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Post-Stroke Posttraumatic Stress Disorder: A Review

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Subject Terms

cognitive impairment; cerebrovascular disease/stroke; mental health

Introduction

Despite a recent decline in mortality[1], cerebrovascular disease (stroke) remains the fifth leading cause of death in the United States and the leading preventable cause of disability[2]. Furthermore, quality of life (QOL) following stroke depends on more than retention of physical functioning and ability[3]. Psychological health and well-being are known to associate with QOL outcomes[4]; for example, an estimated 30% of stroke survivors develop depression[5]. While post-stroke depression (PSD) is a well-researched and established phenomenon[6–8], far less is known about the development of post-stroke posttraumatic stress disorder (PTSD)[9]. Researchers have hypothesized that acute medical conditions such as asthma attack[10] and myocardial infarction[11] can precipitate PTSD, but PTSD specifically following a stroke has only been formally conceptualized for less than two decades[12]. Despite the relative paucity of research on post-stroke PTSD, this paper attempts to consolidate what is currently known: elaborating on the construct of post-stroke PTSD, weighing the conflicting prevalence reports in the literature, exploring identified or hypothesized premorbid and post morbid predicting factors, and summarizing promising future directions for research. Identifying gaps or conflicting findings may facilitate future studies to identify risk factors for post-stroke PTSD and establish a more efficacious management strategy.

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Epidemiology

Stroke is one of the leading causes of disability in adults[2]. With estimated prevalence rates of 20–65%, depression is a pervasive consequence of stroke that has been linked to cognitive deficits including memory, nonverbal problem solving, attention, and psychomotor speed[13]. Likewise, depressive symptoms experienced up to twelve months following hospitalization for subarachnoid hemorrhage (SAH) are tightly associated with QOL, more so than both demographic variables -and hemorrhage severity[14]. But PSD is not the only mental health outcome that may follow stroke; post-stroke PTSD has similarly been shown to impact mental health and QOL as well[15]. Beyond mood dysregulation, post-stroke PTSD has also been associated with an increased risk of non-adherence to medication[16], which presents a great obstacle for long-term management of cerebrovascular and psychiatric disorders.

In its most recent iteration, the DSM-V defines PTSD as a “trauma and stressor-related disorder,” comprising of four core symptomatic clusters: intrusion, avoidance, negative alterations in cognitions and mood, and alterations in arousal or reactivity[17]. This disorder, therefore, is quite different from PSD: while both contain elements of negative alterations in cognition and mood, PTSD is differentiated by the hallmark findings of intrusive thoughts and avoidance of stimuli bearing reminders of the trauma, in addition to experiencing hyperrarousal and heightened fear (see table 1 for diagnostic criteria and screening measures). Notably, despite the differences in diagnostic criteria, a recent systematic review noted that no studies examined both psychological sequelae of depression and PTSD post-stroke; thus, overlap and comorbidity between these conditions has yet to be elucidated[7]. Yet, medical disorders are known to cause psychological trauma[18] (e.g. congenital heart disease[19], subarachnoid hemorrhage[20]). This is especially more common if the patient requires ICU care[21]. As psychological trauma is the core, key feature around which PTSD develops, many of these inpatient conditions can lead a patient to develop PTSD[21]. Indeed, the number of cerebrovascular conditions associated with PTSD is rising: transient ischemic attack[22], spontaneous cervical artery dissection[23], and subarachnoid hemorrhage[24] to name a few.

Despite the modest increase in identifying PTSD in stroke survivors, the raw prevalence of post-stroke PTSD has been very difficult to identify. Studies report conflicting incidence rates: 4%[25], 10%[12], 18%[24], 30%[22], and as high as 37%[26]. In an effort to sort through this significant variability, a meta-analysis of survivors of stroke and transient ischemic attack reported that one out of every four patients experienced significant PTSD symptoms in the year following a cerebrovascular event[27]. Overall, these findings are much higher than the prevalence of PTSD in the general population, which is estimated to be 6.8%[28].

