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Descriptive Analysis between TBI Diagnosis and Neurodegenerative Disease Outcomes in an Older Adult Population

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Abstract

Currently, the largest health crises in the world are neurodegenerative diseases, which affect millions per year. While studies examine plausible causative agents, associations between neurodegenerative disease and traumatic brain injury have been noted. Motivated by previous studies linking TBI exposure to neurological disease, this study examined TBI and neurodegenerative disease cases from the University of Kansas Medical Center (KUMC) HERON database. Three hundred and thirty-four unique cases were analyzed using Cox hazard regression modeling to characterize patterns in demographics, TBI diagnosis categories, and neurodegenerative disease outcomes for adults with one recorded TBI diagnosis. The Cox hazard model was uninterpretable due to no cases under 30 being identified and loss of variance; therefore, analysis was limited to older adults and focused on exploratory logistic regression analysis as opposed to time-to-event. The majority of associations examined were not statistically robust, but diffuse traumatic brain injury was observed to have a nominally significant association with Lewy Body Dementia. Within the cohort, an individual's sex was associated with Alzheimer's disease diagnosis. These findings describe plausible patterns in TBI-exposed older adult populations but should not be viewed as legitimate population pattern estimates.

Keywords

TBI, Neurodegeneration

1. Introduction

Neurology is a rich field of modern-day medicine. Originally started by Jean-Martin

Charcot in the 19th century, the field has evolved from a handful of neurological diseases to several distinct categories and classifications [1]. Of these classifications, neurodegenerative diseases remain a constant problem. Neurodegenerative diseases are neurological disorders caused by nerve cells in the brain or peripheral nervous system deteriorating, losing functionality, and ultimately dying [2] [3]. In 2016, the Partnership to Fight Chronic Disease Organization conducted a literature review observing the state of neurodegenerative disease in the U.S. At the time, neurodegenerative diseases (including Alzheimer's disease, dementia, ADRD, Parkinson's disease, motor neuron diseases such as amyotrophic lateral sclerosis, spinal muscular atrophy, hereditary spastic paraplegia, primary lateral sclerosis, progressive muscular atrophy, and pseudobulbar palsy) affected 4.7 - 6.0 million Americans, with 272,644 cases resulting in death and roughly 3,011,484 disability-adjusted life years [4]. Compounding the effects on people's health, these diseases accounted for nearly $655 billion in both medical and non-medical expenses during the same timeframe [4].

With most public health problems, they are judged by both prevalence and population perception. Currently, knowledge of Dementia, Parkinson's, and Alzheimer's remains well documented among the U.S. population, but the extent is incomplete. A scoping review from 2023 looked at global perceptions of Parkinson's disease and found three distinct themes [5]. First, the public's understanding of symptoms, causes, and treatment for Parkinson's was lacking [5]. Second, the majority of attitudes about Parkinson's were centered around the social and mental tolls they take on affected individuals and the use of these to drum up support for public participation in prevention/awareness [5]. Finally, public health informational material was slim in availability and quantity [5]. In summary, neurodegenerative diseases have become a health concern that has fallen in the public health hierarchy. Despite falling into the periphery, recent statistics and studies have indicated the need for a reevaluation. Reinvestment into awareness campaigns offers a simple solution, but public health methodology has evolved to focus on the deep-rooted causes of disease. Like other diseases, neurodegenerative disease is caused by several different factors: genetics, environmental hazards, lifestyle choices, and, most notably, aging populations [3] [6]. Currently, one possible causative agent has begun to receive more consideration from researchers due to its observed associations.

Traumatic Brain Injury (TBI) is caused by a forceful bump, blow, or jolt to the head or body, or the piercing of the skull or brain by an object [5]. In the last decade, researchers have studied the association between neurodegenerative disease and TBI to observe the effect of their association [3] [6]-[14]. Certain diseases have been debated over their association (Alzheimer's, Dementia, Parkinson's, ALS, Multiple Sclerosis, etc.), and while many studies support an association, findings remain inconsistent across diseases.

This study was motivated by the gap in understanding of potential long-term neurological health effects caused by TBI exposure. To examine this plausible as-

sociation, a cohort study was conducted using an available older adult population and focuses on describing patterns of neurodegenerative disease diagnosis with documented TBI exposure within this population.

2. Literature Review

Research into the association between TBI and neurodegenerative disease has been conducted at various levels. Most studies have looked exclusively at one neurodegenerative disease. Many of these studies have examined Chronic Traumatic Encephalopathy or CTE [8]-[10] [15] [16]. CTE was originally studied in boxers in the late 1920s but would not rise to national prominence until the early 2000s after Dr. Bennet Omalu observed the first confirmed case of CTE from a deceased former NFL player [9]. Since then, numerous studies have examined the association between TBI and CTE and found evidence to support their association [9] [10] [15] [16]. Currently, most research aims at studying the mechanisms that lead to CTE, including which areas of the brain are affected, what happens to brain matter as CTE forms, and whether treatment methods are viable [9] [16]. One such study interestingly found that patients with CTE formed buildups of hyperphosphorylated Tau proteins in neurons and glial cells, similar to other neurodegenerative diseases such as Alzheimer's, FTD, PiD, and PSP [8].

Other studies have examined another key neurodegenerative disease, Alzheimer's Disease. Alzheimer's Disease is one of the most common forms of dementia in the world, affecting roughly 6 million people over the age of 65 [17]. Research has been conducted on Alzheimer's Disease for decades now and one study examined the question of whether any research related to Alzheimer's Disease and TBI association had produced a consensus among the scientific community [11]. Of eleven studies screened, five claimed an increase in risk, two claimed no increase in risk, and four observed an association but only under certain circumstances [11]. Errors with methodology were noted by the researchers, but this highlights the lack of consensus regarding TBI and Alzheimer's Disease association.

The remaining studies examined the association through different scientific disciplines. One systematic review observed changes in brain morphology, including but not limited to reduction of volume and cortical thickness [11]. Another systematic review study examined eighteen studies with a total of 3,263,207 patients and found that TBI was associated with neurodegenerative diseases dementia, FTD, and TDP-43 (OR: 1.93, [95% CI: 1.47 - 2.55], $p < 0.0001$; OR: 4.44, [95% CI: 3.86 - 5.10], $p < 0.0001$; OR: 2.97, [95% CI: 1.35 - 6.53], $p < 0.0001$) [11]. No associations with Alzheimer's or Parkinson's were observed [12]. Similar studies examined working-age populations and found similar results of increased risk of neurodegenerative disease or neurological damage [13].

These studies have been conducted at multiple levels: proteomics, microbiology, epidemiology, and systematic literature reviews. Currently, many studies support an association, though findings vary. With minor exceptions, including a brain morphology study, most prior studies and reviews examined adults and older

adult populations, which reflected the influence of aging on neurodegenerative disease risk. Understanding disease risk at all ages is important, but the emphasis reflects the trend of neurodegenerative diseases occurring more in older adults, where age acts as a risk factor. Understanding these associations further remains a focus for public health research, but evaluation should be performed using a dataset specified for early-life exposures.

3. Methodology

The objective of this study was to examine the association between TBI and neurodegenerative disease outcomes with an older-adult TBI retrospective descriptive study. Prior literature hypothesized that early-life TBI outcomes may increase susceptibility to later-life neurodegenerative disease outcomes; however, the dataset used was unable to procure any TBI cases before the age of 30, thus preventing examination of early-life TBI exposure and restricting the analysis within the study population to associations among older adults. As a result, this analysis is reflective of an adult-skewed population and focuses on associations within the cohort instead of age at exposure.

A retrospective descriptive study was conducted using de-identified electronic health records from the University of Kansas Medical Center (KUMC) Healthcare Enterprise Repository for Ontological Narration (HERON) Database. A prospective cohort study would be more suitable for precise records of exposure timing and patient follow-up but was not achievable due to resource availability.

