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Racial Differences in Idiopathic Intracranial Hypertension

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Abstract

Objective—To evaluate racial differences in idiopathic intracranial hypertension (IIH).

Methods—Medical records of all consecutive patients with definite IIH seen between 1989 and 2006 were reviewed. Demographics, associated factors, and visual function at presentation and follow-up were collected. Black patients were compared to non-black patients.

Results—We included 450 patients (197 black, 253 non-black). Obesity, systemic hypertension, anemia, and sleep apnea were more common in blacks than in non-blacks ($p \leq 0.01$). Cerebrospinal fluid opening pressure was higher in blacks (40 vs. 34 cm CSF, $p < 0.001$). Visual acuity, visual field loss, and degree of papilledema at presentation and follow-up were worse in blacks ($p \leq 0.01$). Diagnostic and therapeutic measures were similar between blacks and non-blacks, except for optic nerve sheath fenestration ($p = 0.01$) and lumbar puncture ($p = 0.03$), both more commonly performed on black patients. The relative risk of severe visual loss for blacks compared with non-blacks was 3.5 (95% CI 2.0–5.8, $p < 0.001$) in at least one eye and 4.8 (95% CI 2.1–10.9, $p < 0.001$) in both eyes. Logistic regression analysis supported race, anemia, body mass index, and male gender as independent risk factors for severe visual loss and suggested that racial differences may be partially accounted for by differences in CSF opening pressure, body mass index, and frequency of anemia.

Conclusion—Black IIH patients were more likely than non-black IIH patients to have severe visual loss in at least one eye. This difference did not appear to result from diagnosis, treatment, or access to care, but may partially relate to differences in other risk factors. Black patients have a more aggressive disease and may need closer follow-up and lower thresholds for early intervention.

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Dr. Bruce has nothing to disclose.
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Introduction

Idiopathic intracranial hypertension (IIH) is relatively common in young obese women regardless of ethnic background.^{1–7} Prognosis is variable, with severe visual loss occurring in up to 10% of cases.⁸ Although few series have specifically evaluated racial differences in IIH, it has been suggested that black patients may have more severe outcomes from IIH.^{9–11} The purpose of this study was to compare the characteristics of IIH in blacks and non-blacks.

Methods

The study was approved by our university's Institutional Review Board. All consecutive charts for patients given the diagnosis code of IIH or disc edema seen by our neuro-ophthalmology service between 1989 and 2006 were identified and reviewed. Only patients with definite IIH diagnosed according to the modified Dandy criteria were included: 1) signs and symptoms of increased intracranial pressure, 2) no localizing signs except abducens nerve palsy, 3) CSF opening pressure ≥ 25 cm with normal CSF composition, and 4) normal neuroimaging (ruling out venous sinus thrombosis).¹²

Although the study was a retrospective chart review, all patients had been evaluated in a standardized fashion by experienced neuro-ophthalmologists (NN and VB), including documentation of body habitus, blood pressure, and complete neuro-ophthalmic examination with formal visual fields, fundus photography, review of neuroimaging tests, and recording of factors associated with IIH. Demographic information regarding age, gender, and race were collected. Race was assessed by the judgment of the examiner based on patient appearance. When the examiner was uncertain of the patient's race, the patient was asked to report their race. Medication use (current and recent), the presence or absence of several associated factors (recent weight gain, known sleep apnea, anemia (hemoglobin < 12 g/dL), systemic hypertension, endocrine disorders, and pregnancy), symptoms (headache, tinnitus, diplopia, and transient visual obscuration), Snellen visual acuity, formal visual field (Humphrey automated perimetry or Goldmann perimetry), and dilated funduscopy appearance were recorded. Contributing medications considered included vitamin A preparations, minocycline, cyclosporine, doxycycline, tetracycline, and recent discontinuation of steroids. The contributing medications were grouped by their presence or absence in each patient for analysis. Degree of obesity was rated by clinical impression or body mass index (BMI) into four categories corresponding to World Health Organization BMI cutoff points: underweight (< 18.5), normal ($18.5–24.9$), overweight ($25–29.9$), or obese (≥ 30).¹³ Pre-diagnosis duration of symptoms, CSF opening pressure, height, weight, medical treatments, surgical treatments, follow-up duration, and visual outcome were also recorded.

