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Review Article

Review on stroke induced depression

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ABSTRACT

One of the frequent and dangerous aftereffects of stroke is post-stroke depression (PSD). About one in three stroke survivors had depression following their stroke. It had a significant impact on functional recovery, which resulted in a low standard of living. Even worse, there is a clear correlation between it and a high death rate. Our goal in doing this evaluation was to come up with a thorough and cohesive knowledge of PSD based on both recently released research and well-known works. We discovered that the incidence of PSD varies from 11 to 41% within a two-year period, based on a significant number of researches. The severity of the stroke, the location of the lesion, past history of depression, and other factors all has a role in the development of PSD. The DSM criteria are currently the primary basis for diagnosing PSD, and they are often coupled with different depression scales. However, there isn't a single, cohesive process that explains PSD which now include aberrant neurotrophic response, elevated inflammatory markers, lowered monoamine levels, glutamate-mediated excitotoxicity, and dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis. Pharmacotherapy and psychosocial therapies are currently used in the treatment of PSD. Even though researchers have made significant progress, many problems still need to be solved. In particular, the PSD's mechanism is not entirely understood.

Keywords: Depression, HPA axis dysfunction, Stroke, Death, DSM-5 criteria

INTRODUCTION

The incidence and death of stroke have steadily decreased during the past 50 years. The primary cause of this reduction is the enhanced acute care provided to stroke patients. However, around two-thirds of stroke victims continue to have a persistent impairment necessitating the need for rehabilitation services. Neuropsychiatric (NP) disorders have been observed in stroke survivors, which can have a detrimental impact not only on social relationships and general quality of life, but also on the recovery of motor function. Following a stroke, it is common to see depression, anxiety disorder, catastrophic reaction, or psychosis.[1]

Stroke is described as an abrupt stoppage of blood flow to the brain that results in thrombotic, embolic, or hemorrhagic events that cause irreversible tissue damage. Depression, anxiety disorder, apathy, cognitive disorder, mania, psychosis, pathological affective display, catastrophic reactions, lethargy, and anosognosia are some of the NP conditions that have been linked to cerebrovascular disease.[2]

A depressive state following the beginning of a stroke is frequently referred to as PSD. PSD affected about one-third of stroke survivors. According to a 1977 study by Folstein et al stroke patients had a higher likelihood of depression (45% versus 10%) than orthopedic patients.

Depressive symptoms are substantially linked to greater mortality and have a poor relationship with functional outcome.[3] This article will briefly discuss PSD incidence, risk factors, evaluation and treatment, and prevention.

EPIDEMIOLOGY

An estimated 30-35 percent of people have PSD on average, with a range of 20 to 60%.

The primary cause of the high variability in the prevalence rate is methodological issues, such as the use of different diagnostic criteria, different depression rating scales, different evaluation timing, and different patient enrollment criteria in their prospective study. It was preferred to assess depressive symptoms as a result of stroke rather than depression because fewer patients met the DSM-IV criteria for depression, indicating a higher incidence of depressive symptoms than depressive syndromes.[4] After a stroke, the prevalence of PSD appears to peak three to six months later and then fall to roughly 50% of the initial rates after a year. Due to their impairment, PSD appears to be more common among Asian and aphasic patients as well as hospitalized stroke patients receiving in-patient therapy.[5]

There have been reports of gender variations in PSD frequency, with women diagnosed with serious depression twice as often as males. Over time, the incidence of PSD continues to be high, reaching 29% at the 3-year follow-up and roughly 19% at the 7-year follow-up.[6]

RISK FACTORS FOR PSD

Genetic factors

Polymorphisms in the serotonin transporter gene (SERT) are thought to be related to PSD. Two genetic variants of SERT are the 5-HTTLPR and the STin2 VNTR.[7] Studies have linked methylenetetrahydrofolate reductase (MTHFR) and apolipoprotein E (ApoE) to higher risks of major depressive disorder (MDD) and stroke.[8]

Age

Thirteen studies found no association between older age and PSD, whereas three studies indicated a relationship between older age and PSD. Furthermore, the majority of PSD patients were older, according to a recently released study. As such, it is unclear if age raises the risk of PSD.