3.1. Ethics/Data Governance

All data were obtained de-identified by the HERON repository. All data were de-identified prior to access, and reidentification is prohibited by institutional policy. HERON access is restricted to KUMC faculty, students, and sponsored researchers. All researchers must complete Human Subject Protection training and an institutional data-use agreement before access is granted.

This project was reviewed by the University of Kansas Medical Center (KUMC) Institutional Review Board and deemed IRB exempt as secondary research of de-identified health record data. No identifiable information was used.

3.2. Data Search

Data were extracted using the HERON query system. The system identifies patients based on ICD9 and ICD-10 codes. HERON stores encounter-level billing codes, thus meaning diagnoses may have appeared multiple times for a single patient but do not represent a unique clinical event.

Selection criteria were determined by three categories.

1) At least one recorded ICD-9/10 diagnosis of Alzheimer's Disease or Dementia with Lewy Bodies.

2) At least one recorded ICD-9/10 diagnosis for TBI. TBI diagnoses included concussions, cerebral contusions/lacerations, intracranial hemorrhages of the

subarachnoid, subdural, or extradural layer, diffuse TBI, and other specified or unspecified TBI. Due to encounter-level codes being unable to distinguish distinct TBI cases from follow-up, at least one diagnosis was used for defining exposure.

Additional demographic material—race, sex, ethnicity, etc.—was included for descriptive statistics. Family history and child abuse history were extracted automatically by the query but left out of the analysis. See **Appendix A** for breakdown of data search.

Three hundred and thirty-four patients met the selection criteria and were collected. A de-identified dataset was used, which included patient ID numbers, data referenced in the HERON search, demographic information, start/end dates of visits, billing codes, and additional health record codes. This study was unable to distinguish incidence from prevalence for neurodegenerative disease records due to a lack of pre-injury medical history; therefore, all outcomes used are reflective of first diagnosis documentation in the HERON System and not clinical onset.

3.3. SAS Data Cleaning

The dataset was provided in multiple Excel files and was merged using the Statistical Analysis System (SAS). The first file contained diagnosis code paths and was kept for any coding clarification. The next file was empty and was unused. The next file contained patients' family history, including patient ID number, HERON code, variable ID, the family member's disease or injury, the relative's association to the patient, and internal HERON coding language. The next file included patient-specific data, including vital status, birth date (this was censored out due to being deidentified), death date, age, sex, language, race, marital status, ethnicity, religion, and date of last visit. The last file was a continuation of the descriptive path for HERON codes. The two files containing patient diagnosis records and family records were merged using SAS. The merged file was cleaned and contained the following variables: patient ID number, vital status, death date, age, sex, language, race, marital status, ethnicity, religion, last visit date, diagnosis code, and first recorded visit date.

Before analysis, a new column was created indicating if the diagnosis was TBI or neurodegenerative disease, then read into one of two columns that stated the name of the disease/injury. Since all data was de-identified, date of birth was not provided. To calculate patient age at diagnosis, an approximation was used by examining the visit dates and recorded age at the time of encounter. After completion, data was minimized, for each patient, to include only the first record of TBI diagnosis and first neurodegenerative disease diagnosis. These represent the earliest records available by the HERON database but do not necessarily indicate onset. An additional variable was created to classify patients as either "developing" (≤30 years of age when diagnosed with TBI) or "non-developing" (>30 years of age when diagnosed with TBI). No patients collected fell into the "developing" category; resulting in no variance for this exploratory variable. This variable was for documentation of the originally intended analysis and was not used in further

analysis. Estimates should be interpreted with caution, since they reflect encounter timing and not clinical onset.

The primary hypothesis was tested using Cox Proportional Hazards Regression. Due to a lack of patients who experienced TBI before 30 years of age, the exposure variable had no variance, making the Cox Model uninterpretable. Additionally, a survival curve was created for descriptive analysis and comparison with existing literature.

Secondary analysis examined additional variables of encounter type, specific TBI diagnosis categories and neurodegenerative disease, and demographics. An additional variable was added to the dataset that indicated the visit type (HERON designates visits as either initial, subsequent, or sequela). All three were tested using logistic regression modeling and were for investigational purposes and indicate within-cohort associations as opposed to risk-based conclusions. All regression analyses were exploratory; adjustment for multiple comparisons was not applied. The data were limited by the encounter-level nature of the data and the lack of a non-TBI comparison group.

4. Results

The average age of patients was roughly eighty-three years old, with the youngest patient being thirty-one and the oldest being ninety. Zero patients experienced a TBI diagnosis before the age of thirty; therefore, the Cox model was uninterpretable due to loss of variance. The survival curve showed a fifty percent drop in survival probability after one year, and the survival probability reached zero after roughly seventeen years after TBI diagnosis (two hundred seventy-three of the three hundred thirty-four records were used in the analysis). This survival curve reflects the timing of records in the HERON system and is not a representation of any disease progression, onset, or latency.

Secondary analysis testing of TBI diagnosis category frequency indicated Traumatic Subdural Hemorrhages as the most frequent, with one hundred forty records (20.77%), Concussions with one hundred twenty-seven (18.84%), Unspecified Intracranial Injuries with one hundred (14.84%), Cerebral Laceration or Contusion with twelve (1.78%), Contusion or Laceration of the Cerebrum with eight (1.19%), Contusion or Laceration of the Left Cerebrum with five (0.74%), Contusion or Laceration of the Right Cerebrum with five (0.74%), Diffuse Traumatic Brain Injury with ten (1.48%), Epidural Hemorrhage with six (0.89%), Intracranial Injury with twenty-five (3.71%), Other and Unspecified Intracranial Hemorrhage with twenty (2.97%), Subarachnoid, Subdural and Extradural Hemorrhage with seventy-five (11.13%), Traumatic Cerebral Edema with five (0.74%), Traumatic Hemorrhage of the Cerebrum with twenty-five (3.71%), Traumatic Hemorrhage of the Left Cerebrum with four (0.59%), Traumatic Hemorrhage of the Right Cerebrum with twelve (1.78%), Traumatic Subarachnoid Hemorrhage with eighty-eight (13.06%), and Unspecified Focal Traumatic Brain Injury with seven (1.04%).

These frequencies are an indication of encounter-level ICD codes and may be affected by reporting of the same injury across multiple visits (Figure 1). Reported TBI categories reflect ICD-9 and ICD-10 diagnostic label records and not standardized injury phenotypes.