However, these results are to be interpreted with the understanding that a few studies included transient ischemic attack as well as stroke. Furthermore, there is a scarcity of research that distinguishes between hemorrhagic and ischemic stroke subtypes when determining post-stroke QOL and the prevalence of post-stroke PTSD[29]; notably, recent meta-analyses have not made this distinction[27]. As the etiologic origin of stroke affects patients differently both in severity and nature of symptoms, these subtypes may differently predispose patients to developing PTSD. The discrepancies between various studies may

indicate either inconsistencies in study methodology (sample size, inclusion criteria, operationalization or definition of PTSD) or differences in the propensity for these various conditions to give rise to PTSD. If the latter is true, then it becomes imperative for future research to outline careful inclusion and exclusion criteria and be more specific in both reporting their findings and extrapolating to various patient populations. Regardless, with prevalence findings as variable as those reported above, it is clear that the medical community could benefit from more robust methodologies to discern the prevalence of post-stroke PSTD.

Predictive Factors of Post-Stroke PTSD

In the following section, we examine recent findings regarding variables that may predict post-stroke PTSD development, although most studies examining mental health outcomes of cerebrovascular events focus on depression. Studies tend to rely on linear modeling and correlation analysis to identify significant predictors. Here, we review demographic variables (age and sex), as well as psychological and socioeconomic variables of interest. Given that the etiology of PTSD in a non-stroke population is complex, multifactorial, and irreducible, we are not surprised to find that many studies examining post-stroke PTSD development report conflicting results. Nevertheless, there are a few promising trends that we discuss below, with the hope that future inquiries into these relationships can more clearly elucidate the mechanisms by which stroke can precipitate PTSD.

Age

Studies examining the impact of age on post-stroke PTSD vary in demographic composition and sample size. A small study in Switzerland found no significant relationship between age and PTSD symptoms following a non-severe stroke[30]. Another UK study on stroke patients similarly found no correlation between age and the number or severity of PTSD symptoms at follow-up[31]. A study on SAH patients 3-years post-discharge found that age did not significantly differ in patients with and without PTSD symptoms[32]. However, other studies conflict with these null findings. For example, in a large analysis of stroke patients in Northern Manhattan and the Bronx, the mean age of those with PTSD was 7 years lower than those without[33]. In an analysis on TIA patients, it was found that, while age distribution did not significantly differ between subjects with or without PTSD, a logistic regression model did reveal age to be a significant protective factor against PTSD symptoms, such that older patients were less likely to develop the condition[22]. Taken in combination, these studies indicate that perhaps younger age contributes to post-stroke PTSD development.

Sex

Despite being one of the most commonly investigated correlates, there is conflicting evidence as to whether sex affects the development of post-stroke PTSD. It has been known for decades that females are at greater risk for PTSD than males, with a lifetime prevalence ratio of 2:1 when compared to men[34]. However, given the qualitative difference in trauma experienced between medical and non-medical PTSD, it remains necessary to examine if sex differences exist in post-stroke development. Although a definitive consensus has not yet been reached, existing literature indicates that females may be more prone to developing

post-stroke PTSD than males: in a large analysis of stroke and TIA survivors, patients who exhibited symptoms consistent with PTSD were over twice as likely to be female than male[33]. In fact, another study found that, of all medical and socio-demographic variables examined in stroke survivors, only sex associated with PTSD symptoms or diagnosis upon follow-up[35]. However, there are studies in which sex and post-stroke PTSD are independent variables[31].