Sex

Previous research revealed that a woman's gender increased her likelihood of acquiring PSD. A study conducted in 2018 included 259 patients with ischemic stroke, and after a year, 94 of them were diagnosed with PSD. They discovered that female patients with depression were more common. According to a systematic review, seven studies linked gender to PSD, while thirteen studies showed no correlation between gender and PSD. It is still up for discussion as a result.[9]

History of depression

Depression before to a stroke may raise the risk of stroke. Stroke risk is independently correlated with depression. Conversely, a higher risk of PSD may result from depression experienced before a stroke. The most current meta-analysis of pre-stroke depression found that the odds of PSD were significantly increased by depression before to stroke, with an average prevalence of 11.6%.[10]

Stroke severity

According to a systematic evaluation of observational studies evaluating the relationship between PSD and stroke severity, PSD and stroke severity were positively correlated.[11]

Lesion location

A large body of research indicated that PSD was highly correlated with the site of the brain injury. According to Robinson a key factor in determining the degree of depression is the proximity between the frontal pole and the brain lesion area.[12] Lesions that specifically damaged the left side pre-fronto-subcortical circuits linked to depression were found in PSD patients, who also had a greater number and volume of infarcts.[13]

Other factors

There was a strong correlation between depression symptoms and a lack of social support. Patients with acute ischemic stroke who were married were more likely to experience all negative outcomes, particularly if they had only completed middle school.[14]

DIAGNOSIS

The four main clusters of symptoms that make up the clinical picture of depression are mood changes (depressed mood, lack of interest), psychomotor changes (weakness, anxiety, psychomotor impairment), cognitive changes (insecurity, regret, suicidal thoughts, social isolation), and body or neurovegetative changes (early arousal or changes in circadian rhythm, changes in appetite, problems with concentration).

Primary criteria for identifying depressed stroke patients

Hamilton depression rating scale (HDRS)

Because of its established validity and reliability in measuring depressed symptoms in stroke patients, the HDRS has been utilized extensively by researchers and medical professionals. It has 2 versions (14 and 21 items). It depends on somatic and behavioral symptoms.

Sensitivity in stroke populations: 71% (cut- off 11) and specificity in stroke populations: 87% (cut- off 11).[15]

Patient health questionnaire-9 (PHQ-9)

A number of studies have shown the validity of using the PHQ-9 to screen for major depressive disorder (major depression). The PHQ-9 has nine questions. PHQ has been shown to have more diagnostic validity and efficiency when compared to the initial primary care evaluation of mental disorders (PRIME-MD), which was carried out by clinicians.[16]

General health questionnaire (GHQ)

Originally, Goldberg developed another scale known as the GHQ. The GHQ score demonstrated the validity and reliability of the GHQ to measure the PSD because it was highly correlated with the scores of other depression rating scales.[17]

Beck depression inventory (BDI)

Beck created the BDI in 1961, which includes the 21-items and assess six of nine symptoms of DSM III criteria. Many investigators have also used it as it is very simple, not time consuming and no training is required to admister. Clinicians can determine if patients are depressed and to what extent based on the overall score but it cannot discriminate between major and minor depression.

Sensitivity in stroke populations: 80% (cut-off 10) and specificity in stroke populations: 61% (cut-off 10).[18]

Center for epidemiological studies depression scale (CES-D)

A brief self-assessment tool, excellent internal stability, and sufficient test-retest reliability, which includes 20 items. When comparing participants 60 years of age/older to DSM-IV criteria, increased sensitivity and specificity are seen and not determined in PSD evaluation.

Sensitivity in stroke populations: 73-86% (cut-off 16) and specificity in stroke populations: >90% (cut-off 16).[19]

Geriatric depression scale (GDS)

Includes 30-items, specifically designed to screen for depression in the elderly, sensitivity is impacted by female gender and cognitive disability.

Sensitivity in stroke cohorts: 85% (cut-off >11) and specificity in stroke cohorts: 64% (cut >off 11).[20]

PSD prediction scale (DePreS)

It is a useful tool to help doctors and clinicians determine how high the risk of depression is in the first week following a stroke. Includes 10 items. Expertise and training are required.[21]

Hospital anxiety and depression scale (HADS)

It is a two-dimensional device and includes 14 items, evaluates depression and anxiety. Somatic symptoms of depression are excluded.