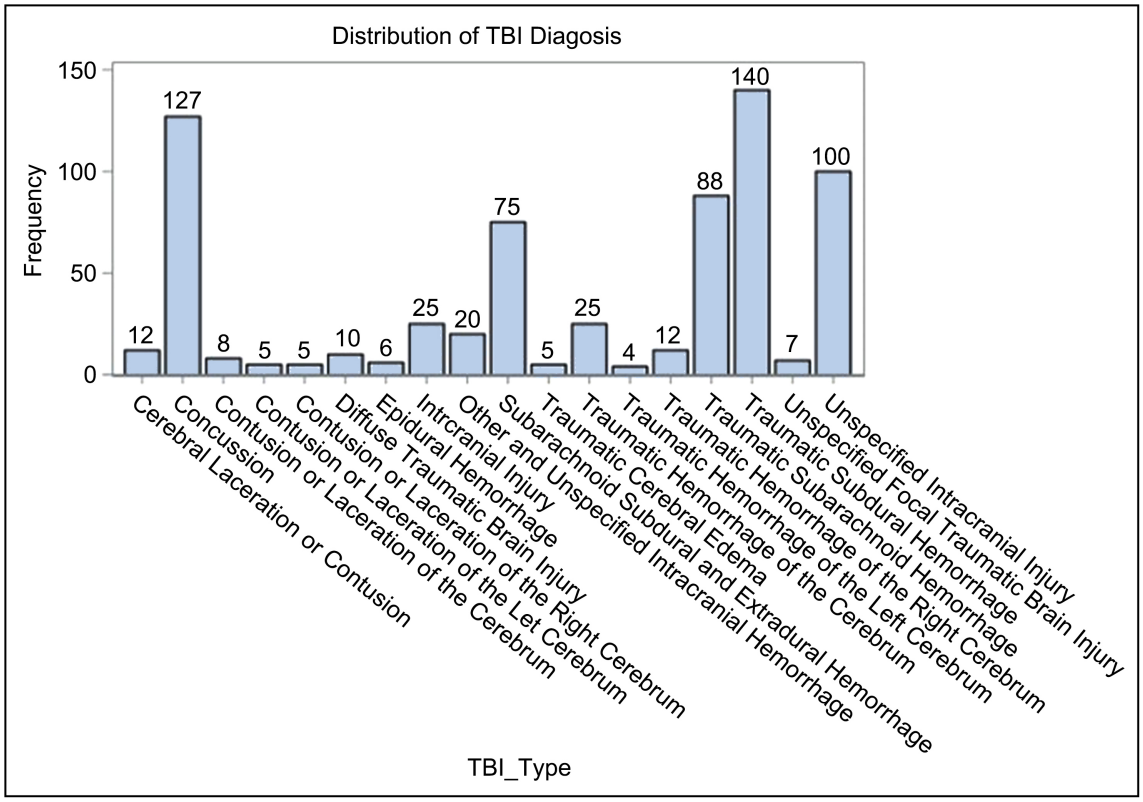

Figure 1. Distribution of TBI diagnosis by ICD code.

For frequency data of encounter types by TBI diagnosis, three hundred forty-seven (71.40%) were initial encounters, fifty-one (10.49%) were subsequent encounters, and eighty-eight (18.11%) were sequelae (Figure 2). Traumatic Subdural Hemorrhage had the most initial encounter records with one hundred eighteen (24.28%), followed by Concussions with forty-five (9.26%) and Traumatic Subarachnoid Hemorrhage with sixty-seven (13.79%). Unspecified Intracranial Injury had the highest number of subsequent encounter records with thirty-eight (7.82%), followed by Traumatic Subdural Hemorrhage with five (1.03%) and Traumatic Subarachnoid Hemorrhage with four (0.82%). Concussions had the highest number of sequelae records with twenty-four (4.94%), followed by Unspecified Intracranial Injury with nineteen (3.91%) and Traumatic Subdural Hemorrhage and Traumatic Subarachnoid Hemorrhage tied with seventeen (3.50%) (Chi-Square Value = 131.3714, p-value = <0.0001). For neurodegenerative disease diagnosis, Alzheimer's Disease had the highest number of recorded cases with four hundred ten (87.98%) and Lewy body Dementia had fifty-six (12.02%) recorded diagnoses.

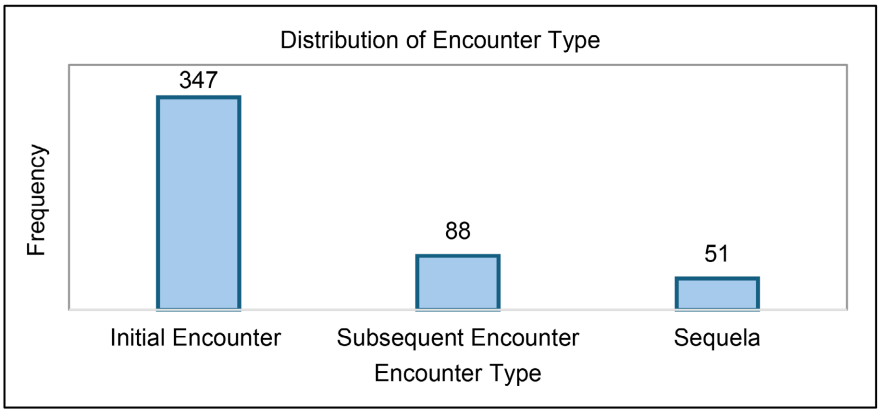

Figure 2. Distribution of encounter type for TBI and neurodegenerative disease diagnosis.

For logistic regression of TBI diagnosis against Alzheimer's diagnosis, concussion diagnosis had an odds ratio of 0.717 (95% CI: [0.312, 1.648], p-value = 0.4341). Cerebral laceration or contusions diagnosis had an odds ratio of 1.187 (95% CI: [0.140, 10.072], p-value = 0.8748).

Contusion or Laceration of the Cerebrum diagnosis had an odds ratio of 1.700 (95% CI: [0.053, 54.678], p-value = 0.7644). Contusion or Laceration of the Left Cerebrum diagnosis had an odds ratio of 0.885 (95% CI: [0.023, 34.642], p-value = 0.9479). Contusion or Laceration of the Right Cerebrum diagnosis had an odds ratio of 3.200 (95% CI: [0.056, 182.993], p-value = 0.5732). Diffuse Traumatic Brain Injury diagnosis had an odds ratio of 0.263 (95% CI: [0.050, 1.383], p-value = 0.1148). Epidural Hemorrhage diagnosis had an odds ratio of 0.352 (95% CI: [0.043, 2.899], p-value = 0.3317). Intracranial Injury diagnosis had an odds ratio of 0.380 (95% CI: [0.122, 1.186], p-value = 0.0958). Other and Unspecified Intracranial Injury diagnosis had an odds ratio of 0.895 (95% CI: [0.190, 4.207], p-value = 0.8878). Subarachnoid, Subdural, or Extradural Hemorrhage diagnosis had an odds ratio of 0.507 (95% CI: [0.198, 1.296], p-value = 0.1559). Traumatic Cerebral Edema diagnosis had an odds ratio of 0.416 (95% CI: [0.046, 3.719], p-value = 0.4324). Traumatic Hemorrhage of the Cerebrum diagnosis had an odds ratio of 1.575 (95% CI: [0.257, 9.651], p-value = 0.6233). Hemorrhage of the Left Cerebrum diagnosis had an odds ratio of 0.401 (95% CI: [0.030, 5.357], p-value = 0.4894). Traumatic Hemorrhage of the Right Cerebrum diagnosis had an odds ratio of 0.207 (95% CI: [0.033, 1.294], p-value = 0.0922). Traumatic Subarachnoid Hemorrhage diagnosis had an odds ratio of 1.712 (95% CI: [0.531, 5.518], p-value = 0.3682). Traumatic Subdural Hemorrhage diagnosis had an odds ratio of 0.629 (95% CI: [0.283, 1.400], p-value = 0.2562). Unspecified Focal Traumatic Brain Injury diagnosis had an odds ratio of 0.426 (95% CI: [0.052, 3.494], p-value = 0.4269). Unspecified Intracranial Injury diagnosis had an odds ratio of 1.362 (95% CI: [0.560, 3.310], p-value = 0.4958). Within this TBI-exposed cohort, no TBI diagnosis category was found to have shown any nominal associations with Alzheimer's disease. These results reflect within-cohort associations and should not be interpreted as population-level risk estimates.

