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THE INCIDENCE OF POST-STROKE DEPRESSION IN ADULTS WITH APHASIA IN AN
ACUTE CARE SETTING

by

CHRISTINA ZANELLA

Under the Direction of Jacqueline Laures Gore, PhD

ABSTRACT

Post-stroke depression (PSD) affects at least one out of every three stroke survivors worldwide and presents with a variety of symptoms. The likelihood of its development relates to individual biological, psychological, and social circumstances, yet incidences are rarely addressed in the literature. Moreover, the presence of aphasia in some cases makes identifying PSD challenging. The purpose of this study was to investigate the incidence of PSD in adults with aphasia compared the incidence of PSD in adults without aphasia in an acute care setting, and to identify risk factors for PSD in adults with aphasia. Results indicated that adults with aphasia are 7.408 times more likely to exhibit PSD than adults without aphasia. Logistic regression controlling for the presence of aphasia showed a significant relationship between aphasia severity and PSD. Adults with aphasia were 2.06 times more likely to experience PSD with every 1-point increase in aphasia severity.

INDEX WORDS: Post-stroke depression, Aphasia, Relative risk, Logistic regression

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CHRISTINA ZANELLA

A Thesis Submitted in Partial Fulfillment of the Requirements for the Degree of

Master of Arts

in the College of Arts and Sciences

Georgia State University

2020

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Christina Zanella
2020

THE INCIDENCE OF POST-STROKE DEPRESSION IN ADULTS WITH APHASIA IN AN
ACUTE CARE SETTING

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December 2020

DEDICATION

This endeavor is dedicated to those who recognize that words alone will never adequately convey any first-person experience. More specifically, it is dedicated to those who have, in spite of that, shared with me the most enlightening and enriching communications. My own experience would be unreal without them.

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ALPHABETICAL LIST OF ABBREVIATIONS

AFib	atrial fibrillation
BDI	Beck Depression Inventory
CAB	other cerebral artery branch
CAD	coronary artery disease
CES-D	Center for Epidemiological Studies Depression Scale
CI	confidence interval
CVA	cerebrovascular accident
DSM-5	Diagnostic and Statistical Manual of Mental Health Disorders 5
HAM-D	Hamilton Depression Rating Scale
ICA	internal carotid artery
IRB	Institutional Review Board
LIV	Life Interests and Values Cards
MCA	middle cerebral artery
MDD	major depressive disorder
MI	myocardial infarction
MSNC	Marcus Stroke and Neuroscience Center
NIHSS	National Institute of Health Stroke Scale
OR	odds ratio
PHQ-2	Patient Health Questionnaire 2
PHQ-9	Patient Health Questionnaire 9
PSD	post-stroke depression
TIA	transient ischemic attack
VAMS	Visual Analog Mood Scale

1 INTRODUCTION

Post-stroke depression (PSD) affects at least one out of every three stroke survivors worldwide (Towfighi, et al., 2017) and presents with manifold symptoms (see de Coster, Leentjens, Lodder, & Verhey, 2005; Nakase, Tobisawa, Sasaki, & Suzuki, 2016; Skånér, Nilsson, Sundquist, Hassler, & Krakau, 2007). Because the likelihood of developing PSD relates to individual biological, psychological, and psychosocial factors, specific risk factors for developing PSD are a challenge to pinpoint and have therefore been neither adequately nor consistently reported. Moreover, it has been a challenge determining prevalence rates, and incidences are rarely addressed in the literature.

These and other problems related to PSD abound across the literature. The most apparent complications are with maintaining consistency with assessment methods and with incorporating the measurement of variables such as stroke type, stroke laterality, functional hemisphericity, time of onset, and specific lesion sites, among others. An additional variable and potential confound that may arise in experimental design is the presence of aphasia in participants. Because aphasia could be a hindrance to communicating effectively, and therefore threatens the integrity of results, its presence is often listed as part of the exclusion criteria for experiments investigating PSD (see Baccaro, et al., 2019; Caeiro, Ferro, Melo, Canháo, & Figueira, 2012; de Coster, et al., 2005; Donnellan, Hickey, Hevey, & O'Neill, 2010; Nishiyama, et al., 2010; Skånér, et al., 2007). Still, the presence of post-stroke aphasia is common, and is often enough to have a detrimental impact on stroke survivors' quality of life (Baker, Worrall, Rose, & Ryan, 2018; Haley, Womack, Harmon, & Williams, 2015; Northcott & Hilari, 2011), and could therefore contribute to both the onset and continuance of PSD.

With nearly six million stroke-related deaths reported worldwide each year, stroke itself is among the top three causes of death (Feigin, et al., 2014). Additionally, meta-analyses have revealed that the presence of PSD among groups significantly increases mortality rates (Bartoli, Brita, Crocamo, Clerici, & Carrà, 2018; Cai, Mueller, Li, Shen, & Stewart, 2019), with PSD contributing to a 35-fold increase in the likelihood of stroke-related death (Razmara, et al., 2017). One resounding question, then, asks whether PSD could be exacerbated by the presence of aphasia. If PSD is exacerbated by aphasia, then stroke survivors living with aphasia may be at an increasingly higher risk. It is for that reason critical for researchers and health care professionals to better understand the incidence of PSD in adults with aphasia and the risk factors contributing to its development.

