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Characteristics of Central Retinal Vein Occlusion in African Americans

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Abstract

Purpose: This article studies whether the characteristics and clinical course of African American patients with central retinal vein occlusion (CRVO) differ from other racial groups. **Methods:** This retrospective cohort study included consecutive patients diagnosed with CRVO at Duke Eye Center, Durham, North Carolina. Presenting characteristics, examination findings, treatment course, and functional and structural outcomes were compared based on patient-reported race. **Results:** A total of 479 patients with CRVO were included (64.7% white, 22.2% African American, 1.7% mixed race, and 11.4% other races). African American patients were older (68.1 vs 64.3 years, $P = .049$), more likely to be hypertensive ($P = .001$) and diabetic ($P = .000$), and had higher rates of open-angle glaucoma ($P < .000$). Presenting visual acuity (VA) was worse in African Americans (logarithm of the minimum angle of resolution 1.25 vs 0.96, $P = .010$). There were no significant differences in the proportion of patients requiring panretinal photocoagulation, intravitreal antivascular endothelial growth factor (anti-VEGF), or intravitreal corticosteroid; however, analysis of treatment-naive individuals showed a higher number of anti-VEGF injections in the first year in African Americans. Final VA was not significantly different between groups, but African Americans had higher rates of neovascular sequelae (25.0% vs 11.8%, $P = .019$; odds ratio, 2.295, $P = .088$). **Conclusions:** African Americans with CRVO presented with more severe visual impairment and more systemic and ocular risk factors for CRVO. Treatment-naive African Americans had a greater treatment burden during the first year of follow-up.

Keywords

African American, antivascular endothelial growth factor (anti-VEGF), central retinal vein occlusion, panretinal photocoagulation

Introduction

Retinal vein occlusions (RVOs) are a significant cause of visual morbidity and are the most common acquired retinal vascular disease after diabetic retinopathy.[1] Depending on the location of the occlusion, RVO can be classified as *branch* (when the occlusion is at the level of an arteriovenous crossing), *hemispheric*, or *central* (occlusion at the level of the lamina cribrosa). Established risk factors for central RVO (CRVO) include older age, hypertension, diabetes mellitus, smoking, and open-angle glaucoma.[1-3]

Most of the currently available data on the incidence and prevalence of CRVO are from population-based studies largely focused on Caucasian- or white-majority populations,[1,4] although some data have recently emerged on Hispanic,[5] African American,[5] South Asian,[6] Chinese,[7,8] and Asian Malay individuals.[9] Pooled data from multinational studies show no racial differences in prevalence of RVO.[10] Despite this, the effect of race on the presentation, characteristics, treatment course, and outcome of CRVO remains unknown.

To our knowledge, there are no studies in the published literature that describe differences in the presentation of CRVO among different racial groups. This study aims to compare the presenting features and clinical course of CRVO in African American patients vs patients of other races, who all underwent treatment at the same tertiary referral center.

Methods

This retrospective cohort study was conducted following approval by the institutional review board of the Duke University School of Medicine. The Duke Enterprise Data Unified Content Explorer system was used to identify patients diagnosed with

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Table 1. Demographic Data and Baseline Presenting Features, Analyzed by Patient-Reported Race.

	Total Study Cohort (N = 479)	African Americans (n = 106)	Non–African Americans (n = 373)	*P*
Age, mean ± SD, y	65.4 ± 14.5	68.1 ± 12.8	64.3 ± 14.9	.049[a]
Male, %	46.8	44.3	47.5	.571
Hypertension, %	79.1	91.9	75.6	.001[a]
Diabetes mellitus, %	38.9	58.2	33.8	.000[a]
Smoking, %	17.9	22.4	16.7	.228
Anticoagulant or antiplatelet use, %	56.0	65.3	53.8	.078
Open-angle glaucoma, %	42.2	60.0	37.1	.000[a]
IOP, mean ± SD, mm Hg	17.2 ± 6.9	18.9 ± 10.0	16.7 ± 5.7	.054
logMAR visual acuity (Snellen equivalent)	1.02 (20/209)	1.25 (20/356)	0.96 (20/182)	.010[a]
CME, %	72.7	73.4	72.5	.868
CST, mean ± SD, μm	490 ± 284	507 ± 314	485 ± 277	.619
Subretinal fluid, %	26.4	21.5	27.6	.321
Macular ischemia, %	31.1	25.6	32.2	.421
Foveal intraretinal hemorrhage, %	39.8	57.6	35.2	.001
RAPD, %	28.5	26.7	29.0	.723
Ischemic CRVO, %	44.8	54.4	42.4	.104
Prior PRP, %	15.0	6.3	17.1	.032[a]
Prior anti-VEGF, %	34.9	16.7	39.5	.001[a]
Prior corticosteroid, %	12.8	1.7	18.2	.005[a]