For logistic regression of TBI diagnosis against Lewy Body Dementia diagnosis, Concussion diagnosis had an odds ratio of 1.563 (95% CI: [0.763, 3.323], p-value = 0.2455). Cerebral Laceration or Contusions diagnosis had an odds ratio of 0.282 (95% CI: [0.087, 5.923], p-value = 0.7587). Contusion or Laceration of the Cerebrum diagnosis had an odds ratio of 0.605 (95% CI: [0.018, 20.579], p-value = 0.7801). Contusion or Laceration of the Left Cerebrum diagnosis had an odds ratio of 0.336 (95% CI: [0.007, 16.556], p-value = 0.5834). Contusion or Laceration of the Right Cerebrum diagnosis had an odds ratio of 0.220 (95% CI: [0.003, 16.123], p-value = 0.4892). Diffuse Traumatic Brain Injury diagnosis had an odds ratio of 6.787 (95% CI: [1.478, 31.174], p-value = 0.0138). Epidural Hemorrhage diagnosis had an odds ratio of 2.003 (95% CI: [0.245, 16.402], p-value = 0.5173). Intracranial Injury diagnosis had an odds ratio of 2.399 (95% CI: [0.824, 6.990], p-value = 0.1087). The diagnosis of Other and Unspecified Intracranial Injury had an odds ratio of 0.793 (95% CI: [0.173, 3.629], p-value = 0.7646). Subarachnoid, Subdural, or Extradural Hemorrhage diagnosis had an odds ratio of 2.032 (95% CI: [0.853, 4.842], p-value = 0.1096). Traumatic Cerebral Edema diagnosis had an odds ratio of 1.760 (95% CI: [0.199, 15.558], p-value = 0.6112). Traumatic Hemorrhage of the Cerebrum diagnosis had an odds ratio of 0.727 (95% CI: [0.134, 3.953], p-value = 0.7117). Traumatic Hemorrhage of the Left Cerebrum diagnosis had an odds ratio of 7.250 (95% CI: [0.664, 79.182], p-value = 0.1044). Traumatic Hemorrhage of the Right Cerebrum diagnosis had an odds ratio of 3.136 (95% CI: [0.461, 21.333], p-value = 0.0922). Traumatic Subarachnoid Hemorrhage diagnosis had an odds ratio of 0.405 (95% CI: [0.127, 1.286], p-value = 0.1252). Traumatic Subdural Hemorrhage diagnosis had an odds ratio of 1.497 (95% CI: [0.713, 3.143], p-value = 0.2866). Unspecified Focal Traumatic Brain Injury diagnosis had an odds ratio of 2.893 (95% CI: [0.444, 18.833], p-value = 0.2665). Unspecified Intracranial Injury diagnosis had an odds ratio of 0.843 (95% CI: [0.386, 1.842], p-value = 0.6691). Of the ICD-coded TBI diagnoses, Diffuse Traumatic Brain Injury showed a nominally significant odds ratio with Lewy Body Dementia. These results reflect within-cohort associations and should not be interpreted as population-level risk estimates.

Demographic analysis was performed descriptively using regression modeling and frequency analysis. Any results should be carefully interpreted because of small subgroup sizes. For sex, there were one hundred eighty (53.89%) female patients, and one hundred fifty-four (46.11%) male patients recorded. For race, two hundred fifty-three (75.75%) patients identified as White, fifty-one (15.27%) identified as Black, eighteen (5.39%) identified as others, five (1.50%) declined to identify their race, two (0.60%) identified as two races, two (0.60%) identified as American Indian, two (0.60%) identified as Asian, and one (0.30%) identified as Pacific Islander. For ethnicity, eighteen (5.39%) patients identified as Hispanic, three hundred eleven (93.11%) did not identify as Hispanic, and five (1.50%) refused to answer. For vital status, one hundred nineteen (84.40%) patients were deceased at the time of data collection, three (2.13%) patients were DDU at the time of data

collection, and nineteen (13.48%) patients' vital status was reported as SSA (Social Security Administration). For primary language spoken, three hundred twenty-two (96.41%) spoke English, five (1.50%) spoke Spanish, three (0.90%) were unknown, two (0.60%) spoke Nepalese, one (0.30%) spoke German, and one (0.30%) spoke Russian. For marital status, one hundred sixty-four (49.25%) were married, one hundred three (30.93%) were widowed, forty-one (12.31%) were single, twenty-one (6.31%) were divorced, three (0.90%) were unknown, and one (0.30%) was listed as having a life partner. For religion, eighty-six (25.75%) were Catholic, fifty-two (15.57%) were none of the above, forty-one (12.28%) were Christian, thirty-eight (11.38%) were Baptist, twenty-eight (8.38%) were Methodist, twenty-five (7.49%) were Protestant, twelve (3.59%) were Unknown, nine (2.69%) were Presbyterian, nine (2.69%) were Lutheran, six (1.80%) were Episcopal, four (1.20%) were Jewish, four (1.20%) were Non-Denominational, three (0.90%) were Ecumenical, three (0.90%) were Other, two (0.60%) were Church of God Christ, two (0.60%) were Jehovah Witnesses, two (0.60%) were Pentecostal, one (0.30%) was Atheist, one (0.30%) was Buddhist, one (0.30%) was Church of Christ, one (0.30%) was Greek Orthodox, one (0.30%) was Hindu, one (0.30%) was Holiness, one (0.30%) was Nazarene, and one (0.30%) was Unity. See **Appendix B** for a breakdown of demographic data for the study population.

For logistic regression modeling, females had an odds ratio of 1.963 (95% CI: [1.012, 3.808], p-value = 0.0460) for Alzheimer's disease. Males had an odds ratio of 0.509 (95% CI: [0.263, 0.998], p-value = 0.0460) for Alzheimer's disease. Females had an odds ratio of 0.557 (95% CI: [0.308, 1.006], p-value = 0.0523) for Lewy Body Dementia. Men had an odds ratio of 1.797 (95% CI: [0.994, 3.247], p-value = 0.0523) for Lewy Body Dementia.

For racial associations, whites had an odds ratio of 0.893 (95% CI: [0.412, 1.936], p-value = 0.7745). No association could be calculated for black individuals due to no distinguishability between predicted probabilities. Asians had an odds ratio of 0.711 (95% CI: [0.017, 29.707], p-value = 0.8580). Pacific Islanders had an odds ratio of 0.426 (95% CI: [0.005, 39.916], p-value = 0.7127). Two or more raced individuals had an odds ratio of 0.711 (95% CI: [0.017, 29.707], p-value = 0.8580). American Indians had an odds ratio of 0.711 (95% CI: [0.017, 29.707], p-value = 0.8580). Declined to answer individuals had an odds ratio of 0.420 (95% CI: [0.054, 3.261], p-value = 0.4064). Other classified individuals had an odds ratio of 0.940 (95% CI: [0.230, 3.837], p-value = 0.9310). For Lewy Body Dementia, whites had an odds ratio of 1.086 (95% CI: [0.544, 2.168], p-value = 0.8154). Blacks had an odds ratio of 1.024 (95% CI: [0.457, 2.296], p-value = 0.9536). Asians had an odds ratio of 1.046 (95% CI: [0.025, 43.547], p-value = 0.9810). Pacific Islanders had an odds ratio of 1.815 (95% CI: [0.020, 162.386], p-value = 0.7948). Two or more race individuals had an odds ratio of 1.406 (95% CI: [0.025, 43.547], p-value = 0.9810). American Indians had an odds ratio of 1.406 (95% CI: [0.025, 43.547], p-value = 0.9810). Declined to answer individuals had an odds ratio of 1.762 (95% CI: [0.228, 13.629], p-value = 0.5872). Other classified individuals had an odds ratio of 1.192

(95% CI: [0.349, 4.074], p-value = 0.7798).

For Ethnicity, people who identified as Hispanic had an odds ratio of 0.432 (95% CI: [0.138, 1.351], p-value = 0.1491). People who did not identify as Hispanic had an odds ratio of 1.689 (95% CI: [0.562, 5.082], p-value = 0.3508). Those who refused to answer had an odds ratio of 1.581 (95% CI: [0.065, 38.266], p-value = 0.7783). People who identified as Hispanic had an odds ratio of 2.256 (95% CI: [0.779, 6.535], p-value = 0.1339). People who did not identify as Hispanic had an odds ratio of 0.619 (95% CI: [0.223, 1.716], p-value = 0.3566). Those who refused to answer had an odds ratio of 0.470 (95% CI: [0.019, 11.355], p-value = 0.6419).