1.1 Post-Stroke Depression

In an extensive review, Medeiros, Roy, Kontos, and Beach (2020) explain that PSD is most often defined using specific criteria from the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). In some ways, it is directly comparable to the general major depressive disorder (MDD), although it is attributable to an underlying cerebrovascular accident (CVA). Some specific symptoms of PSD, however, are slightly different than those seen accompanying MDD (Medeiros, et al., 2020).

1.1.1 Symptoms

PSD, like MDD, manifests a multitude of signs and symptoms including sleep disturbances, changes in appetite, psychomotor retardation and agitation (de Coster, et al., 2005; Nakase, et al., 2016), sadness, fatigue (de Coster et al., 2005; Skånér, et al., 2007), dizziness, generalized pain, difficulty concentrating, irritability, and fluctuant changes in weight (Skånér et al., 2007). Unlike those of MDD, the symptoms of PSD typically include both cognitive

impairment and some form of physical disability and are directly attributable to a specific cause (Medeiros et al., 2020). Regardless of its underlying etiology, though, PSD can be both disabling (Kneebone & Lincoln, 2012; Paolucci, et al., 2019), and still arduous to identify (Robinson & Jorge, 2016).

1.1.2 Risk Factors

Risk factors should be recognized as multifactorial, consisting of separate yet interactive biological, psychological, and psychosocial components. Researchers seem to have distinguished some risk factors for developing PSD, however, the inconsistencies in reported results make validating those factors a contentious issue.

For example, most researchers propose vascular complications as a biological cause of PSD (de Ryck, et al., 2014; Nakase, et al., 2016), although some argue there are no such correlations (Baccaro, et al., 2019). While those researchers focus on general complications, others prefer to concentrate on more precise mechanisms. Namely, Shen et al. (2019) explicated the association between serum lipid profiles and the risk of developing PSD. Both lower levels of high-density lipoprotein cholesterol and elevated ratios of low-density lipoprotein significantly increased the risk (Shen, et al., 2019). Some researchers even expand their perspectives and take into consideration potential environmental risk factors. Gu, Luan, Ren, Zhu, and He (2018) suggested that the season of the year during which a stroke occurs contributes to the risk. The authors explained that the lower circulating levels of vitamin D that were seen in stroke patients during wintertime made the resulting PSD more severe (Gu, Luan, Ren, Zhu, & He, 2018).

At least one study suggests that the risk of developing PSD is proportional to the number of stroke lesions (Jiang, Lin, & Li, 2014), and with advances in neuroimaging, more studies

exploring risk factors focus on the laterality of stroke lesions. Mitchell et al. (2017) conducted a meta-analysis and inferred that the risk of developing PSD was higher following a stroke occurring in the left hemisphere of the brain (Mitchell, et al., 2017). Nonetheless, there is still some controversy. While additional studies have arrived at similar conclusions (Jiang, et al., 2014), some claim the laterality of lesions shares no relationship with the risk (Baccaro, et al., 2019; Nakase, et al., 2016).

Additional risk factors may include pre-existing biological conditions such as hypertension (Jiang, et al., 2014; Nakase, et al., 2016) and diabetes mellitus, concurrent habits such as smoking and excessive alcohol consumption (Nakase, et al., 2016), and the number of comorbid conditions (Fei, et al., 2016). Comorbid cognitive dysfunction is a psychological risk factor for both PSD (Jiang, et al., 2014) and its accompanying suicidal ideation (Baccaro, et al., 2019). The risk is higher among those with a familial history of mood disorders (Mitchell, et al., 2017), and among those who have previously taken antidepressant medications (Fei, et al., 2016).

The primary psychosocial issues contributing to the risk of developing PSD are poor or outright lack of social support (de Ryck, et al., 2014; Fei, et al., 2016; Jiang, et al., 2014), with relationship problems making PSD three times as likely to materialize (de Ryck, et al., 2014). Additional potential risk factors for PSD might include age, gender, ethnicity, race, and the amount of time that has passed since a stroke occurred. Earlier studies indicate a greater likelihood of developing PSD when stroke occurs at a younger age (Abbas, Shahbaz, Umer, Umer, & Irfan, 2017; McCarthy, et al., 2016), and a significantly greater prevalence of PSD among women than men (Paradiso & Robinson, 1998; Poynter, et al., 2009). By focusing on racial-ethnic groups, Fei et al. (2016) concluded that the prevalence of PSD was significantly

higher among Latino groups than non-Latino groups (Fei, et al., 2016). Several studies investigating race as a risk factor for PSD indicate non-Hispanic whites are most likely to be diagnosed soon after experiencing a stroke (Goldmann, Roberts, Parikh, Lord, & Boden-Albala, 2016; Jia, et al., 2009), but one claims there is no significant association between race and PSD in the chronic post-stroke stage (Goldmann et al., 2016).