Snellen visual acuity was converted to logMAR visual acuity for analysis. Formal visual fields were systematically reviewed for all patients. Humphrey visual field mean deviations were recorded. All visual field defects (both Humphrey and Goldmann) were graded on a 1 to 4 scale as 1) normal; 2) enlargement of the blind spot; 3) nasal or temporal defect; or 4) diffusely constricted. Papilledema was graded with the Frisen staging scheme¹⁴ by systematic review of fundus photography: stage 0 defines a normal optic nerve head and stage 5, severe papilledema. Severe visual loss in an eye was defined by the United States criteria for legal blindness (best corrected visual acuity less than or equal to 20/200 or total central visual field less than 20 degrees) and assessed at the last available visit. Patients were divided into two groups based on race for statistical comparisons, with all non-black patients grouped together for statistical analysis.

All patients had definite IIH by the modified Dandy criteria, but two aspects of our population merit further mention. Although all patients received a lumbar puncture with documented elevation of CSF opening pressure, the specific value was sometimes unavailable. Clinically appropriate neuroimaging was available or performed for all patients to rule out cerebral venous thrombosis. However, as the patient population is representative of an actual clinical practice, there were occasionally practical limitations to obtaining ideal imaging studies, such as body habitus preventing entry into imaging gantries and changes in the clinical usage of MRI and MR venography over the study period.¹⁵ MRIs were all reviewed at the time of diagnosis and MR venography or CT venography was obtained when there was a doubt regarding possible cerebral venous thrombosis. Those patients who could not have a MRI, had a head CT with contrast, often associated with CT venography.

Statistical analysis was performed with R: A language and environment for statistical computing (R Foundation for Statistical Computing, <http://www.R-project.org>). Continuous and ordinal variables were compared between groups using the Mann-Whitney U test. Pearson's Chi Square with Yates' continuity correction or Fisher's exact test, as appropriate, were used to compare the frequency distribution of categorical variables between groups. These tests were two tailed, and significance was set at 5%. Univariate analyses for race and severe visual loss in at least one eye versus other factors were undertaken. Multiple imputation of missing data was performed using a fully conditional specification model ($m=100$) with race, gender, age, sleep apnea, anemia, hypertension, pre-diagnosis duration, body mass index, CSF opening pressure, duration of follow-up, and severe visual loss in at least one eye as covariates.¹⁶ With severe visual loss in at least one eye as the outcome, the effect of race was assessed when adjusted with logistic regression individually by each of the factors found significant in the univariate analyses of race or severe visual loss in at least one eye. A logistic regression analysis was also performed with outcome severe visual loss in at least one eye with all of the significant variables from the univariate analyses of race and severe visual loss included as predictors. In this model, significance at the 5% level was used as the cutoff for predictor inclusion, and each predictor's effect on race was studied by removing it individually from the final, reduced model. Race, gender, age, sleep apnea, anemia, hypertension, pre-diagnosis duration, body mass index, CSF opening pressure, duration of follow-up, and severe visual loss in at least one eye were the variables studied by the logistic regression models.

Results

We included 450 consecutive patients with definite IIH seen over a 17 year period. Age ranged between 2 and 62 years with a mean of 29 years (standard deviation 11). There were 408 (91%) women and 42 (9%) men. There were 246 (55%) white, 197 (44%) black, 5 (1%) Hispanic, and 2 (<1%) Asian patients.