Abbreviations: anti-VEGF, antivascular endothelial growth factor; CME, cystoid macular edema; CRVO, central retinal vein occlusion; CST, central subfield thickness; IOP, intraocular pressure; logMAR, logarithm of the minimum angle of resolution; PRP, panretinal laser photocoagulation; RAPD, relative afferent pupillary defect.
[a]Statistically significant results, $P < .05$.

a CRVO between January 1, 2008, and July 1, 2016, at Duke Eye Center, Durham, North Carolina. Potential study participants without a patient-reported race in their medical record were excluded from this study.

The diagnosis of CRVO was confirmed based on clinical examination and multimodal imaging. Data collected from the baseline visit included demographic characteristics, medical and ocular history, duration of symptoms prior to presentation, any prior treatment of CRVO, presenting visual acuity (VA), presence and severity of cystoid macular edema (CME) on spectral-domain optical coherence tomography, and features on fluorescein angiography (FA). Follow-up data included treatment given—such as panretinal laser photocoagulation (PRP), intravitreal antivascular endothelial growth factor (anti-VEGF) or corticosteroids—and VA and clinical examination findings at follow-up visits.

Because ultra-widefield FA was not available for all patients during the follow-up period, a patient was said to have an ischemic CRVO if either of the following criteria were met during the first year of follow-up: (1) neovascular sequelae due to CRVO, or (2) counting fingers vision or worse attributable to the CRVO as well as physician-confirmed relative afferent pupillary defect. These criteria previously have been shown to be both sensitive and specific for determining whether a CRVO is ischemic[11] and have been correlated with severity of ischemia on ultra-widefield FA.[12]

Statistical analysis was performed using SPSS (version 21, IBM) to calculate descriptive statistics for demographic data, risk factors, presenting features, and outcomes based on racial group. Statistical significance was defined as P less than .05 and calculated using Pearson chi-squared test for categorical variables and t test for continuous variables. Multivariable logistic regression models were created to assess the relationship between race and outcome variables of interest, controlling for the confounding effects of age, sex, hypertension, diabetes mellitus, presence of open-angle glaucoma, smoking status, and use of antiplatelet agents or anticoagulants.

Results

A total of 479 patients presenting consecutively with CRVO were included. Of these, 310 (64.7%) were white, 106 (22.1%) were African American, 8 (1.7%) were of mixed race, and 55 (11.4%) were other races including Asian and Hispanic. Table 1 describes the demographic data of our study participants. Mean follow-up duration was 36.7 months in the whole cohort (mean, 37.1 months in African Americans, 35.4 months in non–African Americans, $P = .568$).

Comorbidities and Risk Factors

The mean age in our study cohort was 65.4 years at CRVO onset, with a slight female preponderance (46.8% male). African American patients were older at presentation compared with non–African Americans (mean age, 68.1 years vs 64.3 years, $P = .049$). There was no significant difference in sex distribution ($P = .571$) between racial groups.

African Americans presenting with CRVO were also more likely than their counterparts to be hypertensive ($P = .001$), have diabetes mellitus ($P = .000$), and have open-angle glaucoma ($P = .000$). One patient who was African American had

Table 2. Visual and Structural Outcomes, Analyzed by Patient-Reported Race.