1.1.3 Prevalence and Incidence

With its overall prevalence ranging from 20 to 65% among survivors (Robinson & Jorge, 2016), PSD is one of the most commonly reported problems continually afflicting those who have experienced a stroke (Kneebone & Lincoln, 2012; Robinson & Jorge, 2016; Wang, et al., 2018). Such a wide range, however, tells that accounts of its prevalence have been variable. Since researchers' suggestions regarding the incidence of PSD are also questionable, it can be assumed that both the prevalence and the incidence of PSD are understudied. There are several notable dissimilarities between existing studies. One is which psychological assessment measures are used to determine participants' states of depression, among which inconsistencies could be to blame for varying accounts of prevalence. Another is at what points in time following the onset of stroke data are collected. Determining the incidence of any condition requires researchers to indicate an exact period of time (Bonita, Beaglehole, & Kjellström, 2006).

The Hamilton Depression Rating Scale (HAM-D) is among the most commonly used assessment measures, with prevalence reports of PSD ranging from 13.6% to 36.2% (Baccaro, et al., 2019; Bour, et al., 2010; de Coster, et al., 2005; Gu, et al., 2018; Jiang, et al., 2014; Shen, et al., 2019; Shimoda & Robinson, 1999). Several longitudinal studies actually combine measures to learn more about PSD and its time of onset. In one, Bour et al. (2010) followed 138 stroke

survivors over the course of one year, using visits with those participants roughly every three months to check for signs of depression. Beginning at one month following stroke, a research physician used the Structured Clinical Interview of the DSM-IV to determine whether or not participants showed signs of depression, and if they did, whether that depression was a major or minor depression. Throughout the course of that year, participants completed multiple depression scales, including the Beck Depression Inventory (BDI), the Hospital Anxiety and Depression Scale, the 17-item HAM-D, and the 90-item Symptom Checklist. A total of 50 participants (36.2%) experienced the onset of depression during that year. Twenty suffered with minor depression and 30 suffered with major depression, but of those 50, over half were identified as living with depression after a period of only one month (Bour, et al., 2010).

In nearly all of the research reviewed here, researchers gather data beginning at one month since the onset of stroke and consistently thereafter every three months up to 18 months (Baccaro, et al., 2019; Bour, et al., 2010; de Coster, et al., 2005; de Ryck, et al., 2014; Skånér, et al., 2007) Notable exceptions include those that gather data during the acute phase following stroke (Nakase, et al., 2016; Shen, et al., 2019). Only one was less specific, requiring participants to have experienced a stroke sometime within the previous five years, claiming the time since onset would have no significant effect (Fei, et al., 2016).

Further examples of relevant research encompass different designs. Mitchel et al. (2017) conducted a meta-analysis and found that 33.5% of cases were living with a depressive disorder at some time following a stroke. Times since stroke ranged from two days to seven years (Mitchell, et al., 2017). In a cross-sectional study, researchers Ryck et al. (2014) found that 24.5% of their participants reported feeling depressed beginning at one month following stroke. Follow-up visits revealed those percentages increased both at three-months (27.1%) and at six-

months (28.3%). It should be noted in this case, however, that the number of participants in attendance at each follow-up was different (de Ryck, et al., 2014), and that attrition rates could affect the reported distributions.

1.2 PSD and Aphasia

Aphasia is among the most common problems encountered following a stroke (Caeiro, et al., 2012) affecting one in three survivors (Khedr, et al., 2020; Townend, Brady, & McLaughlan, 2007). It has, perhaps in itself, led to chronic states of depression (Haley, et al., 2015). Aphasia is a set of conditions that affect a person's ability to either comprehend and/or express verbal messages (Spreeen & Risser, 2003), and is a consequence of acquired cerebral lesions (Basso & Cubelli, 1999).

Shehata, Mistikawi, Risha, and Hassan (2015) conducted a cross sectional study of 61 individuals, 30 of whom had survived a stroke and live with aphasia, and 31 of whom had survived a stroke but do not live with aphasia. Using the BDI, they were able to determine that those who were living with aphasia presented with significantly higher rates of PSD (Shehata, Mistikawi, Risha, & Hassan, 2015). Baseline measurements in another study indicated that 47.5% of participants living with aphasia were also dealing with PSD, whereas only 29.1% of the participants living without aphasia were (Wang, et al., 2018).