Table 1 details the characteristics of black and non-black patients. Black patients were older (median: 30 vs. 27 years, $p=0.02$) and were more obese than their non-black counterparts (median BMI: 39.8 vs. 34.2, $p<0.001$). Blacks had higher rates of several medical conditions: anemia (11% vs. 2%, $p<0.001$), sleep apnea (9% vs. 4%, $p=0.01$), and hypertension (32% vs. 11%, $p<0.0001$). There was no difference in the frequency of initial symptoms of increased intracranial pressure between blacks and non-blacks. However, black patients reported less headache at initial neuro-ophthalmology evaluation (85% vs. 91%, $p=0.045$). Pre-diagnosis duration of symptoms trended toward significance and was shorter among black patients (median: 4 vs. 8 weeks, $p=0.066$), and follow-up duration was shorter among black patients (median: 14 vs. 22 weeks, interquartile range 4–47 vs. 7–55 wks, $p=0.01$).

A reliable value of CSF opening pressure was available on 347 patients (77%), and CSF opening pressure was higher in blacks than non-blacks (median: 40 vs. 34 cm CSF, $p<0.001$). MRI was obtained in 93% of all included patients. Those who were unable to undergo MRI, had a head CT with contrast, often with CT venography. MR venography or CT venography was obtained on 24% of black and 23% non-black patients.

Regarding treatment, there were no significant differences regarding medication use, dietary changes, or CSF shunting procedures (Table 1). Repeat lumbar punctures (median: 2 vs. 1, $p=0.03$) and optic nerve sheath fenestration (unilateral: 15 vs. 14, bilateral: 17 vs. 7, $p=0.01$) were more frequently performed in blacks than non-blacks. Clinically, visual acuity (median: 0.11 vs. 0.00 logMAR, $p=0.002$), visual fields ($p=0.003$), and papilledema ($p<0.0001$) were worse among black patients at initial visit (Table 2). Although all of these factors improved in both groups at follow-up, they remained worse among blacks: visual acuity (median: 0.11 vs. 0.00 logMAR, $p=0.01$), visual fields ($p<0.0001$), and papilledema ($p=0.0003$).

Twenty three percent of blacks and seven percent of non-blacks experienced severe visual loss in at least one eye. The unadjusted relative risk of severe visual loss for blacks vs. non-blacks was 3.5 (95% confidence interval (CI): 2.0–5.8, $p<0.001$) for at least one eye and 4.8 (95% CI: 2.1–10.9, $p<0.001$) for both eyes. The unadjusted odds ratio (OR) of severe visual loss in at least one eye for blacks vs. non-blacks was 4.1 (95% CI=2.3–7.6, $p<0.001$).

A univariate analysis of the patients with severe visual loss in at least one eye compared to those without was performed. With regard to potential risk factors, patients who were black ($p<0.001$), men ($p<0.001$), more obese ($p=0.006$), or who had sleep apnea ($p=0.002$), anemia ($p<0.001$), or hypertension ($p<0.001$) were more likely to have severe visual loss. Two symptoms were less frequently reported by patients with severe visual loss at initial neuro-ophthalmology evaluation: pulsatile tinnitus (26% vs. 39%, $p=0.05$) and diplopia (8% vs. 21%, $p=0.01$). Cerebrospinal fluid opening pressure was higher among patients with severe visual loss (median: 41 vs. 36 cm CSF, $p=0.01$). Surgical procedures were more frequent among patients with severe visual loss: CSF shunting procedures (39% vs. 17%, $p<0.001$) and optic nerve sheath fenestrations (48% vs. 6%, $p<0.001$).

When comparing patients with and without severe visual loss in at least one eye, we found no difference in the frequency of headache as the initial symptom (18/62=71% vs. 294/388=76%, $p=0.42$) or at first neuro-ophthalmic evaluation (58/62=94% vs. 341/388=88%, $p=0.19$). There was also no difference between the frequency of headache between black patients with severe visual loss and non-black patients with severe visual loss (42/45=93% vs. 16/17=94%, $p=0.91$).