Psychological Variables

Studies examining psychological predictors of post-stroke PTSD most commonly include negative cognitive appraisals, maladaptive coping, alexithymia, and avoidance. Many studies show there is an association between dysfunctional coping strategies and the severity of PTSD symptoms[36–37]. Negative cognitive appraisals are tainted, distorted perceptions that cause an individual to have negative feelings about an event they experience; these appraisals are intrinsic, personal qualities of cognitive styles. There is evidence that patients who have negative cognitive appraisals also have worse mental health outcomes post-stroke[38–39]. In post-stroke PTSD specifically, cognitive appraisals are predictive of symptoms such that greater negative trauma appraisals resulted in more PTSD symptoms[30]. Independently, another study found a significant correlation between measures of negative cognitions about one's self and the world and the severity of PTSD symptoms following a stroke[25]. These findings illustrate that negative cognitive appraisals are a construct that have some predictive value on the development of post-stroke PTSD.

Another construct that may affect post-stroke PTSD are coping strategies. Maladaptive coping strategies are ways in which individuals cope with stress and adversity that are poorly adaptable and correlate with burnout, depression, and anxiety. Although few studies have specifically examined coping strategies in a stroke population, one study has shown that maladaptive coping strategies are predictive of post-stroke PTSD symptoms on follow-up[22]. In addition, alexithymia, a psychological description of the difficulty or inability to convey and identify emotions, has been found to associate with post-stroke PTSD as well. In a prospective study, Alexithymia at baseline predicted severity of symptoms on follow-up[40].

Ultimately, the landscape of these studies is murky. Studies suffer from small sample sizes and non-uniform follow-up times. In addition, few studies examine the interplay between a myriad of psychosocial factors, instead just examining one factor at a time in different samples. It is possible that broadening the scope of the constructs included may allow researchers to sort through these relationships and identify the factors that are most predictive.

Socioeconomic Status

Prior research has examined associations between specific socioeconomic variables (income, level of education, race, etc.) and post-stroke PTSD. Unfortunately, relationships have thus far been difficult to elucidate. For example, lower education and unemployment status proved to predict post-stroke PTSD in one study conducted in Harlem, New York[33]. Additionally, in a study from Geneva, Switzerland, lower levels of education (fewer than 11 years) correlated with post-stroke PTSD symptoms[30]. However, other studies have not

shown this relationship[25, 31,35,41]. Furthermore, the relationship between unemployment and post-stroke PTSD is potentially confounded by stroke severity, such that a more severe stroke prevents employment from being feasible and elicits a stress disorder response in patients[33]. Also, different studies have not uniformly operationalized or measured socioeconomic predictors. Income and employment were found to be significant in studies in which they were included[33]; though many others did not consider them[25, 30,35,41]. Robust socioeconomic variables that predict post-stroke PTSD have yet to fully determined.

Insurance Status

While insurance status is not an issue in some European countries, it has been shown to affect QOL in other countries such as the United States. For the countries in which insurance status remains an issue, it is an important variable to consider with respect to post-stroke PTSD. Previous research has focused on the association between insurance status and PSD, rather than post-stroke PTSD, finding correlations between uninsured status and depressive symptoms[42]. However, the relationship between PTSD (and post-stroke PTSD) and insurance status remains relatively unexplored, with few studies differentiating between the insurance statuses of patients pre- and post-stroke. In an attempt to elucidate post-stroke PTSD predictors in a population of patients in Harlem, one study accounted for the insurance status of patients, noting whether they had commercial insurance, Medicare, or Medicaid (this category also includes those who are uninsured) at time of admission[33]. Medicaid (or low income) status conferred an increased risk of developing post-stroke PTSD[33]. Few other studies directly relate insurance status, stroke, and PTSD. One study with indirect implications regarding this relationship investigated medication adherence, finding post-stroke PTSD patients to demonstrate more “ambivalence towards medications” than those without post-stroke PTSD[16]. Given the nature of insurance—to provide compensation for medical needs—uncertainty about taking medication may result from insurance status-related factors. Understanding this relationship is critical for future research, as insurance status and medication non-adherence by proxy impair positive QOL outcomes.