1.2.1 *Modifying Measures*

Too few studies have investigated the incidence of PSD among groups living with aphasia. As its incidence is studied, it is important to consider which measures are being used to confirm the presence of PSD in persons living with aphasia. The majority of depression scales in use depend on a person's ability to communicate, and therefore cannot always provide an effective tool in these cases (Wang, et al., 2018). Consequently, most studies investigating the

prevalence of PSD state the presence of aphasia as an exclusion criterion due to the potential confounds participants' difficulties communicating may introduce (Baccaro, et al., 2019; Caeiro, et al., 2012; de Coster, et al., 2005; Donnellan, et al., 2010; Nishiyama, et al., 2010; Skånér, et al., 2007). In a systematic review of 60 studies exploring the prevalence of PSD in aphasia, Townend et al. (2007) found that less than half of those studies used an appropriately adapted diagnostic tool to identify depression. The modifications they did find included using cards with key phrases to elicit the necessary answers, limiting interviews to depend solely on responses to dichotomous scales, and relying on visual analogue scales when verbal replies were not possible to obtain (Townend, et al., 2007).

Several attempts have been made to validate modified versions of measures. For instance, Radloff (1977) detailed the development of the Center for Epidemiologic Studies Depression Scale (CES-D) and indicated that it was originally developed to be used as a research tool measuring depression rather than a diagnostic tool. The CES-D consists of 20 items that were selected from depression scales that had been previously validated. Each item asks a respondent to self-report how often they had felt a specific way during the past week and is scored using a four-point scale. The overall range of scores runs from zero to 60 indicating the frequency of depressive symptoms occurring throughout the past week (Radloff, 1977). Ashaie, Hurwitz, and Cherney (2019) described how a speech-language pathologist used a modified version of the CES-D to test for depression in 144 participants living with post-stroke aphasia. The questions were delivered on laminated cards, and the speech-language pathologist would read them aloud so that the participant could provide answers. The speech-language pathologist would repeat questions if asked. One benefit these modifications may have bestowed was the ability to detect subthreshold levels of depression. Their study found that 19.44% of participants

exhibited depression, while 22.22% met the established criteria for subthreshold depression (Ashaie, Hurwitz, & Cherney, 2019).

Haley et al. (2015) acknowledge that, because the tools used to screen for depression often depend on participants' ability to produce and comprehend language, those tools may not be suitable to screen for depression in people with aphasia. Their study was designed to estimate the validity of the Visual Analog Mood Scale (VAMS) when used to measure the moods of people living with aphasia. They were able to estimate congruent validity by comparing items on the VAMS with similar items that appear on the Life Interests and Values cards (LIV). There was a strong correlation between indicators of happiness, and moderate correlations between indicators of sadness, anger, and worry (Haley, et al., 2015). Barrows and Thomas (2018) designed a comparable prototype consisting of seven dichotomous scales that enabled participants in their study to report their mood. They readily acknowledge, however, that the instrument has yet to be applied in the acute post-stroke stage (Barrows & Thomas, 2018). A more encompassing search through the literature could impart the practicalities of different versions of the VAMS in screenings.

1.2.2 Proxy Respondents

Some studies recognize the aforementioned challenges and rely on proxy measures to estimate the prevalence of PSD in aphasia. Hilary and Byng (2009) recognized that those who had been living with severe cases of aphasia would not have been able to report symptoms of depression using traditional measures and would have to rely on proxy respondents. In their study, they delivered the Stroke and Aphasia Quality of Life scale to proxy respondents nominated by individuals living with severe aphasia. They then compared their scores to those obtained in earlier studies that gathered data through proxy respondents nominated by

individuals living with mild to moderate aphasia and through individuals living with aphasia who were able to self-report. The measured quality of life scores for people living with severe aphasia were significantly lower than those that were gathered in both earlier studies (Hilari & Byng, 2009).

While proxy respondents seem to provide a compensatory measure, depending on the way they are recruited, their recruitment may deny initial participants' autonomy. Respect for persons is obligatory to adhere to ethics in research, and no participant should feel depersonalized (Horner, 2020). It would be best to find an appropriately modified and validated depression screening tool.

1.3 Problems Noted

The majority of studies reviewed herein had taken baseline measures of depression at a period of one month following the onset of stroke. It should be readily acknowledged that, during that interim, a multitude of extraneous variables can impact both the presence and severity of depression. Those extraneous variables make determining causal relationships unlikely. Measures of depression might best be gathered in an acute care setting where it will be possible to assess more direct relationships between PSD and risk factors.

While there has been contentious debate regarding those relations, one additional variable could be the presence of post-stroke aphasia. Because obtaining valid scores on the most typically utilized measures of depression depends on a person's ability to communicate, persons with aphasia are often excluded from experiments investigating matters related to PSD. Scores obtained through any measure may be affected by the type and severity of aphasia. An appropriately modified screening tool should be utilized.

In addition to the aforementioned shortcomings, studies do not always run analyses related to demographic information or pre-existing conditions. Research should take into account the relation between PSD and factors such as age, gender, race, and co-occurring medical diagnoses (or comorbidities, see Abbas, et al., 2017; de Coster, et al., 2005; Fei, et al., 2016; Jia, et al., 2009; Jiang, et al., 2014; McCarthy, et al., 2016; Nakase, et al., 2016; Paradiso & Robinson, 1998; Poynter, et al., 2009; Skånér, et al., 2007).

