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Racial differences in adult-onset MRI-negative temporal lobe epilepsy

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ABSTRACT

Objective: We recently detected a significant racial difference in our population with temporal lobe epilepsy (TLE) at the University of Alabama at Birmingham (UAB) seizure monitoring unit. We found that Black patients were more likely than their White counterparts to carry a TLE diagnosis. Using this same patient population, we focus on the patients with TLE to better describe the relationship between race and epidemiology in this population.

Methods: We analyzed the data from patients diagnosed with TLE admitted to the UAB seizure monitoring unit between January 2000 and December 2011. For patients with a video electroencephalography (EEG) confirmed diagnosis of TLE (n = 385), basic demographic information including race and magnetic resonance imaging (MRI) findings were collected. Descriptive statistics and multivariate logistic regression were used to explore the relationship between MRI findings, demographic data, and race.

Results: For Black patients with TLE, we found that they were more likely to be female (odds ratio [OR] = 1.91, 95% confidence interval [CI]: 1.14–3.19), have seizure onset in adulthood (OR = 2.39, 95% CI: 1.43–3.19), and have normal MRIs (OR = 1.69, 95% CI: 1.04–2.77) compared to White counterparts with TLE after adjusting for covariates.

Conclusions: These data suggest that Black race (compared to White) is associated with higher expression of adult-onset MRI-negative TLE, an important subtype of epilepsy with unique implications for evaluation, treatment, and prognosis. If validated in other cohorts, the findings may explain the lower reported rates of epilepsy surgery utilization among Blacks. The racial differences in surgical utilization could be due to a greater prevalence of an epilepsy that is less amenable to surgical resection rather than to cultural differences or access to care.

1. Introduction

Several epidemiologic studies have documented a higher prevalence of epilepsy among non-White patients as compared to White patients [1–5], yet we lack an explanation. The underpinnings of these racial differences in epilepsy, similar to other racially skewed morbidities, are undoubtedly multifactorial, likely involving an array of genetic, biological, and social factors; however, an exploration of factors remains to be undertaken [6]. Previously, most epilepsy-based epidemiologic work has been conducted through population surveys or insurance databases, a study design that is not amenable to exploring important differences in classification and etiology. It is effectively only possible to obtain accurate epilepsy type diagnoses with detailed history, video electroencephalography (EEG) seizure monitoring, and neuroimaging.

We recently reported on a significant racial difference in the population with temporal lobe epilepsy (TLE) in the University of Alabama at Birmingham (UAB) seizure monitoring unit. We found that Blacks within a 10-year period (2000–2011) were much more likely than their White counterparts to carry a TLE diagnosis. Using this same patient population, we focus on the patients with TLE of all races to better describe the epidemiology of this subsection of the population. Through this more refined investigation, we seek to explore the possibility of any systematic differences among racial groups to help explain the different rates of diagnosis at UAB. While we are still far from describing large-scale racial differences in epilepsy with confidence, any information to enable understanding of the mechanisms for these racial disparities will impact not only our ability to further investigate these differences, but also to address and develop strategies to minimize them.

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Temporal lobe epilepsy, a focal epilepsy often due to structural lesion, is an important syndrome of interest which deserves further exploration. Temporal lobe epilepsy is difficult to control adequately with medical therapies, and patients often require surgical evaluation. While surgery has been shown to vastly improve quality of life [7], the morbidity associated with a prolonged period of seizure intractability, the burden of surgical evaluation, the surgery itself, and the postoperative period cannot be ignored. Indeed, it should be of no surprise that TLE treatment represents a disproportionate cost of the overall treatment of epilepsy in the United States [8]. Given this large expense, it would be concerning if one of our most resource-poor communities, our Blacks patients [9], were disproportionately shouldering this burden. While our initial examination of the data provided no obvious reason to explain these disparities, we seek to explore the data further, now including the magnetic resonance imaging (MRI) brain results of our patients with TLE in an attempt to explain these differences. Our guiding hypothesis was that imaging could provide further clues as to the etiology of such disparities.

2. Methods

We analyzed the data from a subset of patients admitted to the UAB seizure monitoring unit between January 2000 and December 2011. Data were analyzed for only patients with TLE who also received MRIs ($n = 385$). A TLE diagnosis was defined by patient history and was confirmed through video-EEG monitoring. Temporal lobe epilepsy was diagnosed if a patient's habitual seizures had initial ictal EEG findings reflecting F7/8, FT9/10, or SP1/2 voltage maximums, and semiology congruent with EEG localization.