For marital status, married individuals had an odds ratio of 0.730 (95% CI: [0.380, 1.403], p-value = 0.3456). Widowed individuals had an odds ratio of 0.939 (95% CI: [0.468, 1.883], p-value = 0.8590). Widowed individuals had an odds ratio of 0.941 (95% CI: [0.356, 2.485], p-value = 0.9019). Divorced individuals had no measurable association with Alzheimer's Disease due to indistinguishable probabilities. Unknown individuals had no measurable association due to indistinguishable probabilities. The individuals listed as having a life partner had an odds ratio of 0.426 (95% CI: [0.005, 39.916], p-value = 0.7127). For Lewy Body Dementia, married individuals had an odds ratio of 0.998 (95% CI: [0.557, 1.791], p-value = 0.9958). Widowed individuals had an odds ratio of 1.198 (95% CI: [0.645, 2.224], p-value = 0.5681). Single Individuals had an odds ratio of 1.157 (95% CI: [0.490, 2.730], p-value = 0.7394). Divorced individuals had no measurable association due to indistinguishable probabilities. Individuals listed as unknown had an odds ratio of 0.743 (95% CI: [0.024, 23.061], p-value = 0.8655).

Individuals listed as having a life partner had an odds ratio of 1.815 (95% CI: [0.020, 162.386], p-value = 0.7948).

For Language and Religion, no calculable associations could be determined for any of the languages or religions against either neurodegenerative disease use. Sex was observed to be the only demographic variable that met nominal significance odds ratios with Alzheimer's disease within this older-adult TBI cohort. These associations indicate patterns for this older-adult TBI cohort and should not be used to generalize outside of the dataset due to small sample sizes for certain subgroups and encounter-level coding. These findings should be interpreted cautiously given the limitations of the dataset due to ICD codes being encounter-level, the lack of early-life TBI cases, and no non-TBI comparison group.

5. Discussions

The Cox model used for the primary hypothesis was uninterpretable due to no patients experiencing a TBI diagnosis before the age of 30, which resulted in a loss of variance for the exploratory variable. While disappointing, it highlights the need for additional research with a larger population and more attention to acquiring patients with injury history in the age ranges in question.

Given the uninterpretable nature of the Cox model due to the loss of variance in the exploratory variable, the survival curve ultimately serves as a reflection of

the descriptive patterns within the older-adult cohort. The curve depicts the timing from first TBI diagnosis to first diagnosis of neurodegenerative disease within the older-adult cohort; however, these results should not be interpreted as any legitimate estimation of disease risk or progression. Due to the diagnosis dates in HERON being encounter-level coding compared to clinical onset and the lack of early-life TBI cases, testing of the initial hypothesis was impossible. Therefore, this curve is representative of the time between recorded diagnoses in the HERON database and not disease progression, onset, or latency.

The distribution of neurodegenerative disease diagnoses within this older-adult cohort reflected higher counts of Alzheimer's disease compared to Lewy Body Dementia. This is consistent with national prevalence data. Encounter-type analysis showed most cases were recorded as initial encounter; however, these codes are not an indication of any pre-existing records prior to inclusion in the HERON database. Further studies should aim to determine if additional diagnoses exist from other healthcare providers, something this study did not have the resources to accomplish.

TBI diagnosis category distribution for this cohort represented encounter-level ICD coding compared to distinct injury types. Concussion, subdural hemorrhages, and subarachnoid hemorrhages were the most reported categories. Concussions can be caused by the brain colliding with the inner part of the skull, making localization of the brain damage hard [12]. Similarly, subdural and subarachnoid hemorrhages are buildups of pressure between the brain and the skull, caused by a torn blood vessel in the meninges that can occur in various locations around the brain [14] [18]. This makes inference of injury location challenging purely based on ICD coding. Interpretation of these results should be done carefully due to ICD-9 and ICD-10 codes recording them differently. ICD-9 codes combined certain categories like subdural and subarachnoid hemorrhages together compared to ICD-10, which kept them separate, making direct comparison challenging. Additionally, the incorporation of an "Unspecified Intracranial Injury" creates further challenges by providing neither localization nor structural information of the injury. These results do not represent any form of pattern regarding injury severity or localization. Logistic regression analysis provided mixed results regarding TBI diagnosis categories associated with neurodegenerative disease. This outcome was understandable given issues with small sample sizes and the encounter-level nature of ICD coding. Diffuse Traumatic Brain Injury was the only category that produced a nominally significant odds ratio with Lewy Body Dementia. This should be interpreted cautiously, as the number of records for diffuse TBI was ten; thus, the result could be apart from sample size effects rather than true association. Many of the TBI categories had similar issues of small counts. Additionally, many categories produced inflated confidence intervals and quasi-complete separations, making regression estimates unstable. These results are primarily caused by the same issues with differences in ICD-9 and ICD-10 category grouping, making these findings a reflection of cohort patterns instead of injury-specific risk for neuro-

degenerative disease. Due to the low numbers in frequency for TBI diagnostic categories and the lack of adjustment for multiple comparable groups, any nominally significant findings—particularly for low-frequency diagnoses such as Diffuse TBI—are susceptible to Type 1 errors and should be interpreted cautiously. See Figure 3 and Figure 4 for ROC curves for each regression model.

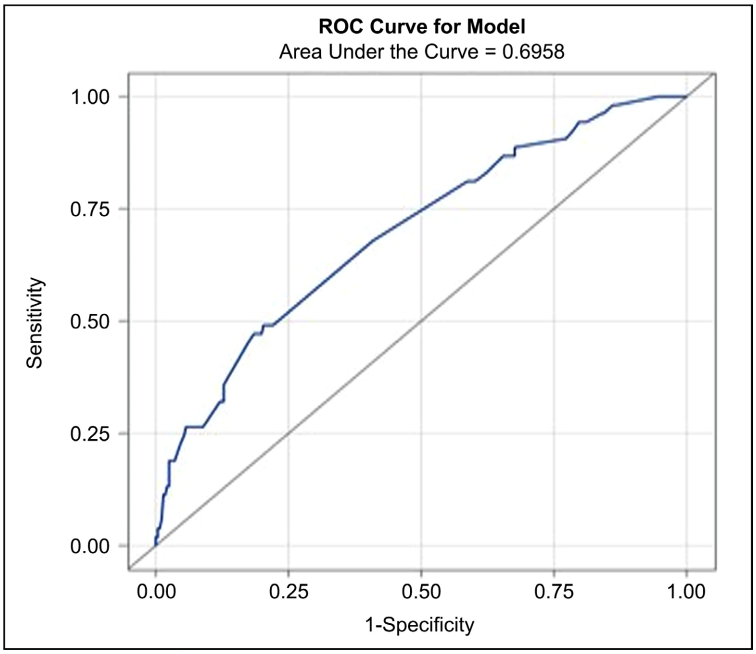

Figure 3. ROC curve for logistic regression of Alzheimer's disease against TBI diagnoses.

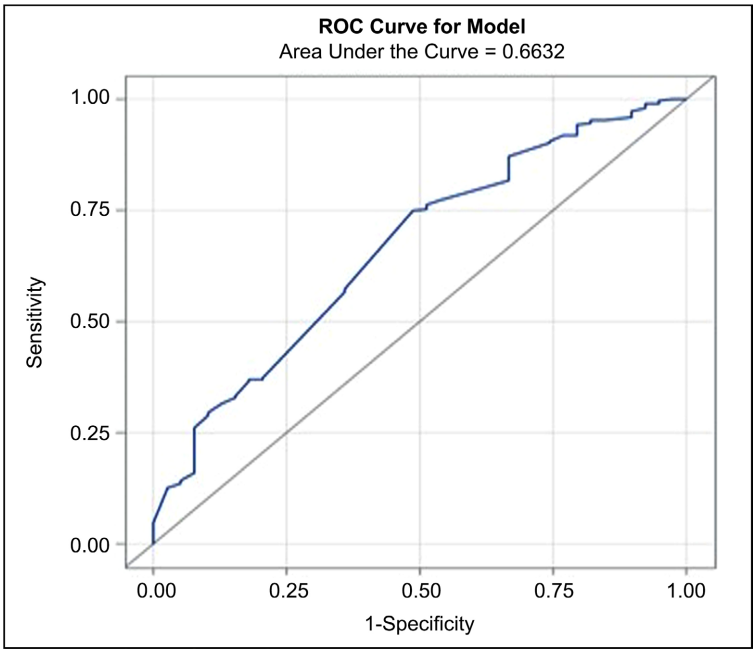

Figure 4. ROC curve for logistic regression of Lewy body dementia against TBI diagnoses.