It is perhaps the need to take into account its multifactorial nature that makes finding agreement on the risk factors associated with PSD problematic. Furthermore, the co-occurrence of post-stroke aphasia introduces potential confounds in relevant experimental designs, making investigating the incidence of PSD among the affected population challenging. This research will attempt to address those problems.

2 OBJECTIVES

This current study seeks to explore the incidence of PSD in adults with aphasia in an acute care setting and to identify potential risk factors for developing this mental health disorder. A review of the literature has identified several key areas in which more data is needed to better understand PSD in adults with aphasia. Specifically, the research questions and hypotheses are as follows:

1. What is the incidence of PSD in adults with aphasia in an acute care setting?
2. How does this incidence of PSD compare to patients who are post-stroke without aphasia in the same acute care setting?
3. What are the risk factors for PSD in adults with aphasia in an acute care setting?

Based on varying reports of the overall prevalence of PSD (approximately 20-65%), it is hypothesized that the incidence of PSD in adults with aphasia will be higher than the incidence of PSD in adults without aphasia. This study will explore the potential risk factors of age, gender, race, selected comorbidities, and aphasia severity. Based on the review of previous literature, it is hypothesized that age, gender, race, selected comorbidities and aphasia severity will each be a risk factor.

3 METHODS

IRB approval for this study was obtained through Georgia State University's Office of Research Integrity under the determination that it would not be classified as research with human subjects (see Appendix A). Permission to access records found within Grady Memorial Hospital's Epic medical records system was obtained through the research coordinators at the hospital's Marcus Stroke and Neuroscience Center (MSNC).

3.1 Data Collection

A total of 1095 records were extracted. Records were from patients seen at MSNC's acute care setting throughout the calendar year of 2019. Each record was deidentified before being given a unique identification number. The selected data from each record detailed a patient's medical history (indicating conditions such as dyslipidemia, hypertension, diabetes mellitus, previous stroke, etc.), any noted priorly prescribed antidepressants, antidepressants prescribed at discharge, stroke lesion site(s), aphasia score, depression score, gender, age, race, ethnicity, and zip code of residence. The selected data was compiled into a spreadsheet using Microsoft Excel.

Aphasia scores were gathered prior to hospital discharge using Item 9 from the National Institute of Health's Stroke Scale (NIHSS). Patients were first shown an image depicting a family in a kitchen and were asked to describe the scene. They were then shown a picture of six objects (a glove, a key, a cactus, a feather, a chair, and a hammock) and were asked to name each of them. Lastly, they were shown a list of five sentences and were asked to read each of them aloud. Each patient's responses were used to assign a score indicating aphasia severity as outlined in the NIHSS; scores ranged from 0 to 3, with 0 indicating no aphasia, 1 indicating mild-to-moderate aphasia, 2 indicating severe aphasia, and 3 indicating mute, global aphasia (see

Appendix B). An additional column was created in the spreadsheet to categorize cases as either aphasic or non-aphasic.

Depression scores were determined using a modified version of the Patient Health Questionnaire-9 (PHQ-9). According to Kroenke, Spitzer, and Williams (2003), the Patient Health Questionnaire-2 (PHQ-2) is made up using only the first two questions found within the PHQ-9. Using a four-point Likert scale, respondents were asked to indicate, over the past two weeks, how often they had been bothered by a) “little interest or pleasure in doing things” or b) “feeling down, depressed, or hopeless.” Scores obtained using the PHQ-2 ranged from 0 to 6, with a score of 3 being a cutoff indicating the presence of depression (Kroenke, Spitzer, & Williams, 2003). For the purpose of this study, depression scores were dummy coded to create a dichotomous variable, with 1 assigned to indicate the presence of depression and 0 assigned to indicate the absence of depression.

3.2 Statistical Analyses

The spreadsheet was cleaned and imported to create a dataframe in R Studio (version 1.2.5001). Descriptive statistics were gathered using tables before the dataframe was sliced into two different groups, with one representing adults with aphasia (aphasic cases) and the other representing adults without aphasia (non-aphasic cases). The incidence proportions of PSD were calculated among each group and were then combined to calculate the relative risk of developing PSD with aphasia. Incidence proportions indicate the rate of emerging cases of a specific condition within a specified population over a given period of time (Bonita, et al., 2006), while relative risk compares the rate at which a condition affects persons who have been exposed to a potential cause to the rate at which the same condition affects persons who have not (Christie, Gordon, & Heller, 1997).

The incidence proportions of PSD in adults with aphasia and adults without aphasia were calculated by dividing the number of new diagnoses of PSD in adults with aphasia ($n = 176$) by the total number of adults with aphasia ($n = 406$), and the number of new diagnoses of PSD in adults without aphasia ($n = 33$) by the total number of adults without aphasia ($n = 564$) using R Studio. Relative risk was calculated using MedCalc for Windows, version 19.5.3 (MedCalc Software, Ostend, Belgium).