We further focused on all patients who received a diagnosis of TLE and then selected patients for whom MRI of the brain was obtained using a specific UAB epilepsy protocol. Magnetic resonance imaging were not obtained in patients who already had MRIs or MRI would not change management. Patients whose scans lacked certain sequences were also excluded. The vast majority of patients initially received an MRI scan and were subsequently diagnosed with TLE at a hospital outside of UAB. Repeat MRIs were only conducted in a subset of patients who were considered for or expressed interest in epilepsy surgery.

All patients underwent MRI studies at either 1.5 ($n = 191$) or 3.0 ($n = 194$) Tesla field strength according to protocols designed by UAB epilepsy imaging group for patients suspected of having focal epilepsy. The 1.5-T scanner was updated to 3.0 T in July 2004. In addition to routine spin-echo sequences, the following volumetric and high-resolution scans were acquired: (1) coronal or sagittal 3D T1-weighted gradient echo (slice thickness, 1.0–1.5 mm), (2) coronal oblique (orthogonal to long axis of the hippocampus) T2-weighted fast fluid-attenuated inversion recovery (slice thickness, 3.0 mm; gap, 1.0 mm), and (3) coronal oblique inversion recovery sequence with a 512×512 matrix and a reconstructed voxel size of $0.5 \times 0.5 \times 3.0$ mm (gap, 1.0 mm).

All of the MRIs were read by either one of two senior epilepsy specialists (RIK and RCK) with board certifications in MRI reading through the American Society of Neuroimaging. The classification system for categorizing MRIs was based on the National Institute of Neurological Disorders and Stroke (NINDS) Common Data Elements for MRI and Epilepsy standards for reporting. Magnetic resonance imaging categories included "normal," "questionable," "multiple," "ambiguous," "localized," or "inadequate." "Normal" scans had no abnormalities. Abnormal features included agenesis, atrophy, cortical thinning, cystic changes, decreased gray-white distinction, dysgenesis, heterotopic tissue, hypertrophy, hyperplasia, hypoplasia, loss of architecture, or alternative malformation [10]. Scans categorized as "localized" had one of the aforementioned abnormalities, whereas scans categorized as "multiple" had more than abnormality. "Inadequate" scans were uninterpretable because of poor image quality, most often due to motion artifact. "Questionable" was used to describe an indeterminate study where findings were not diagnostically conclusive, and finally, the term "ambiguous" was used to describe scans with lesions that were too extensive to implicate a single localization. Examples lesions that would meet the ambiguous criteria include a multilobar malformation or a large territory infarct. Overall, interpretations were broken down into the following three categories based on the standards for reporting of diagnostic accuracy (STARD) guidelines: (1) nonlesional or "normal" scans, (2) indeterminate or "questionable" scans, and finally, (3) lesional scans including "multiple," "ambiguous," and "localized" [11].

Other variables included in this analysis were race, age of onset, age at the time of monitoring, and sex. Race was self-reported and was defined as non-Hispanic White, Black, other, and not selected. Age of onset refers to the age of the patient at the time of first seizure, and age at the time of monitoring refers to the age of the patient at the time of admission to the epilepsy monitoring unit (EMU) at UAB. Age was treated as a categorical variable in increments of 10 years in the descriptive portion of the analysis and then was consolidated into two categories in the multivariate analysis to distinguish between adult and childhood/young adult onset. Patients age 19 years and older at the time of onset ("19+") are considered the adult-onset population, and patient from 0 to 18 years of age or "<19" is considered the childhood/young adult-onset population.

To answer the question of racial difference, the final analysis was confined to patients with self-identified race as either White or Black. Patients identifying as "other" or patients who did not select a race were excluded. For simplicity, MRI reads were further consolidated into the following two categories: "normal" or "abnormal." "Normal" scans included scans read as "normal" or "questionable." "Abnormal" scans included those read as "multiple," "ambiguous," or "localized." "Inadequate" scans were excluded. The multivariate analysis explores the relationship between MRI findings and age of onset, race, and sex. Initially, a univariate analysis was conducted to calculate odds ratios (ORs) with 95% confidence intervals (CIs) to identify independent associations. Variables with a significant association in the univariate analysis were then adjusted for in the multiple logistic regression analysis. All data analysis was performed using STATA version 12.0 software (StataCorp). The study proposal was reviewed and approved by the research and ethics committees of the UAB Institutional Review Board.