Type of Stroke

As indicated previously, the etiologic origin of the stroke, hemorrhagic or ischemic, may unequally predispose individuals to developing PTSD, given that symptomatic presentation and morbidities are different. While there are no large analyses that specifically address this question, there are a few studies that examine post-stroke PTSD in patients following hemorrhagic stroke, namely SAH, in order to identify more specific predictors. In a prospective cohort study on SAH patients, sleep and fatigue indices upon follow-up were highly correlated with worse PTSD symptom severity[26]. A study on a similar sample found that fear, specifically about the recurrence of an SAH, was highly associated with experiencing PTSD symptoms[43]. These findings suggest that there are specific factors associated with hemorrhagic stroke associated with post-stroke PTSD; it remains to be seen if these identified factors are unique to SAH, or if they are associated with more stroke subtypes as well.

Pathophysiology and Biological Correlates

While there have been many discoveries made in recent years about the neural underpinnings of PTSD in the general population[44–45], far less is known about the biological correlates of PTSD following stroke. Understanding the effect of lesion location, or if a patient has a biological predisposition to adverse mental health outcomes, would be useful in any case. This is especially true here given the distinct medical and biological nature of post-stroke PSTD as compared to non-medical PTSD, which lacks a clear physiological precipitant. Therefore, here we briefly review relevant findings and discuss future directions for research.

It has been demonstrated that there are replicated structural abnormalities in the cortex between patients who suffer from PTSD. The most common macroscopic abnormality is a reduction of the volume of the ventromedial pre-frontal cortex, as well as reduced hippocampal volume[44, 46]. However, it remains unclear whether the structural findings are a diathesis in the development of PTSD, or rather a consequence of experiencing stress[47]. Nevertheless, researchers have proposed that the ventromedial pre-frontal cortex plays a role in suppressing attention allocation to trauma-related stimuli in healthy subjects, and attenuation of this response explains the experience of PTSD in patients[48]. Intrahemispheric lesion location in stroke patients has been postulated to have an effect on the development of mood disorders[49], and it is possible that the site of intracerebral bleeding might affect post-stroke PTSD development. Indeed, more recent neuroimaging studies examining the effect of local lesions in trauma-exposed (non-stroke) individuals found that lesions in the amygdala or ventromedial pre-frontal cortex resulted in a reduction of PTSD symptom intensity, rather than conferring increased risk[50]. These findings, while relevant here, have caveats: first, the population of patients did not involve stroke survivors, thus only indirectly addressing the question of post-stroke PTSD, and the finding contradicts the hypothesis that damage to the ventromedial pre-frontal cortex or amygdala would result in more severe post-stroke PTSD. Unfortunately, to date, there are no studies that we are aware of that examine the impact of stroke location on the development of post-stroke PTSD, and those that have attempted to characterize lesion effect on PSD have been inconclusive[51]. It is conceivable, given previous findings in non-stroke populations, that the location of intracranial bleeding and tissue infarction may shape psychological prognosis.

Beyond macroscopic findings from imaging or post-mortem research, dysregulation of neuroendocrine functioning has been implicated both in PTSD and depression in a post-stroke population. The hypothalamic-pituitary axis responsible for cortisol secretion at the adrenal glands has been particularly closely examined; it has been found that lower baseline levels of cortisol are linked to PTSD[52]. As Pitman and colleagues point out, the idea that a cortisol deficit plays a role in PTSD pathogenesis is corroborated with findings that abnormal glucocorticoid levels interfere with memory, learning, stress adaptation, and resilience[44, 53]. Furthermore, a recent report on the administration of high-dose hydrocortisone following trauma exposure in a human population reported that the treatment resulted in reduced risk of PTSD development[54]. Again, these findings, though not from a stroke-specific population, indicate that deficits in cortisol may predispose an individual to develop PTSD.