	Total Study Cohort (N = 479)	African Americans (n = 106)	Non–African Americans (n = 373)	*P*
Final logMAR visual acuity (Snellen equivalent)	1.14 (20/276)	1.30 (20/400)	1.10 (20/252)	.083
PRP during follow-up, %	28.6	37.3	26.4	.078
Intravitreal anti-VEGF at initial visit, %	63.0	73.4	60.3	.052
Number of anti-VEGF injections in first year	3.3	3.1	3.3	.543
Intravitreal corticosteroid, %	11.4	12.1	11.2	.855
CME at final visit, %	46.1	47.0	45.8	.868
CST at final visit, mean ± SD, μm	324 ±179	302 ±144	329 ±187	.357
Subretinal fluid at final visit, %	3.3	3.4	3.3	.983
Neovascularization, %	14.3	25.0	11.8	.019[a]
Iris	9.6	11.8	9.0	.463
Angle	5.7	8.1	5.1	.401
Disc	2.5	4.3	2.0	.271
Elsewhere	3.8	8.5	2.4	.018[a]
Vitreous hemorrhage	7.9	11.3	7.0	.235
RVO in fellow eye during follow-up, %	17.9	24.3	16.1	.117

Abbreviations: anti-VEGF, antivascular endothelial growth factor; CME, cystoid macular edema; CST, central subfield thickness; logMAR, logarithm of the minimum angle of resolution; PRP, panretinal laser photocoagulation; RVO, retinal vein occlusion.
[a]Statistically significant results, $P < .05$.

sickle cell disease with retinopathy in the fellow eye, whereas another patient who was African American had sickle cell trait with no retinopathy in either eye. There were no significant differences in rate of smoking ($P = .228$), use of hormone replacement therapy ($P = .297$), or use of anticoagulant or antiplatelet agents ($P = .078$) between racial groups.

Central Retinal Vein Occlusion Clinical Features and Presentation

African American patients presented earlier than others (median duration of symptoms: 1 month vs 2 months, $P = .048$), with worse presenting VA (logarithm of the minimum angle of resolution [logMAR] VA 1.25 [Snellen equivalent, 20/356] vs 0.96 [Snellen, 20/182], $P = .010$) and a higher incidence of fovea-involving intraretinal hemorrhage (57.6% vs 35.2%, $P = .001$). There were no differences between groups regarding the following clinical parameters at baseline: proportion with CME, central subfield thickness on optical coherence tomography, subretinal fluid (SRF), or macular ischemia on FA.

As part of a cohort from a tertiary eye center, a number of patients had received treatment elsewhere prior to presentation. In the entire cohort, 5.1% of patients had received PRP (for management of neovascular sequelae), 32.7% intravitreal anti-VEGF (for treatment of macular edema), and 12.4% intravitreal corticosteroid (for management of macular edema). African American patients were less likely than other patients to have received prior treatment, be it PRP ($P = .032$), intravitreal anti-VEGF ($P = .001$), or intravitreal corticosteroid ($P = .005$).

Treatment Course and Outcomes

Table 2 outlines the final visual and structural outcomes. During the study period, 28.6% required PRP and 63.0% received intravitreal anti-VEGF. The average number of anti-VEGF injections given in the first year of treatment was 3.4. After adjusting for the higher proportion of treatment-naive individuals in the African American group, there were no significant differences in the administration of PRP or intravitreal anti-VEGF injections among African American patients compared with those of other races. Intravitreal corticosteroids were administered in 11.4% of all patients. There were no differences in rates of corticosteroid use between African Americans and patients of other races.

Visual outcomes were not significantly different between groups, and final VA was comparable (final logMAR VA 1.30 [Snellen, 20/400] in African Americans vs 1.10 [Snellen, 20/252] in other races, $P = .083$). There were no differences in central subfield thickness, CME, or SRF at the final visit.

Neovascularization developed in 14.3% of patients in our cohort: neovascularization of the iris in 9.6%, of the angle in 5.7%, of the disc in 2.5%, and elsewhere in 3.8%. At least 1 episode of vitreous hemorrhage was observed in 7.9% of patients during follow-up. African Americans had significantly higher rates of neovascular sequelae than other patients (25.0% vs 11.8%, $P = .019$), particularly rates of neovascularization elsewhere (8.5% vs 2.4%, $P = .018$); however, multivariable logistic regression controlling for the confounding effect of diabetes mellitus revealed no attributable increased risk from race alone (odds ratio 2.295, 95% CI, 0.883-5.967, $P = .088$). Table 3 describes the results of multivariable logistic regression analysis examining the contribution of individual risk factors to risk of developing neovascular sequelae.