Subsequent analysis of encounter types showed that TBI-related ICD codes for this cohort were primarily initial encounters, with few being subsequent or sequela. Prior studies have reported that individuals with neurodegenerative disease and any history of prior TBI usually experience more than one TBI diagnosis in their lifetime [11] [19]; this dataset was unable to confirm or deny this pattern. This is due to the encounter-level ICD codes' inability to indicate TBI occurrences outside of KUMC, indicate treatment from other medical providers, or capture diagnoses that may be recorded using older medical record systems. As a result, the abundance of initial encounters likely reflects a limitation in the completeness of the records rather than any injury occurrence patterns.

For demographics, the regression models were unstable due to the sparsity of the data and therefore all results should be cautiously interpreted. Sex was the only variable that met a nominally significant odds ratio for Alzheimer's disease in the cohort. A study performed a review on the pathological and phenotypical characteristics of certain neurodegenerative diseases and how they differed based on sex [20]. In each of the five diseases, they found differences in immune response, mortality rates, risk factors, and genomic variables, with neither sex displaying the same traits [20]. Specifically, with Alzheimer's disease, epidemiological data used in the review showed a 2-to-1 difference in incidence between women and men, with women experiencing Alzheimer's disease for longer periods while experiencing the effects of Alzheimer's disease much faster [20]. Additional studies examined further mechanisms, both biological and hormonal, though the results have been mixed [21]-[24]. While sex differences were observed, the findings should be cautiously interpreted as a possible reflection of the demographic differences in patterns of diagnosis rather than any biological susceptibilities.

Logistic regression showed no significant associations between any race or ethnicity and neurodegenerative disease in the cohort. The distribution of neurodegenerative disease was consistent with prior research, with non-Hispanic whites being most cases followed by Black individuals and then Hispanic individuals [25]. Though no significant associations were observed, race's role in neurodegenerative disease is still being actively investigated. Currently, studies have begun to come out highlighting potential variation in neurodegenerative disease outcomes based on geography. A 2022 study looked at neurodegenerative disease incidence in the U.S. and found national racial patterns like here, but state-level data found contrary results [26]. In Florida and New Mexico, it was found that Hispanics had a higher burden of Alzheimer's disease and Parkinson's disease, while in Illinois and Ohio, Blacks had a higher burden of Multiple Sclerosis than other racial groups, as well as having higher rates of stroke in most southern states [26]. Asian and Pacific Islanders had a higher burden of Alzheimer's disease, Parkinson's disease, Multiple Sclerosis, and stroke in Hawaii, and American Indian/Alaska Natives had a higher burden of Alzheimer's disease, Multiple Sclerosis, and Parkinson's disease in most northeastern states [26]. Further studies looked back at these results and identified biomarkers that may have contributed to the results [26] [27].

These results indicate the sheer depth of race-related differences in neurological disease and highlight that multiple factors may contribute to the differences. While it is unclear if certain groups are underrepresented in this dataset, a comparison with statewide data could determine accurate representation.

Language and religion had similar non-associative patterns as race and ethnicity. Regression modeling indicated no significant links with either neurodegenerative disease outcome. The distribution of religious identity in the cohort was made up largely of Christians and various Christian denominations. Like race, a comparison with statewide data to assess true representation would be useful. The current evidence on religion and spirituality's association with neurological disease is mixed. Recent research has explored whether spirituality or religiosity has any type of preventative effect on neurodegenerative disease, but results remain inconclusive. A Danish study found that dementia and Alzheimer's disease incidence was lower than the national average in Adventist and Baptist communities, while another study found that Parkinson's disease risk was higher in nonreligious adults [28]-[30]. These associations are still misunderstood and may indicate a combination of environmental, social, and biological factors. Presently, this study is unable to evaluate any of these potential mechanisms due to limitations in data, but additional research that examines these patterns in larger and more diverse populations may be beneficial.

Marital status showed no significant associations with neurodegenerative disease in regression modeling. Prior research has described an association between marital status and neurological health, including a study in China that found higher odds ratios of cognitive impairment for singles and divorced individuals, with even higher ratios if they were over 55 [29]. This study had a larger sample size and defined cognitive impairment differently, which may explain the difference in results [31]. Another study examined a cohort of ALS patients and reported longer survival times for married individuals and short survival times were observed primarily with females [32]. These studies indicate a plausible social and demographic effect on neurological outcomes and neurodegenerative disease, but differences in population, diseases examined, and survival metrics make comparisons with this study inappropriate. While this dataset was unable to replicate results or associations like those in these studies, interpretations should be made cautiously due to limitations in the dataset. Marital status may still be relevant for broader healthcare contexts, but this study is unable to evaluate this. Further research should consider using larger sample sizes and possibly including additional neurodegenerative diseases to examine whether similar results occur.

6. Limitations

The loss of variance due to the lack of patients who were diagnosed with TBI before the age of thirty resulted in uninterpretable hazard ratios and an uninterpretable primary hypothesis. Additionally, the use of a small sample size for demographic analysis made the logistic regression models semi-separated, making in-

terpretations difficult and requiring caution.

Use of retrospective over prospective cohort design. With a time-to-event analysis, prospective is a far better study design that allows for more accurate tracking of patients and identification of TBI and neurodegenerative disease. Due to the limitation of time and no funding being provided for this study, a prospective study was not possible, but future studies should aim to use a prospective approach.

Differences in ICD-9 and ICD-10 coding schemes restricted the comparability of the diagnostic categories. ICD-9 contains certain categories in one large subcategory, while ICD-10 kept them separate. Additionally, certain categories were nonspecific, further decreasing interpretability. Consequently, TBI categories are administrative labels and thus do not represent standardized injury phenotypes.

The dataset lacked important exposure characteristics such as TBI severity, number of prior TBI cases, treatment setting, and a comprehensive medical record. These are all factors that influence neurodegenerative disease, and without proper evaluation, the interpretability of the diagnostic associations was limited.

7. Conclusions/Recommendations

In conclusion, the initial primary hypothesis of early-life TBI associations with late-life neurodegenerative disease outcomes was not assessed due to the lack of TBI cases before the age of thirty. Sex was observed to have nominally significant odds ratios with Alzheimer's disease for this older-adult TBI cohort. An exploratory association was observed between Diffuse Traumatic Brain Injury and Lewy Body Dementia; however, additional replication with larger datasets and standardized exposure definitions is needed. Any replications of this study should use a more comprehensive and larger dataset that can go back as far as possible to obtain TBI records. It should also aim to have a single set of ICD diagnosis codes and obtain either family history or DNA samples for Alzheimer's to minimize any skewing of the data. For any studies that wish to examine this further without replication, use of a prospective style study is highly recommended to ensure the most accurate data possible.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix A