3. Results

All patients with TLE and available epilepsy protocol MRI results who were evaluated in the UAB seizure monitoring unit between January 2000 and December 2011 were included in this analysis ($n = 385$). Patients without available MRI results were excluded ($n = 212$).

Approximately half the population had epilepsy onset prior to 20 years of age with a mean age of onset of 22 years of age for the population as a whole. Women represented 57% of the entire sample. The population is predominately White (71%), with a quarter of the population identifying as Black and a small minority as "other" or "not selected" (3.4%). This is representative of the racial makeup of the population in the State of Alabama (72.6% White, 26.3% Black, 1.1% other).

Approximately one-third of the patients had normal MRIs, and just under half (48%) had identifiable lesions. The remainder (23%) had "questionable" or "ambiguous" MRIs, with only one remaining MRI read as "inadequate" (Table 1). Black and White patients had roughly equivalent MRI reads by category with the exception of normal results. Black patients with TLE were much more likely than their White counterparts to have a normal MRI interpretation ($p = 0.002$), whereas White patients were much more likely to have an abnormal interpretation ($p = 0.001$; Table 2). In the multivariate adjusted analysis, the OR for a normal MRI in Blacks as compared to White patients was 1.69 (95% CI: 1.04–2.77; Table 3).

Blacks were also more likely to have adult-onset epilepsy (19 years of age or more at the time of onset) than White patients (71% [95% CI: 61.8–79.2] as compared to 46% [95% CI: 40.4–52.2] respectively), and

Table 1
Population characteristics by race.

	Black no. (%)	White no. (%)	Total
Age of onset (years); mean (SD) = 22.0 (16.5)			
<10	9 (8.9)	81 (30.0)	94 (24.4)
10–19	20 (19.8)	71 (26.3)	97 (25.2)
20–29	17 (16.8)	51 (18.9)	70 (18.2)
30–39	29 (28.7)	25 (9.3)	55 (14.3)
40–49	20 (19.8)	16 (5.9)	36 (9.4)
50+	6 (5.9)	26 (9.6)	33 (8.6)
Duration of epilepsy (years)			
0–2	24 (23.8)	36 (13.4)	63 (16.4)
3–5	15 (14.6)	32 (11.9)	47 (12.2)
6–9	23 (22.8)	31 (11.5)	56 (14.6)
10–19	18 (17.8)	64 (23.8)	83 (21.6)
20–29	9 (8.9)	40 (14.9)	51 (13.3)
30+	12 (11.9)	66 (24.5)	84 (21.9)
Sex			
Female	72 (71.3)	144 (53.3)	221 (57.4)
Male	29 (28.7)	126 (46.7)	164 (42.6)
MRI			
Normal	45 (44.6)	74 (27.4)	122 (31.7)
Questionable	12 (11.9)	28 (10.4)	42 (10.9)
Multiple	8 (7.9)	29 (10.8)	46 (12.0)
Ambiguous	7 (6.9)	35 (13.0)	37 (9.6)
Localized	28 (27.7)	104 (38.5)	137 (35.6)
Inadequate	1 (1.0)	0 (0)	1 (0.3)
Total	101 (26.2)	270 (70.1)	385[a]

Abbreviations: No = number, SD = standard deviation.
[a] Includes patients who self-identified racially as "other" or "not selected".

more likely to be female than White patients (72% [95% CI: 61.8–79.2] as compared to 53% [95% CI: 47.4–59.1]; Fig. 1). In the multivariate adjusted analysis, Blacks with TLE had a 2.4-fold higher odds (OR: 2.39; 95% CI: 1.43–43.19) of adult-onset epilepsy as compared to White patients and a 1.91 OR (95% CI: 1.14–3.19) of being female as compared to White patients with TLE (Table 3).

4. Discussion

This large cohort study of patients evaluated with seizure monitoring reveals the possibility that Blacks represent a disproportionate prevalence of adult-onset MRI-negative TLE. While other studies have noted a disproportionate burden of epilepsy in older Blacks [12], our data further suggest that there may be systematic differences in their underlying pathology. There are likely multiple reasons for this observed difference. Four possible explanations based on review of published literature include underlying differences in (1) autoimmunity, (2) temporal lobe meningoencephaloceles related to idiopathic intracranial hypertension (IIH), (3) genetic differences, and (4) disparities in care.

Recent research has demonstrated a wide range of adult-onset autoimmune encephalitides causing both acute and chronic epilepsy. Autoimmune mechanisms are thought to account for approximately 20% of epilepsies of unknown etiology [13]. While many of these mechanisms

Table 2
MRI classification by race.

