Latino American Population. Retina. 2014; 34(8):1544–1549.
26. Raman R, Rani PK, Reddi Rachepalle S, Gnanamoorthy P, Uthra S, Kumaramanickavel G, Sharma T. Prevalence of diabetic retinopathy in India: Sankara Nethralaya Diabetic Retinopathy Epidemiology and Molecular Genetics Study report 2. Ophthalmology. 2009; 116(2):311–318. [PubMed: 19084275]
27. Grauslund J, Green A, Sjolie AK. Prevalence and 25 year incidence of proliferative retinopathy among Danish type 1 diabetic patients. Diabetologia. 2009; 52(9):1829–1835. [PubMed: 19593541]
28. Stratton IM, Kohner EM, Aldington SJ, Turner RC, Holman RR, Manley SE, Matthews DR. UKPDS 50: risk factors for incidence and progression of retinopathy in Type II diabetes over 6 years from diagnosis. Diabetologia. 2001; 44(2):156–163. [PubMed: 11270671]
29. Chew EY, Ambrosius WT, Davis MD, Danis RP, Gangaputra S, Greven CM, Hubbard L, Esser BA, Lovato JF, Perdue LH, Goff DC Jr, Cushman WC, Ginsberg HN, Elam MB, Genuth S, Gerstein HC, Schubart U, Fine LJ. Effects of medical therapies on retinopathy progression in type 2 diabetes. N Engl J Med. 2010; 363(3):233–244. [PubMed: 20587587]
30. Roy MS. Diabetic retinopathy in African Americans with type 1 diabetes: The New Jersey 725: I. Methodology, population, frequency of retinopathy, and visual impairment. Arch Ophthalmol. 2000; 118(1):97–104. [PubMed: 10636422]
31. Dorchy H, Claes C, Verougstraete C. Risk factors of developing proliferative retinopathy in type 1 diabetic patients : role of BMI. Diabetes Care. 2002; 25(4):798–799. [PubMed: 11919142]
32. Tolonen N, Hietala K, Forsblom C, Harjutsalo V, Makinen VP, Kyto J, Summanen PA, Thorn LM, Waden J, Gordin D, Taskinen MR, Groop PH. Associations and interactions between lipid profiles, retinopathy and nephropathy in patients with type 1 diabetes: the FinnDiane Study. J Intern Med. 2013; 274(5):469–479. [PubMed: 23844944]
33. Tsugawa Y, Mukamal KJ, Davis RB, Taylor WC, Wee CC. Should the hemoglobin A1c diagnostic cutoff differ between blacks and whites? A cross-sectional study. Ann Intern Med. 2012; 157(3): 153–159. [PubMed: 22868832]
34. Bower JK, Brancati FL, Selvin E. No ethnic differences in the association of glycated hemoglobin with retinopathy: the national health and nutrition examination survey 2005–2008. Diabetes Care. 2013; 36(3):569–573. [PubMed: 23069841]

35. Rowley WR, Bezold C. Creating public awareness: state 2025 diabetes forecasts. Popul Health Manag. 2012; 15(4):194–200. [PubMed: 22283662]

Table 1

Previous studies examining risk factors for diabetic retinopathy in African Americans

Study	Diabetes type	AA with PDR, n	Risk factors for PDR reported	Risk factors for PDR reported in AA separately
Davis et al 1998[19]	T1D, T2D	176*	Yes	No
Arfken et al 1998[21]	T1D	17	Yes	No
Harris et al 1998[8]	T2D	5*	No	No
Roy 2000[17, 30]	T1D	137	Yes	Yes
Klein et al 2002[11]	T1D, T2D	16	No	No
Klein et al 2002[3]	T2D	2*	No	No
Emanuele et al 2005[5]	T2D	<74†	No	No
Wong et al 2006[10]	T2D	26‡	No	No
Zhang et al 2010[9]	T1D, T2D	28‡	No	No
Current study	T2D	290	Yes	Yes

*The exact number of patients with PDR was not reported; this approximation is based on percentage of AA patients and PDR participants

†74 was the number of AA participants with moderate non-proliferative diabetic retinopathy or worse; this study did not report the exact numbers or percentages of patients with PDR

‡Number of patients with vision-threatening retinopathy which includes severe non-proliferative diabetic retinopathy, clinically significant macular edema and PDR.

AA, African American; PDR, proliferative diabetic retinopathy; T1D, type 1 diabetes; T2D, type 2 diabetes

Table 2

Risk factors in type 2 diabetes African American participants with and without proliferative diabetic retinopathy, USA

Variable	No DR		PDR		
	n	% or mean (SD)	n	% or mean (SD)	P value*
Age, years	68	61.3 (9.3)	290	59.5 (11.2)	0.21
Female	68	67.6	290	60.3	0.27
Duration of diabetes, years	68	15.3 (5.4)	290	21.2 (10.3)	<0.001
Hemoglobin A_{1C}, %	68	7.6 (1.9)	289	8.5 (2.1)	0.001
Body mass index, kg/m^2	68	34.2 (8.4)	288	33.6 (7.4)	0.55
Waist circumference, cm	68	104.6 (18.1)	276	106.4 (17.0)	0.46
Systolic BP, mmHg	68	136.1 (17.7)	290	152.1 (22.9)	<0.001
Diastolic BP, mmHg	68	81.0 (11.0)	290	82.1 (13.9)	0.54
Total cholesterol, mg/dL	52	173.7 (35.8)	215	179.8 (52.0)	0.42
Triglycerides, mg/dL	51	140.3 (70.6)	211	131.4 (85.5)	0.49
LDL cholesterol, mg/dL	52	97.0 (29.2)	211	102.6 (44.6)	0.39
HDL cholesterol, mg/dL	52	50.0 (19.0)	214	51.9 (18.3)	0.52
Ever smoked	68	36.8	290	35.2	0.81
Use of insulin medication	68	32.4	290	76.2	<0.001

DR, diabetic retinopathy; PDR, proliferative diabetic retinopathy; BP, blood pressure; SD, standard deviation; LDL, low density lipoprotein; HDL, high density lipoprotein

*Chi-square test used for dichotomous variables, t-test used for continuous variables.

Table 3

Logistic regression analysis to study the effect of risk factors for proliferative diabetic retinopathy controlling for age, sex and site in African American participants, USA

Variable	Univariate analysis		Multivariate analysis	
	OR (95% CI)	P value	OR (95% CI)	P value
Duration of diabetes*	1.58 (1.31–1.91)	<0.001	1.62 (1.28–2.03)	<0.001
Hemoglobin A_{1C}	1.31 (1.10–1.55)	0.002	1.04 (0.88–1.23)	0.68
Systolic blood pressure†	1.47 (1.27–1.70)	<0.001	1.65 (1.37–1.97)	<0.001
Insulin treatment	6.63 (3.71–11.8)	<0.001	6.65 (3.33–13.3)	<0.001

OR, odds ratio; CI, confidence interval

*OR calculated per 5 year increase in duration of diabetes

†OR calculated per 10mmHg increase in systolic blood pressure