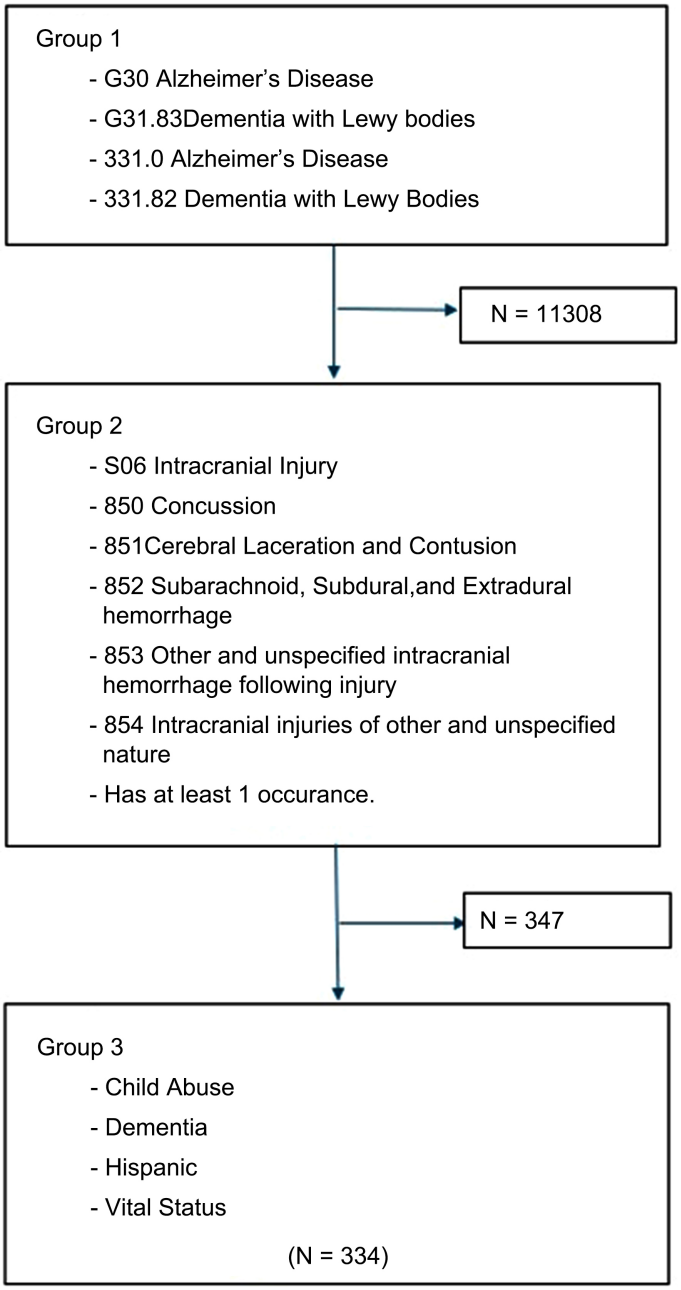

Figure A1. Example of HERON search.

Appendix B

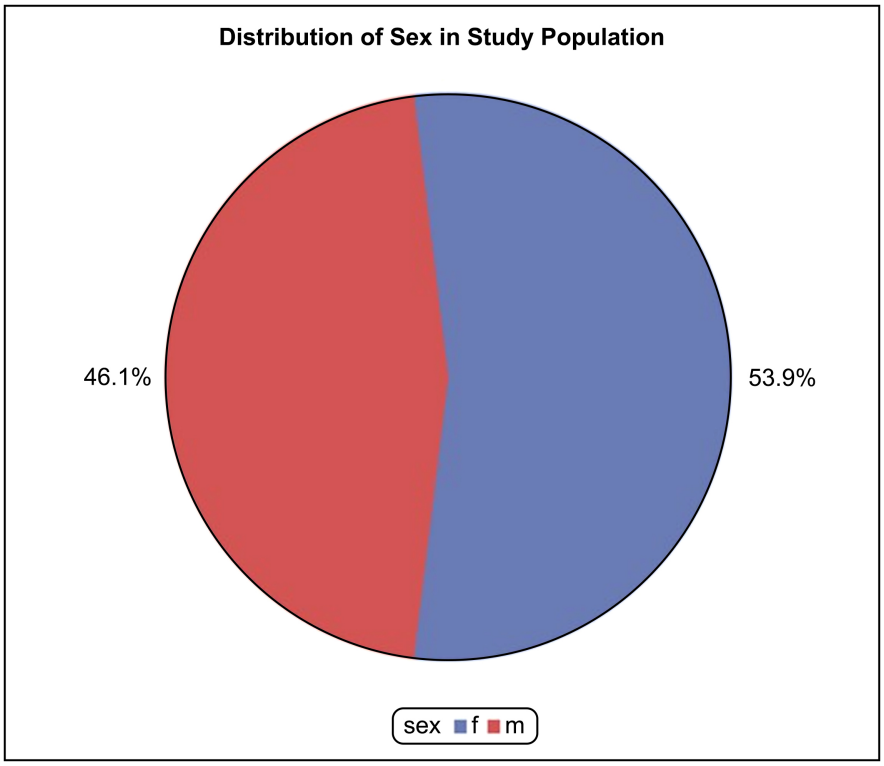

Figure A2. Distribution of sex in the study population.

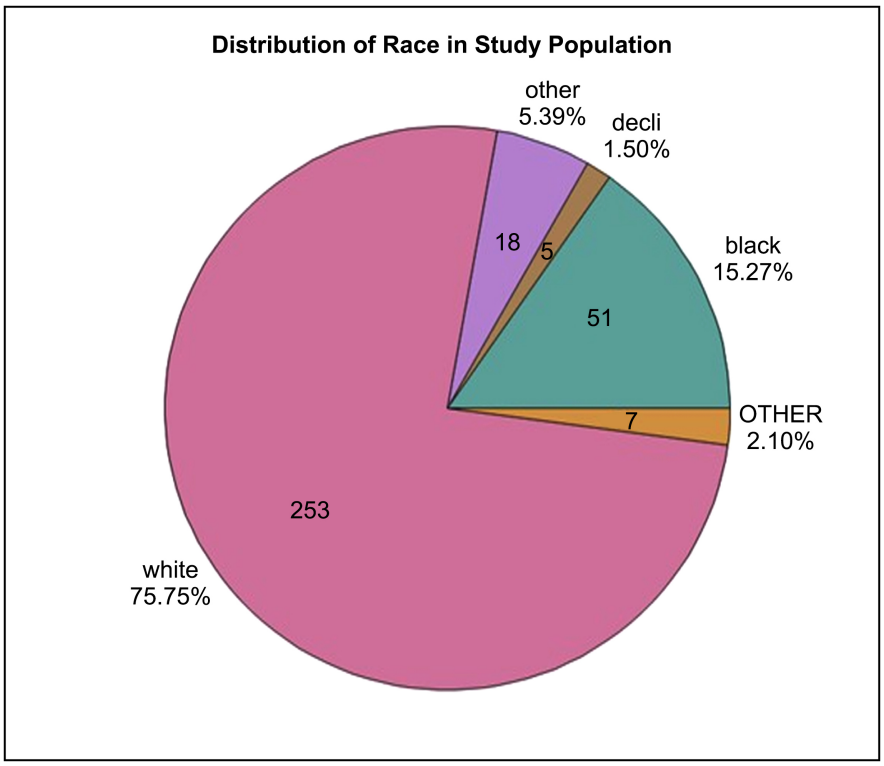

Figure A3. Distribution of race in study population.

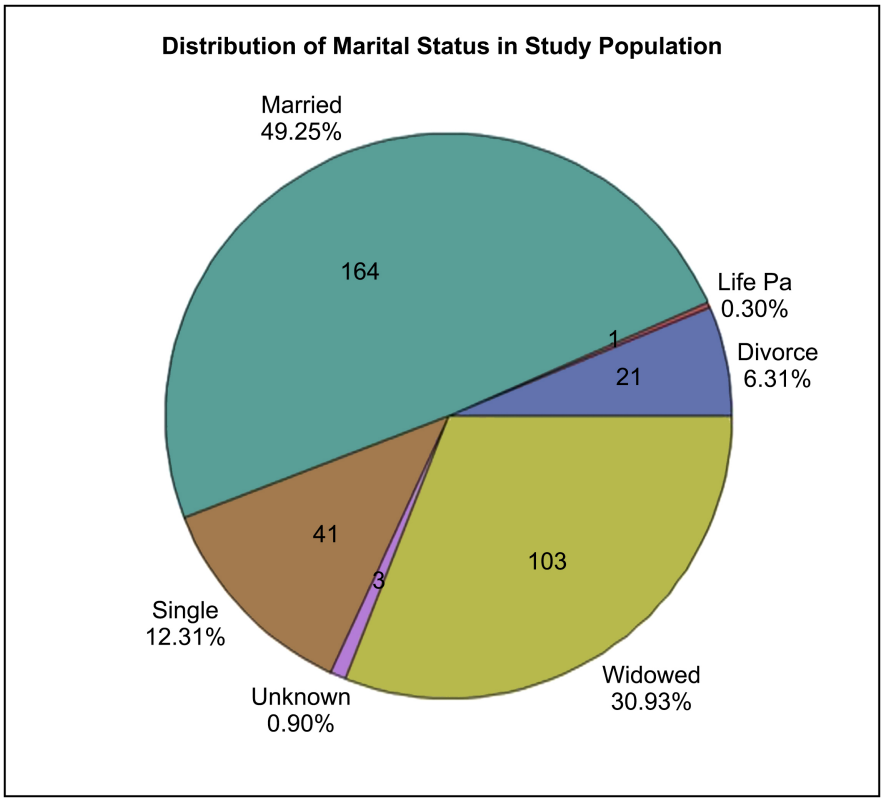

Figure A4. Distribution of marital status in study population.