MRI	Total no.	White no. (%)	Black no. (%)	p-Value
Normal	119	74 (27.4)	45 (44.6)	**0.002**
Indeterminate	40	28 (10.4)	12 (11.9)	0.676
Abnormal[a]	211	168 (62.2)	43 (42.6)	**0.001**

Bold = p-Value that is highly statistically significant.
[a] Abnormal includes scans read as ambiguous, multiple, or localized.

Table 3
Associated risk factors for Black patients with TLE.

	Unadjusted OR (95% CI)	Adjusted OR[a] (95% CI)
Age of onset		
<19	1 (ref)	1 (ref)
19+	2.88 (1.76–4.72)	2.39 (1.43–3.19)
MRI		
Normal	2.18 (1.37–3.48)	1.69 (1.04–2.77)
Abnormal	1 (ref)	1 (ref)
Sex		
Female	2.17 (1.33–3.57)	1.91 (1.14–3.19)
Male	1 (ref)	1 (ref)

Abbreviations: OR = odds ratio, CI = confidence interval, ref = reference group.
[a] Adjusted for age of onset, MRI classification, and sex.

have yet to be well described, certain antibodies such as GAD65 (antiglutamic acid decarboxylase 65) have been shown to cause temporal lobe predominant epilepsy with seizures being the main presenting feature [14,15]. The prevalence of epilepsy related to GAD antibody is not well known given its difficulty in diagnosis; and thus, it is strongly suspected that the incidence is higher than reported. While the more fulminant cases of autoimmune encephalitides are more carefully worked up and well described in the literature, much less is known about autoimmune encephalitis with a more insidious onset. A great deal more research is needed to identify actual clinical and causative relationships to otherwise idiopathic adult-onset focal epilepsies, as well as to describe the epidemiology of these conditions, including possible racial differences. Looking at studies of other autoimmune conditions with neurologic predilections such as systemic lupus erythematosus (SLE), known to be common among Black patients [16,17], could it be possible that there are racial asymmetries in autoimmune epilepsy as well? Indeed, the risk factors identified for Blacks with TLE in our cohort mirror what we would expect for an autoimmune epilepsy: female predominance, adult onset, and MRIs without established epileptogenic lesions.

Beyond autoimmunity, another explanation for the observed racial disparity could center on IIH. Idiopathic intracranial hypertension is known to be not only more common but it is also more aggressive in Blacks [18]. While not previously linked to epilepsy, recent research has linked IIH to temporal lobe meningoencephaloceles [19,20], an increasingly recognized likely cause of drug-resistant TLE in a surgical case series [21]. It is possible that a subset of patients with adult-onset MRI-negative epilepsy in fact have developed occult temporal lobe meningoencephaloceles. While this dataset does not allow for exploration of patient comorbidities, a future project could explore the association between IIH and adult-onset MRI-negative epilepsy.

A third possible explanation for these racial differences could go back to different genetic predispositions. A recent genome wide analysis discovered a genetic variance specific to African Americans as compared to European Americans that has been linked to neuropsychiatric disorders including epilepsy and Alzheimer's [22] providing some early evidence that may support a possible genetic difference to explain our observed differences between Black and White patients. Undoubtedly, many more racial genetic differences in predisposition to various epilepsies will be discovered.

Finally, one has to consider whether potential race-based disparities in diagnosis and care might be an important confound in the observed relative diagnostic differences in this study cohort. Differences in epilepsy evaluation and care based on race and associated socioeconomic factors have been identified with regard to access of care, time to diagnosis, specialist referral, and frequency of emergency department visits due to lack of antiseizure medication [6,23]. And, although one of the strengths of this study is the relative lack of barriers to care provided by UAB, we know from analysis of the larger entire EMU cohort and epilepsy clinical patients that barriers must still exist for both urban and rural Black relative to White patients [5]. Yet, we cannot identify any definite systematic bias that would be required to result in a