Post-Stroke PTSD Affects Quality of Life

Stroke impacts patients beyond the immediate physiological complications; post-stroke PTSD can present longitudinal challenges to patients. Experiencing PTSD symptoms can represent a novel problem for the patient after suffering a stroke: it has been found that fewer than half of people diagnosed with post-stroke PTSD have ever experienced stress disorder symptoms in the past[24], a result that differs from depression, in which 77% of those with PSD had a depressive episode in the past. One study on SAH patients found that, mental and physical QOL scores were predicted by post-stroke PTSD scores[26]. Unfortunately, meta-analyses have not accounted for the impact of PTSD or PSD symptoms on QOL, so it is difficult to tease apart the independent effect of either disorder[29]. Finally, there are few studies examining the direct impact of PTSD symptoms specifically on QOL or physical disability in a post-stroke population. Given all that we know about the importance of PSD in patient outcomes and mortality[8], it would appear that further research on post-stroke PTSD is warranted.

Management of Post-Stroke PTSD

While much has been published on PTSD following acute medical events, fairly few treatment guidelines have been published regarding cerebrovascular-specific etiologies[9]. In fact, there are virtually no reports that examine the benefits of psychotherapy in a post-stroke patient population suffering from PTSD; however, a few on post-stroke depression do exist[55–57]. The most recent examination of different interventions for ischemic stroke found that, beginning 6 months after a stroke, citalopram (anti-depressant) and cognitive behavioral therapy are more successful at reducing depressive symptoms than rehabilitation alone[55]. Notably, data suggests that 9 months after a stroke, cognitive behavioral therapy may be more effective than Citalopram[55]. Problem-solving therapy[56] and art therapy[57] as PSD treatments have also been proposed, but without controlled empirical findings. Unfortunately, none of these therapeutic approaches for PSD have been directly compared with each other to establish a gold standard. Furthermore, it remains to be seen whether they would be beneficial for patients with post-stroke PTSD; given that psychological approaches to managing other post-stroke psychiatric conditions exist, perhaps similar treatments may exist for post-stroke PTSD.

While treatment approaches for PTSD exist[58], there have not been any controlled trials examining the efficacy pharmacological or psychotherapeutic interventions for post-stroke PTSD, specifically. As management of PTSD and depression differ, approaches for these psychiatric conditions should be distinguished in a post-stroke population as well.

Conclusion

Ultimately, much remains unknown about post-stroke PTSD. There are controversial reports regarding prevalence, morbidity, and predictors. Although there are promising findings regarding age and psychosocial variables, many studies have been hindered by differing operationalizations and methodologies. Post-stroke PTSD is a common and debilitating

consequence of stroke, and there is much that still needs to be learned about all the factors that contribute to its effects on patients.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

This table illustrates the differences in diagnostic criteria, screening tools, and questionnaires between depression and posttraumatic stress disorder. There are no widely accepted differences in criteria for diagnosing an individual following a stroke, specifically[13].

Major Depressive Disorder	**Posttraumatic Stress Disorder**
• Five or more of the following symptoms over 2 weeks: **1.** Depressed mood **2.** Anhedonia **3.** Weight loss or weight gain **4.** Insomnia/hypersomnia **5.** Psychomotor agitation **6.** Fatigue **7.** Feelings of worthlessness or guilt **8.** Diminished concentration **9.** Suicidal ideation • The symptoms cause clinically significant distress and/or impairment • The episode is not attributable to the physiological effects of a substance or medical condition	• Exposure to or indirectly witnessing a stressor • The traumatic event is re-experienced by intrusive memories, nightmares, intense distress, etc. • The individual seeks to avoid reminders of trauma-related stimuli • The individual experiences negative alterations in cognition and mood (which can resemble depression, though not necessarily) • Alterations in arousal/reactivity: hypervigilance, etc. • Symptoms persist for over a month, have clinical significance, and are not attributable to another substance or illness
Common Screening Tools and Questionnaires[8,23]	
Beck Depression Inventory	Clinician Administered PTSD Scale
Hamilton Depression Rating Scale	Impact of Event Scale
Major Depression Inventory	Structured Clinical Interview for *DSM-V*
Patient Health Questionnaire (9 or 10 items)	Posttraumatic Stress Diagnostic Scale
Structured Clinical Interview for *DSM-V*	PTSD Checklist Specific for Stroke