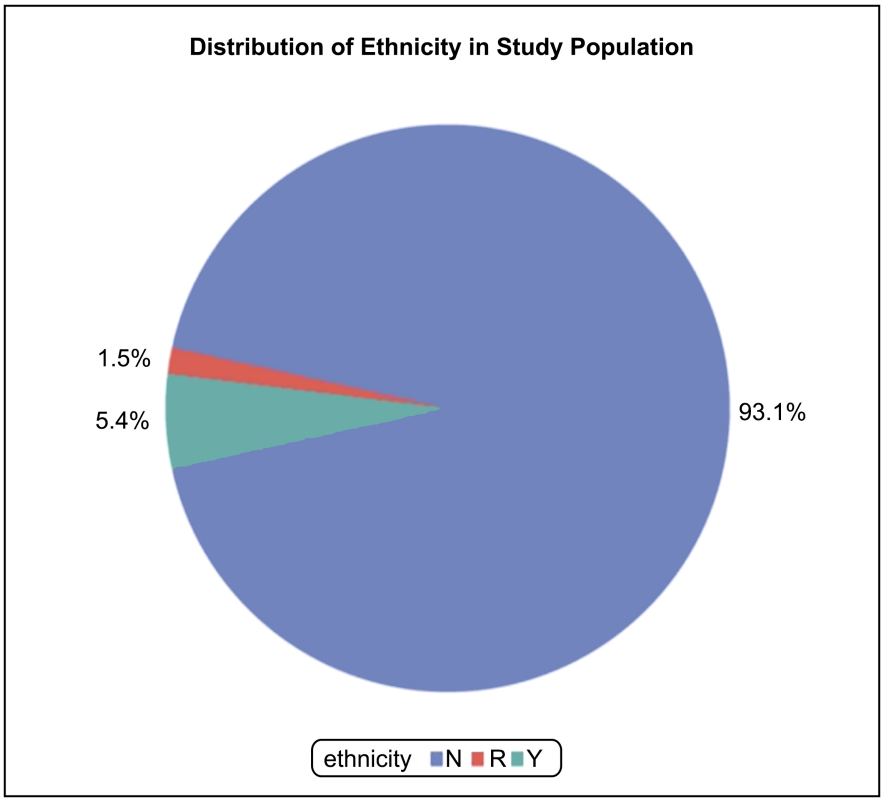

Figure A5. Distribution of ethnicity in study population.

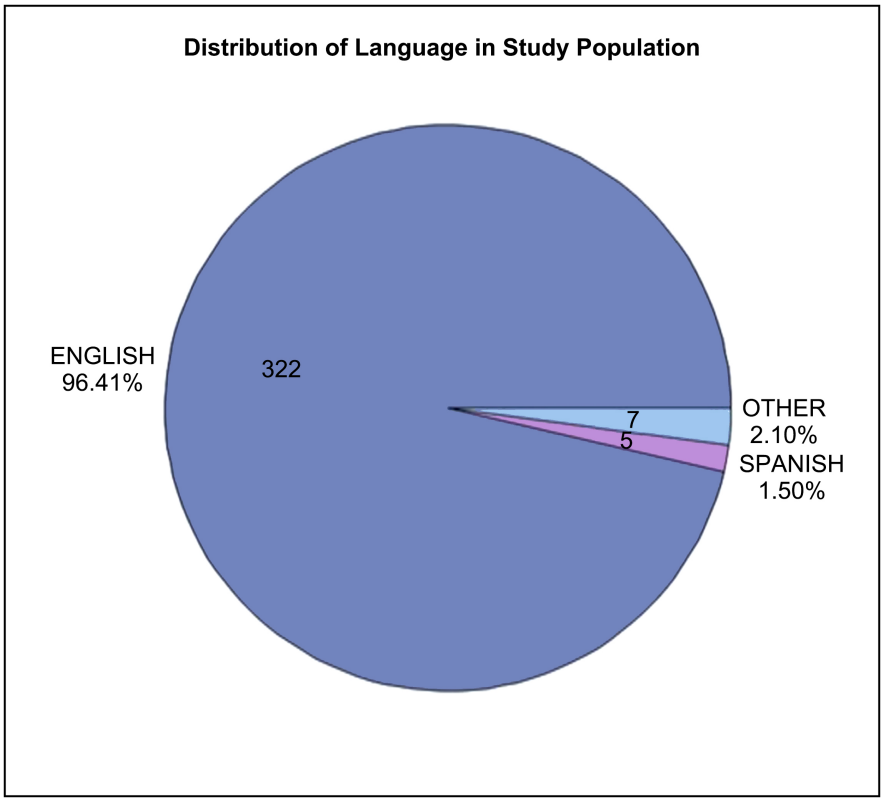

Figure A6. Distribution of language in study population.

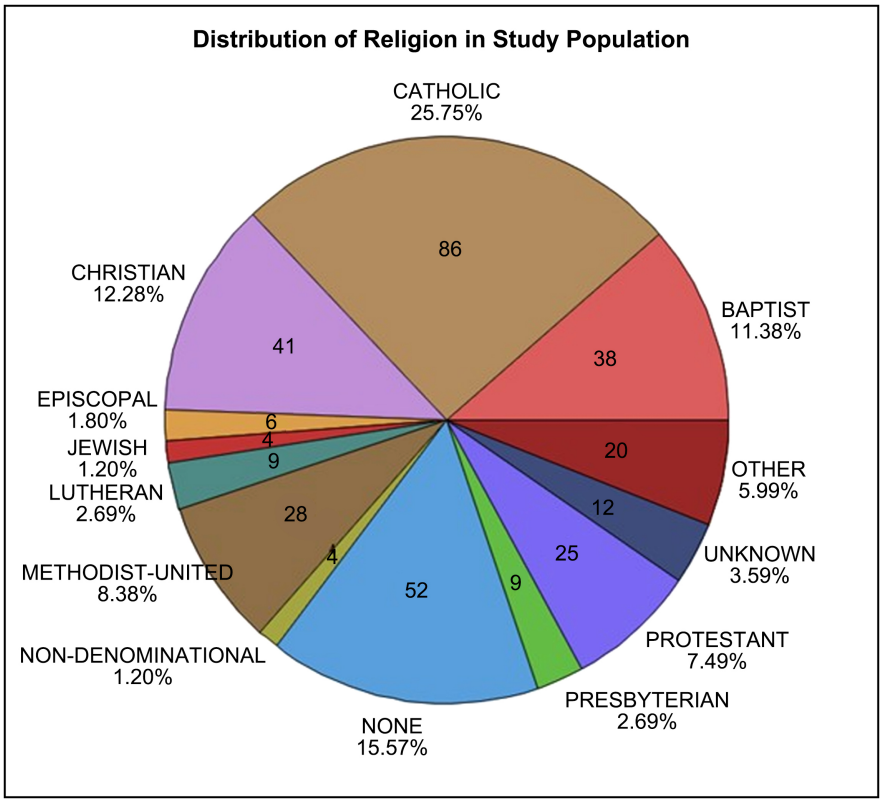

Figure A7. Distribution of religion in the study population.