To control for the differences in potential risk factors and modifying factors between blacks and non-blacks, logistic regressions were performed. Race remained a significant risk factor for severe visual loss when it was adjusted individually by each of the other factors considered in the analysis (OR 3.6–4.3). When all factors were considered together, race (OR 2.7 black vs. non-black, 95% CI=1.4–5.3, $p=0.003$), anemia (OR 4.0 present vs. absent, 95% CI=1.5–10.4, $p=0.005$), body mass index (OR 1.23 per 5 unit increase, 95% CI=1.05–1.47, $p=0.01$), CSF opening pressure (OR 1.16 per 5 cm CSF increase, 95% CI=1.10–1.40, $p=0.04$), and gender (OR 4.8 men vs. women, 95% CI=1.6–14.1, $p<0.001$) were independent risk factors for severe visual loss. Longer follow-up duration was also associated with a greater risk of severe visual loss (OR 1.02 per month, 95% CI=1.004–1.04, $p=0.03$). Individual removal of gender and follow-up duration from the model had minimal influences on the effect of race (OR for race 2.69–2.71 without each of these factors), while anemia, body mass index, and CSF opening pressure had larger influences (OR for race

3.12–3.33 when each of these factors was removed from the model individually). This suggests that the higher frequency of anemia, higher CSF opening pressure, and higher BMI among black patients partially accounts for their increased risk of severe visual loss.

Discussion

We present a large series of IIH patients in which we found that black patients were 3.5 times more likely than non-black patients to have severe visual loss in at least one eye, and nearly five times as likely to be legally blind by U.S. criteria. Although race had been previously suspected to be an important risk factor in IIH, few studies have evaluated blacks in IIH.^{9–11} A study of IIH among men⁹ had four black patients, three of whom had severe visual loss and all of whom were hypertensive, suggesting that “black male hypertensives may represent a more serious situation and may require earlier surgical intervention before they begin to lose vision”. Two later studies failed to find such an association.^{10,11} A prospective study of 50 IIH patients, including 31 blacks and 19 non-blacks, did not find race to be a significant factor for the deterioration of visual function.¹¹ Similarly, a retrospective study of 77 IIH patients (50 black, 24 non-black, 3 race unknown) found no significant race differences with regard to symptoms, CSF opening pressure, or visual outcome.¹⁰ However, the size of both of these studies was likely underpowered to find such a relationship, if present.

Previous studies have suggested many other potential associations with the development of IIH such as female gender, obesity, use of certain medications, pregnancy, and endocrine disorders.⁸ Other studies have suggested that male gender, hypertension, recent weight gain, anemia, sleep apnea, puberty, and older age may be associated with poorer outcomes, but no consistent risk factors have emerged.^{9, 11, 17–23} The analyses in this study regarding factors associated with severe visual loss support black race, male gender, hypertension, higher degrees of obesity, anemia, and sleep apnea as associated with poorer outcomes. Interestingly, diplopia and pulsatile tinnitus were less frequently reported among patients with severe visual loss from IIH at their initial neuro-ophthalmology evaluation. Although it is not surprising that patients who had severe visual loss in one or both eyes would not experience diplopia, it is unclear why pulsatile tinnitus would occur less frequently.

Because many associated factors were more common or more severe among the black patients in our study (Table 1), we hypothesized that some may have partially or fully accounted for the worse visual outcomes experienced by black patients. Thus, a multivariate analysis was undertaken to control for the interaction between these variables and showed that race, gender, CSF opening pressure, body mass index, and anemia were significant independent risk factors for the development of severe visual loss. The analysis also suggested that the higher frequency of anemia, higher body mass indexes, and higher CSF OP among black patients in our study may have partially accounted for the more frequent visual loss the black patients experienced. Interestingly, another study¹⁰ also found mean CSF opening pressure to be higher among black patients by 2.8 cm compared with white patients, although the difference was non-significant.