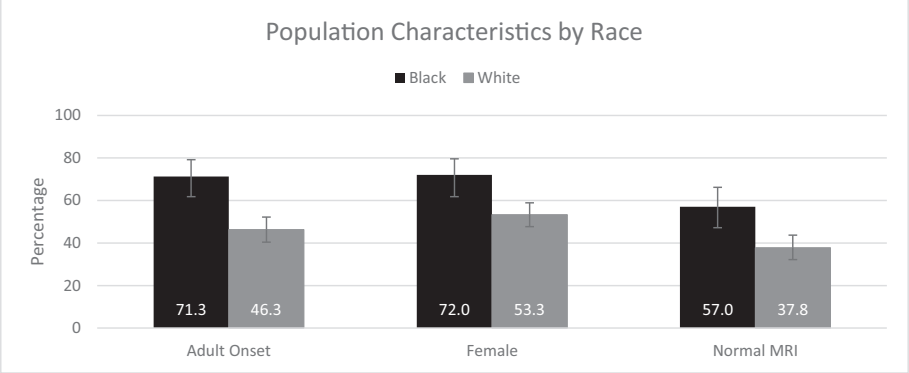

Fig. 1. Population characteristics by race. *Adult onset indicates patients with epilepsy onset after 18 years of age.

significantly greater proportion of cases diagnosed specifically as adult-onset MRI-negative TLE. Moreover, the representation of Black patients in the specific subgroup with TLE of the larger SMU cohort closely mirrored Alabama demographics for race.

Thus, independent of the explanation for these racial differences, our findings suggest that Blacks have a greater predisposition to developing adult-onset idiopathic TLE. And, while previous studies have suggested that Blacks have decreased access to epilepsy surgery [24], our data suggest that Blacks may, in fact, be more likely to have a form of TLE less amenable to surgery, and thus may require a novel approach to treatment. Indeed, the greatest rate of success from surgery has been shown in patients with MRI lesions that are concordant with EEG localization [25]. These patients with visualized MRI concordant lesions are more likely to be seizure-free following epilepsy surgery, than those undergoing surgery with a diagnostically negative MRI [26]. An MRI without a definite epileptogenic lesion should not preclude surgical evaluation [27], but the risk benefit ratio is different for these patients as they are at higher risk for deficits and have lower likelihood of seizure freedom after surgery.

This study has a number of strengths including our large population size, larger than has been studied previously, and one that accurately reflects the racial demographics of the UAB catchment area–the entire state of Alabama and neighboring southeast regions. Unlike most other epidemiologic studies in epilepsy, our data reflect accurate epilepsy type diagnosis from video-EEG monitoring, the gold standard in epilepsy diagnosis. Beyond standardization of diagnosis, our MRI imaging and interpretations were all acquired according to epilepsy specific protocols in a dedicated epilepsy-imaging program at UAB, ensuring high quality and internal consistency in imaging and interpretation.

There are also limitations to note. Patients admitted to the UAB EMU have more often than not medically intractable disease and do not fully represent the epilepsy population as a whole. Magnetic resonance imaging scans were also not obtained on all patients as an MRI is not routinely conducted as part of an EMU admission. Within our cohort, Blacks were less likely than Whites to receive an MRI (67% vs. 58%; $p = 0.04$). Since the EMU did not restrict admissions during the study period–providing service for epilepsy management, diagnosis, and presurgical evaluation–any patients not proceeding toward surgical treatment, or followed regularly in epilepsy center may not have had an epilepsy protocol MRI acquired at UAB. We know from a previous study of the entire population of patients admitted to the EMU during this period that a higher proportion of Blacks presented from rural counties away from UAB [5]. Given this geographic difference, it is likely that some selection bias affected acquisition of epilepsy protocol MRI performed at UAB. Still, despite this difference, the patient characteristics between Black and White patients with and without UAB acquired MRI described in Table 1 are roughly equivalent.

Finally, we acknowledge that nearly half the cases had 1.5-T scans that may be less sensitive to detection of subtle epileptogenic lesions compared to 3-T scans. We believe that the differences are small (effectively no difference in detection of hippocampal sclerosis or other circumscribed lesions), and most importantly, scan type was equally represented in both Black and White populations.

In conclusion, this study points to the possibility that adult-onset MRI-negative TLE occurs more often in Black compared to White patients in the Southeastern United States. While the reasons for this racial difference are unclear, the data suggest possible race specific pathophysiological mechanisms that deserve further exploration including a possible link to autoimmunity and/or meningoencephalocepheles in the setting of IIH. Finally, the data also support a closer look at surgical candidacy and outcomes with regard to race.

Author contributions

Study concept and design: S.E.A., N.A.L., L.W.V.H., J.P.S., R.C.K.; Acquisition and analysis of the data: S.E.A., N.A.L., A.C.W., L.W.V.H., J.P.S., R.I.K., R.C.K.; Drafting the manuscript and/or figures: S.E.A., J.P.S, R.C.K.

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Declaration of competing interest

None of the authors has any conflict of interest to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.yebeh.2019.106501.

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