Focusing exclusively on the group representing adults with aphasia, the selected variables of age, gender, race, and aphasia scores were used to explore potential risk factors contributing to the development of PSD ($\alpha = .05$). Selected comorbidities (obesity; history of drug or alcohol abuse; previous stroke or transient ischemic attack; coronary artery disease or prior myocardial infarction; dyslipidemia; diabetes mellitus; atrial fibrillation; history of smoking; hypertension; history of depression indicated by previously prescribed antidepressants; see Appendix C) were initially included in the analyses, however, no significant relationships were found to exist in any of the models explored, therefore those analyses were excluded from the results.

4 RESULTS

4.1 Descriptive Statistics

Records of cases with conditions mimicking stroke (intracranial bleeding, epileptic seizures, etc., $n = 114$) and records with no reported depression scores ($n = 11$) were removed from the dataframe, leaving a total of 970 records that were included for analysis. Patients' ages ranged from 19 to 112 years ($M = 64.67$, $SD = 14.925$). Forty-six percent of patients identified as female, while 54% identified as male. Regarding race, less than 1% identified as American Indian or Alaskan Native, 1% identified as Asian, 60% identified as Black or African American, less than 1% identified as Native Hawaiian or Pacific Islander, 28% identified as White, and approximately 10% remained unidentified. Of the entire sample, 42% presented with aphasia, and 22% with post-stroke depression (see Appendix D).

4.2 Incidence Proportions and Relative Risk

The incidence proportion of PSD in adults with aphasia was 43.3% while the incidence proportion of PSD in adults without aphasia was 5.8% over the course of one calendar year (2019) in Grady Memorial Hospital's acute care setting. Relative risk showed that adults with aphasia are 7.408 times more likely to exhibit PSD than adults without aphasia (95% CI: 5.2249 – 10.5057, $p < 0.001$).

4.3 Post-stroke Depression and Aphasia

Logistic regression analyses were conducted to explore potential risk factors for the development of PSD in adults with aphasia. Specifically, separate models explored whether the likelihood that an adult with aphasia in the acute care setting would exhibit PSD was related to age, gender, race, or aphasia severity. No significant relationships were found to exist between age and PSD ($p = 0.935$), gender and PSD ($p = 0.889$), or race and PSD ($p = 0.980$). A

significant relationship, however, was found to exist between aphasia severity and PSD ($p < .001$), with patients 2.06 (OR) times more likely to experience PSD with every 1-point increase in aphasia severity (95% CI: 1.605 – 2.678).

5 DISCUSSION

The purpose of this study was to assess the incidence of PSD in adults with aphasia compared to the incidence of PSD in adults without aphasia and to identify risk factors for PSD in adults with aphasia. Overall, the results suggest that the incidence proportion of PSD in adults with aphasia is much higher than in adults post-stroke and without aphasia. The results are comparable to those reported in earlier studies that recruited participants much later following stroke (see Shehata, et al., 2015; Wang, et al., 2018). This study was unique in its focus on aphasic cases in an acute care setting; the presence of aphasia was not an exclusion criterion, and the PHQ-2 was administered immediately following recovery from stroke.

Because the determinant depression scores were obtained before hospital discharge following recovery from stroke, more immediate referrals to the appropriate services would have been possible. Pursuing therapy to develop helpful coping mechanisms is a common respite from persistently hurtful interactions with which adults with aphasia contend (see Baker, et al., 2018; Gabriela, Lazarescu, & Kozma, 2016; Haley, et al., 2015; Northcott & Hilari, 2011). Yet, while mental health professionals may know what aphasia is, not many have the experience or the desire to provide services to persons living with it (Morrow-Odom & Barnes, 2019). This study's results showed that with greater severity of aphasia comes an increasing likelihood of contending with PSD. Consequently, many of the needed counseling services are unavailable, and with neither adequate psychological support nor social support, the symptoms of PSD are likely exacerbated (Baker, et al., 2018), and in that way fuel a vicious and draining cycle.

Findings also suggest that the potential risk factors of age, gender, race, and selected comorbidities shared no significant relationship with PSD controlling for the presence of aphasia. Curiously, this is different than the findings of earlier work focused on PSD (see

Abbas, et al., 2017; de Coster, et al., 2005; Fei, et al., 2016; Jia, et al., 2009; Jiang, et al., 2014; McCarthy, et al., 2016; Nakase, et al., 2016; Paradiso & Robinson, 1998; Poynter, et al., 2009; Skånér, et al., 2007). In this study, only aphasia severity shared a significant relationship with PSD. This finding not only aligns with earlier evidence indicating the presence of aphasia is a risk factor for PSD, but also suggests aphasia severity is proportionate to the risk.