Although previous studies have suggested that systemic hypertension may be a poor prognostic indicator,^{9, 11} it was not found to be a significant factor for severe visual loss in our study after controlling for other variables. Because many of the patients in our study were treated hypertensives, this relationship may have been masked. In addition, we considered hypertension only by its presence or absence and not by a numerical value such as mean arterial pressure. This also reduces the power of finding a potentially significant relationship in this study. However, five patients in this study had severe, uncontrolled hypertension. All were black, and 4/5 (80%) were women. Four suffered severe visual loss

in both eyes and one recovered with moderate visual loss (20/40 OD, 20/30 OS), suggesting that severe, uncontrolled hypertension may indeed be a risk factor for severe visual loss in IIH.

Race studies can be easily criticized because they fail to control for socio-economic and other factors that may account for differences seen between races. However, several factors in this study were contrary to these potential criticisms. Our high percentage of black patients is reflective of Atlanta's population (61% black in the city proper and 29% black when including the metropolitan area²⁴) and is consistent with other studies' proportion of black patients which also mirror the underlying demographics of their catchment areas (Iowa⁹, Detroit¹⁰, and New Orleans¹¹). Time to diagnosis was actually longer in non-blacks than in blacks, and medical conditions, such as systemic hypertension, anemia, and sleep apnea, were significantly more common in black than in non-black patients, suggesting black patients had adequate access to primary and specialty care. Although median follow-up was longer among non-blacks than blacks, multivariate analysis revealed this factor was of small magnitude overall and had little influence upon the difference in visual outcomes between non-blacks and blacks. Finally, there were no differences in the application of diagnostic modalities, such as MRI and MRV.

Another potential concern would be that blacks may suffer severe visual loss at a higher rate because they did not experience noticeable symptoms such as headache to bring their visual loss to medical attention earlier. Although blacks had less frequent headache at the first neuro-ophthalmology visit, they had equal rates of headache as a first symptom of the disease, and there was no difference in headache frequency among the patients with severe visual loss and those without.

Finally, there were no significant differences between our black and non-black IIH patients with regard to treatment, with the exception of the frequency of lumbar puncture and optic nerve sheath fenestration procedures. However, in our practice, optic nerve fenestration is reserved for patients with severe visual loss, and repeat lumbar punctures are frequently performed as an immediate measure to reduce CSF opening pressure among more severely affected patients. Thus, these differences likely represent surrogate markers for the more severe visual loss that black patients experienced. This was supported by the univariate analysis of factors related to severe visual loss. One might also be concerned that visual complications from optic nerve fenestration may have biased the results, but only four patients had significant visual loss related to optic nerve fenestration and three of these patients were not black.

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Demographics, risk factors, CSF opening pressure, and treatment of black and non-black patients with idiopathic intracranial hypertension

Table 1

Demographics / Risk Factors	Black (n=197)		Non-Black (n=253)		p
<i>Age (years)</i>	<i>n or median</i>	<i>% or range</i>	<i>n or median</i>	<i>% or range</i>	
	30	(9–56)	27	(2–62)	0.02
<i>Gender (M/W)</i>	18/179	(9%/91%)	24/229	(9%/91%)	0.90
<i>Contributing Medications</i>	11	(5%)	20	(8%)	0.33
<i>Sleep Apnea</i>	18	(9%)	9	(4%)	0.01
<i>Anemia</i>	21	(11%)	5	(2%)	<0.001
<i>Pregnancy</i>	4	(2%)	10	(4%)	0.24
<i>Endocrine disorder</i>	25	(13%)	28	(11%)	0.60
<i>Hypertension</i>	64	(32%)	28	(11%)	<0.0001
<i>Pre-diagnosis duration of symptoms (wks, n=395)</i>	4	(0–500)	8	(0–300)	0.066
<i>Follow-up duration (wks, n=447)</i>	14	(1–380)	22	(1–360)	0.01
CSF opening pressure (cm CSF, n=347)	40	(25–75)	34	(25–65)	<0.001
Weight					
<i>Weight category, first visit (n=374)</i>	0.006				
Underweight	1	(1%)	7	(3%)	
Normal	5	(3%)	22	(10%)	
Overweight	17	(10%)	31	(14%)	
Obese	136	(86%)	155	(72%)	
<i>Weight category, last visit (n=295)</i>	0.007				
Underweight	0	(0%)	3	(2%)	
Normal	7	(6%)	24	(14%)	
Overweight	9	(7%)	24	(14%)	
Obese	107	(87%)	121	(70%)	
<i>Body mass index, first visit (n=250)</i>	39.8	(22.9–83.6)	34.2	(12.6–69.9)	<0.001
<i>Body mass index, last visit (n=214)</i>	39.8	(23.2–83.6)	31.9	(13.6–62.9)	<0.001
<i>Recent weight gain present</i>	37/70	(52%)	58/115	(50%)	0.75