African Americans, being more likely to have open-angle glaucoma and systemic risk factors for CRVO, were observed to have a slightly higher incidence of subsequent RVO in the fellow eye (24.3% vs 16.1%, $P = .117$), but this did not reach statistical significance. Multivariable logistic regression

Table 3. Multivariable Logistic Regression Output Examining Contribution of Individual Risk Factors to Risk of Developing Neovascular Consequences.

Risk Factor	Odds Ratio	95% CI	P
African American	2.295	0.883-5.967	.088
Age	1.002	0.974-1.030	.915
Sex (reference: male)	0.892	0.407-1.955	.776
Hypertension	0.531	0.208-1.357	.186
Diabetes mellitus	2.883	1.252-6.640	.013[a]
Smoking	1.134	0.404-3.186	.811
Hormone replacement therapy	1.349	0.234-7.770	.738
Anticoagulants or antiplatelet agents	0.738	0.321-1.694	.473
Open-angle glaucoma	0.835	0.330-2.114	.704

[a]Statistically significant results, $P < .05$.

Table 4. Multivariable Logistic Regression Output Examining Contribution of Individual Risk Factors to Risk of Developing Any Retinal Vein Occlusion in the Fellow Eye During Follow-up.

Risk Factor	Odds Ratio	95% CI	P
African American	1.379	0.544-3.495	.498
Age	0.986	0.959-1.013	.304
Sex (reference: male)	0.973	0.454-2.086	.945
Hypertension	0.763	0.304-1.916	.565
Diabetes mellitus	1.319	0.569-3.058	.519
Smoking	0.886	0.325-2.419	.814
Hormone replacement therapy	0.000	0.000	.999
Anticoagulants or antiplatelet agents	1.016	0.458-2.254	.969
Open-angle glaucoma	2.596	1.166-5.777	.019[a]

[a]Statistically significant results, $P < .05$.

controlling for the confounding effect of glaucoma revealed no racial predilection for subsequent RVO events in the fellow eye (Table 4).

Analysis of Treatment-Naive Patients

Subgroup analysis of treatment-naive patients (333 patients, 69.5% of the total cohort; 91 African Americans and 242 non–African Americans) showed no difference in time to median presentation between groups (1 month in both groups). Treatment-naive African Americans presented with worse VA than their non–African American counterparts (logMAR VA at baseline 1.27 [Snellen, 20/372] vs 0.94 [Snellen, 20/174], $P = .002$) and were more likely to have fovea-involving intraretinal hemorrhage (56.4% vs 41.1%, $P = .03$). A higher proportion of African Americans had a CRVO classified as ischemic (53.7% vs 34.9%, $P = .016$) during the first year of follow-up. There were no differences between the racial groups in treatment-naive patients with respect to the following at baseline: CME, SRF, and macular ischemia on FA. Among treatment-naive patients with 12 or more months of follow-up, African Americans received more intravitreal anti-VEGF injections in the first year of treatment than their non–African American counterparts (mean, 5.5 vs 4.2 injections, $P = .02$); final VA was slightly worse in African Americans than in the non–African American group, but this difference was not statistically significant (logMAR VA 1.30 [Snellen, 20/400] vs 1.03 [Snellen, 20/214], $P = .077$).

Conclusions

United States Census Bureau data estimate the population of North Carolina to be 62.8% white, 22.2% African American, 9.6% Hispanic, and 7.2% other races.[13] In this study, the distribution of patients presenting with CRVO was reflective of the racial distribution in our direct catchment population, with no increased prevalence of CRVO in any particular group. This is consistent with studies from pooled data that have shown that African Americans have a similar incidence of CRVO compared with white populations.[10] A recent large, longitudinal cohort study based on billing codes has linked African American race to a higher risk of incident CRVO[14]; this association was not corroborated in our data but could be explained by higher prevalence of predisposing factors such as diabetes mellitus, hypertension, and open-angle glaucoma in African Americans.