5.1 Limitations

Certain groups may have been underrepresented in the analyses. Although age was normally distributed and two genders each made up approximately half of the sample, race was more varying. From a total of 970 cases, only two identified as Native Hawaiian or Pacific Islander, only three identified as American Indian or Alaskan Native, and only 13 identified as Asian, each making up no more than 1% of the sample. Furthermore, demographics such as socioeconomic status and levels of education were unavailable and could be confounding (Backhouse, McHutchison, Cvoró, Shenkin, & Wardlaw, 2018).

5.2 Future Directions

Both PSD and its detriment to quality of life among persons living with aphasia are multifactorial in nature (Cruice, Hill, Worrall, & Hickson, 2010; de Ryck, et al., 2014). In addition to the psychosocial circumstances that later perpetuate its cycle, the confirmed presence of PSD in patients with aphasia could point to shared underlying biological mechanisms. The wide and varying range of symptoms associated with PSD (see de Coster, et al., 2005; Nakase, et al., 2016; Skånér, et al., 2007) are comparable to those associated with MDD, which are equally diverse and understudied (see Kennedy, 2008). It is interesting to note that MDD has higher prevalence among persons living with assorted comorbidities such as diabetes, asthma, and arthritis (Moussavi, et al., 2007). Although this particular study neither investigated the specific

symptoms of nor found comorbidities associated with PSD in adults with aphasia, future investigations may find certain sets of conditions to be correlated.

Among the questions that might be asked in future investigations focused on biological variables include whether similar circuitry is disrupted with MDD and with PSD in adults with aphasia. One of the limitations that was encountered in this study was the restricted availability of information regarding specific stroke lesion sites. Of all aphasic cases in this study ($n = 406$), an overwhelming majority ($n = 253$) presented with stroke lesions classified as “other” (see Appendix E). This is because, in those cases, stroke lesions were either not observed with imaging or were multifocal in nature. Studying a larger sample that has been stratified based on stroke lesion sites could yield better information. Furthermore, it appears most of the research supporting a correlation between PSD and stroke lesion site claims lesions occurring in the left frontal region pose the greatest risk (see American Psychiatric Association, 2015). Neither lesion laterality nor patient handedness were reported in this study. Considering that, in the majority of right-handed cases, the onset of aphasia occurs as a consequence of left hemispheric stroke lesions (Berthier, 2005), gathering a sample of aphasic cases exhibiting right hemispheric stroke lesions and running similar tests could benefit our understanding.

5.3 Conclusions

The incidence of PSD in adults with aphasia has been disregarded as the presence of aphasia is often listed as an exclusion criterion in studies investigating PSD. The results of this study indicated that adults with aphasia are over seven times more likely to contend with PSD than adults without aphasia, and that with greater aphasia severity comes an increasing likelihood of experiencing PSD. The simultaneous presence of PSD and aphasia compounds problems affecting stroke survivors’ quality of life. The use of modified depression measures such as the

PHQ-2 could help detect the presence of PSD in adults with aphasia in acute care settings.

Consistent screening in acute care settings could immediately identify the presence of PSD, allow healthcare professionals to provide referral to treatment, and, as part of future endeavors, help researchers draw the best conclusions regarding any associated risk factors.

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APPENDICES

Appendix A

INSTITUTIONAL REVIEW BOARD

Mail: P.O. Box 3999
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May 26, 2020

Principal Investigator: Jacqueline Laures Gore

Key Personnel: Dotson, Vonetta M; Laures Gore, Jacqueline; Zanella, Christina

Study Department: Gerontology

Study Title: The Incidence of Post-Stroke Depression in Adults with Aphasia in an Acute Care Setting

Submission Type: Application for Designation of Not Human Subjects Research

IRB Number: H20711

Reference Number: 360629

Thank you for your Application for Designation of Not Human Subjects Research. Based on the information provided, this submission has been determined to be not human subjects research. This correspondence should be maintained with your records.

Please do not hesitate to contact the Office of Research Integrity at 404-413-3500 if you have any questions or concerns.

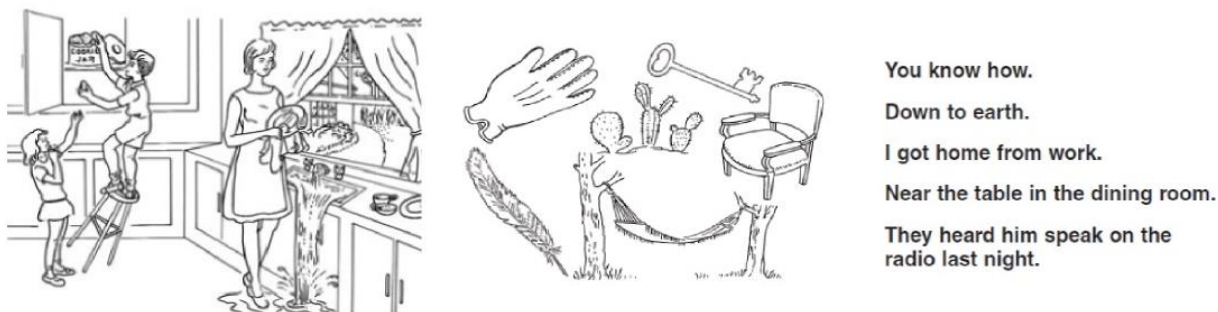
Sincerely,

A handwritten signature in cursive script that reads "Susan Vogtner".