Demographics / Risk Factors	Black (n=197)		Non-Black (n=253)		p
	n or median	% or range	n or median	% or range	
Amount of recent weight gain (lbs, n=75)	40	(10–150)	29	(5–120)	0.10
Treatment					
Medication	190	(96%)	240	(95%)	0.42
Diet modification	41/120	(34%)	67/164	(41%)	0.25
Number of lumbar punctures	2	(1–20)	1	(1–25)	0.03
CSF shunt	37	(19%)	53	(21%)	0.57
Repeat CSF shunt	16/37	(43%)	20/53	(38%)	0.60
<i>Optic nerve sheath fenestration</i>					0.01
Unilateral	15	(7%)	14	(6%)	
Bilateral	17	(8%)	7	(3%)	

n=450 unless otherwise noted, M=men, W=women, wks=weeks

Table 2
Clinical examination of black and non-black patients with idiopathic intracranial hypertension.

	Black (n=197)	Non-Black (n=253)	p
<i>Visual acuity, first visit (logMAR, n=415)</i>	0.11 (-0.29, 3.34)	0.00 (-0.29, -2.65)	0.002
<i>Visual acuity, last visit (logMAR, n=434)</i>	0.11 (-0.29, 4.06)	0.00 (-0.29, -3.00)	0.01
<i>Visual field grade, first visit (n=353)</i>			0.003
1	27 (18%)	48 (24%)	
2	48 (32%)	88 (44%)	
3	27 (18%)	30 (15%)	
4	50 (33%)	35 (17%)	
<i>Visual field grade, last visit (n=425)</i>			<0.0001
1	62 (35%)	125 (50%)	
2	29 (16%)	59 (24%)	
3	36 (29%)	31 (13%)	
4	50 (28%)	33 (13%)	
<i>HVF MD, first visit (n=231)</i>	-4.94 (-0.27, -31.9)	-4.88 (-0.01, -26.6)	0.27
<i>HVF MD, last visit (n=206)</i>	-3.56 (-0.11, -31.5)	-3.40 (-0.14, -17.9)	0.23
<i>Severe visual loss (n = 447)</i>			<0.0001
None	149 (77%)	236 (93%)	
Unilateral	19 (10%)	10 (4%)	
Bilateral	26 (13%)	7 (3%)	
<i>Papilledema, first exam (n=371)</i>			<0.0001
0	1 (1%)	1 (<1%)	
1	5 (3%)	18 (9%)	
2	15 (9%)	46 (22%)	
3	45 (28%)	93 (44%)	
4	54 (33%)	32 (15%)	
5	42 (26%)	19 (9%)	
<i>Papilledema, last exam (n=448)</i>			0.0003

	Black (n=197)	Non-Black (n=253)	p
0	101 (52%)	134 (53%)	
1	31 (16%)	66 (26%)	
2	26 (13%)	30 (12%)	
3	24 (12%)	22 (9%)	
4	9 (5%)	0 (0%)	
5	4 (2%)	1 (<1%)	

n=450 unless otherwise noted, *Severe visual loss=corrected visual acuity less than or equal to 20/200 or total visual field less than 20°, HVE MD=Humphrey visual field mean deviation (dB)