To our knowledge, this is the first study examining the differences in presenting characteristics, treatment course, and outcomes between African American patients diagnosed with CRVO and patients of other racial groups. In our study, we found that African American patients with CRVO presented with worse characteristics at baseline, specifically with significantly worse VA and higher rates of foveal intraretinal hemorrhage, and tended to present earlier to a tertiary center. Additionally, among treatment-naive patients, African Americans received a greater number of anti-VEGF injections during the first year of follow-up and were more likely to have an ischemic CRVO.

Although the exact pathogenesis of CRVO remains unknown, histopathological studies of eyes with CRVO have shown thrombus formation posterior to the lamina cribrosa.[15] At the lamina cribrosa, the optic nerve narrows significantly from approximately 4 mm to 1 mm, and at this point, the luminal diameter of the central retinal vein is at its narrowest.[16] Information from studies of eyes with open-angle glaucoma have demonstrated racial differences in anatomical structure of the optic nerve head[17] and lamina cribrosa deformability[18] between individuals of African and European descent. Since the lamina cribrosa is presumed to be the primary site of pathology in CRVO, these variations may also explain the differences in the presenting characteristics of CRVO in African-Americans compared to other races.

Despite worse presenting characteristics at baseline, African American patients were less likely to have received prior treatment before presenting to a tertiary care center. This may be explained in part by potential disparities in health-care access and use among different racial groups and socioeconomic classes.[19] Despite this, final VA was comparable in both groups, although a statistically nonsignificant trend toward worse final VA was present in African Americans.

Higher rates of diabetes mellitus and hypertension in African Americans observed in our study are consistent with rates reported elsewhere.[20,21] When examining patients with CRVO who are of African descent, clinicians should be aware of the importance of systemic assessment and control of such risk factors, particularly diabetes, because these may have a measurable impact on the risk of progression to neovascular sequelae.

Our study is limited by its retrospective nature and lack of baseline FA data on all patients. Additionally, we did not assess the impact of socioeconomic factors on clinical outcomes in this cohort. Nevertheless, this article provides valuable information regarding the presenting features and clinical course in African American individuals with CRVO. Of note, our understanding and management of CRVO evolved during this study period (2008-2016); the use of anti-VEGF has increased in the years since the CRUISE[22] and BRAVO[23] trials were published in 2010 and their extension trial HORIZON[24] in 2012, as has the use of corticosteroids for treatment of macular edema since the SCORE-CRVO[25] and GENEVA[26,27] trials in 2009 and 2010, respectively.

Patients who presented in the early years of our study received 4 or fewer intravitreal anti-VEGF (mostly ranibizumab) injections during the first year of treatment. In contrast, patients who presented from 2013 to 2016 received 7 to 13 anti-VEGF injections in the first year of treatment. We now know that a delay in initiating treatment with intravitreal anti-VEGF leads to lower gains in VA. Moreover, as shown in the HORIZON[24] and GALILEO[28] trials, improvement in VA that is achieved with monthly monitoring and treatment is unlikely to be sustained with quarterly monitoring and as-needed treatment. The combination of delayed anti-VEGF treatment and less-than-monthly dosing may explain in part the poorer VA outcomes in our study, particularly in patients presenting between 2008 and 2012.

In conclusion, African American individuals with CRVO presented with worse VA and more systemic and ocular risk factors for CRVO than their non–African American counterparts. Although visual outcomes were similar, treatment-naive African American patients had a greater treatment burden during the first year of follow-up and greater rates of ischemic CRVO than other racial groups. Additionally, African Americans were more likely to have a subsequent RVO event in the fellow eye, which may be secondary to higher rates of CRVO risk factors such as diabetes, hypertension, and open-angle glaucoma. The clinical course of African American patients may be distinct from other racial groups, and appropriate counseling and follow-up to address these differences may be warranted.

Authors' Note

S.Y.K. and G.N.T. contributed equally to this work and are co-first authors.

Ethical Approval

This retrospective cohort study was conducted following approval by the institutional review board of the Duke University School of Medicine (approval number Pro00075701).

Statement of Informed Consent

Informed consent was not sought for the present study because this was a retrospective study involving analysis of anonymized, nonidentifiable data. Waiver of informed consent was obtained from the Duke University School of Medicine Institutional Review Board prior to performing this study.

Declaration of Conflicting Interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: S.F. receives patent royalties from Alcon and is a consultant to Regeneron. The other authors have nothing to declare.

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