Susan Vogtner, IRB Vice-Chair

Figure 1. IRB approval.

Appendix B



You know how.
Down to earth.
I got home from work.
Near the table in the dining room.
They heard him speak on the radio last night.

Figure 2. Images from the NIHSS.

National Institute of Neurological Disorders and Stroke (U.S.). (2011). NIH stroke scale. [Bethesda, Md.]: National Institute of Neurological Disorders and Stroke, Dept. of Health and Human Services, USA.

<p>9. Best Language: A great deal of information about comprehension will be obtained during the preceding sections of the examination. The patient is asked to describe what is happening in the attached picture, to name the items on the attached naming sheet, and to read from the attached list of sentences. Comprehension is judged from responses here as well as to all of the commands in the preceding general neurological exam. If visual loss interferes with the tests, ask the patient to identify objects placed in the hand, repeat, and produce speech. The intubated patient should be asked to write. The patient in coma (question 1a=3) will arbitrarily score 3 on this item. The examiner must choose a score in the patient with stupor or limited cooperation but a score of 3 should be used only if the patient is mute and follows no one step commands.</p>	<p>0 = No aphasia, normal 1 = Mild to moderate aphasia; some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of speech and/or comprehension, however, makes conversation about provided material difficult or impossible. For example in conversation about provided materials examiner can identify picture or naming card from patient's response. 2 = Severe aphasia; all communication is through fragmentary expression; great need for inference, questioning and guessing by the listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannot identify materials provided from patient response. 3 = Mute, global aphasia; no usable speech or auditory comprehension</p>
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Figure 3. Instructions for scoring aphasia taken from the NIHSS.

National Institute of Neurological Disorders and Stroke (U.S.). (2011). NIH stroke scale. [Bethesda, Md.]: National Institute of Neurological Disorders and Stroke, Dept. of Health and Human Services, USA.

Appendix C

Table 1. Selected comorbidities among aphasic and nonaphasic cases.

	<i>Aphasic (n = 406)</i>	<i>Nonaphasic (n = 564)</i>
<i>Selected Comorbidities</i>		
<i>Obesity</i>	22	34
<i>History of drug or alcohol abuse</i>	42	83
<i>Previous stroke or TIA</i>	119	195
<i>CAD or MI</i>	85	86
<i>Dyslipidemia</i>	94	148
<i>Diabetes mellitus</i>	137	193
<i>AFib</i>	83	78
<i>History of smoking</i>	109	169
<i>Hypertension</i>	316	438
<i>History of depression</i>	41	39

Appendix D

Table 2. Descriptive statistics of both aphasic and nonaphasic cases.

	<i>Aphasic</i> (<i>n</i> = 406)	<i>Nonaphasic</i> (<i>n</i> = 564)	<i>Overall</i> (<i>n</i> = 970)
<i>Age range in years</i>	23 to 99	19 to 112	19 to 112
<i>Mean age in years</i> (<i>SD</i>)	66.02 (15.140)	63.70 (14.705)	64.67 (14.925)
<i>Gender</i>			
<i>Male</i>	206	315	521
<i>Female</i>	200	249	449
<i>Race</i>			
<i>American Indian/Alaskan Native</i>	1	2	3
<i>Asian</i>	5	8	13
<i>Black or African American</i>	231	352	583
<i>Native Hawaiian/Pacific Islander</i>	0	2	2
<i>White</i>	129	146	275
<i>Undetermined</i>	40	54	94
<i>Aphasia scores</i>			
<i>Mean</i> (<i>SD</i>)	2.190 (0.838)	-	0.916 (1.209)
<i>None (NIHSS Score = 0)</i>	-	564	-
<i>Mild to moderate (NIHSS Score = 1)</i>	111	-	-
<i>Severe (NIHSS Score = 2)</i>	107	-	-
<i>Mute (NIHSS Score = 3)</i>	188	-	-
<i>Post-stroke Depression</i>			
<i>Absent (PHQ ≤ 2)</i>	230	531	761
<i>Present (PHQ > 2)</i>	176	33	209

Appendix E

Table 3. Stroke lesion sites among aphasic and nonaphasic cases.

<i>Stroke Lesion Sites</i>	<i>Aphasic (n = 406)</i>	<i>Nonaphasic (n = 564)</i>
<i>Basilar artery</i>	6	3
<i>CAB</i>	2	-
<i>Cervical ICA</i>	23	18
<i>Intracranial ICA</i>	3	-
<i>MCA (M1)</i>	95	84
<i>MCA (M2)</i>	20	18
<i>MCA (other)</i>	3	3
<i>Vertebral artery</i>	1	2
<i>Other</i>	253	436