Sensitivity in stroke patients: 91.7% (major depression) (cut- off 5), 88.5% (major and minor depression), specificity in stroke patients: 56% (major depression) (cut-off 5) and 71% (major and minor depression).[22]

Diagnostic and statistical manual of mental disorders (DSM-IV)

DSM-IV-TR is typically used to define PSD. A significant issue with the DSM-IV criteria for PSD is that it also requires the presence of some somatic symptoms (fatigue, loss of energy, weight loss, decreased appetite, sleeplessness, difficulty concentrating, and psychomotor changes) that may also be symptoms of a stroke. In this sense, it is easy to overestimate depression in stroke patients when in fact some symptoms may be directly related to the physical condition.[23]

Zung self-rating depression scale

Self-assessing scale: fully, precisely, and promptly capture the pertinent symptoms, their intensity, and variations in the person's depression

It has been suggested that these depression screening tools be used in combination. While HDRS and PHQ-9 demonstrated the highest specificity, BDI and HDRS exhibited the highest sensitivity.[24]

PATHOPHYSIOLOGY

Below is a description of the primary pathophysiological mechanisms of PSD.

HPA axis dysregulation

The primary neuroendocrine stress response system that controls mood, immunity, and metabolism is the HPA axis. Corticotropin-releasing hormone (CRH) is released by the paraventricular nucleus of the hypothalamus in response to signals from the hippocampal or other tissues. This triggers the pituitary to release adrenocorticotropic hormone (ACTH). Lastly, glucocorticoid synthesis and release are mediated by ACTH on the adrenal cortex. A negative regulation of the HPA axis activity is caused by the effects of glucocorticoids on the hippocampus, hypothalamus, and pituitary. However, it controls the survival and genesis of neurons in the brain. The amounts of cortisol in a patient's urine, plasma, and saliva increased when they were depressed. [25] There are two different kinds of glucocorticoid receptors (GR): GR

and the mineralocorticoid receptor (MR). The HPA-system's glucocorticoid feedback inhibition is mediated by the interaction between MR and GR activation. Therefore, the strong data previously discussed suggested that the HPA axis may be a target for the PSD and that it may play a significant part in the pathogenesis of the disease.[26]

Enhanced inflammation and injury

Smith initially put out the "macrophage theory of depression," which suggested cytokines had a part in depression. TNF-α (tumor necrosis factor) and interleukin-1α (IL-1α), interleukin-1β (IL-1β), and interleukin-6 (IL-6) are examples of these proinflammatory cytokines. [27] Elevations of C-reactive protein (CRP) and IL-6 in blood or plasma were commonly seen in patients with serious depressive disorders. Three mechanisms exist for inflammation to increase HPA axis activity: (1) cytokines acting directly on the brain; (2) glucocorticoid resistance being induced; and (3) indirectly altering GR function. Following damage to the neurons, cytokines are released, and these can activate microglia, which in turn causes inflammation in the affected area. After all, stroke victims experience depression as a result of inflammation.[28]

Alteration in neurotransmitters

The monoaminergic hypothesis and glutamate-mediated excitotoxicity are two hypotheses on depression that are related to neurotransmitters. Monoamines, primarily 5-hydroxytryptamine (5-HT, or serotonin), dopamine (DA), and norepinephrine (NE), have a significant impact on the brain. Patients with depression had lower monoamine levels. [29] The altered concentration of plasma 5-HT, which is a measure of the 5-HT levels in cerebral-spinal fluid (CSF), is therefore a crucial clinical indicator following a stroke and can be utilized to forecast mental illness that may develop after the stroke. Treating PSD may involve the use of serotonin transport inhibitors. Remarkably, the metabolism of monoamines is influenced by the CNS inflammatory response as well. [30] Glutamate levels in the brain and plasma would increase following an acute stroke. Furthermore, cerebral glutamate levels can rise in response to inflammation. Research revealed that the CSF of patients with depression had a considerably greater glutamate concentration than that of healthy controls. We hypothesize that stroke plays a role in the neurotransmitter changes that cause PSD, particularly in 5-HT and glutamate. [31]

Neurotrophic theory in relation to neuroplasticity

A number of neurotrophic factors can prevent neurons from being harmed by depression, which can cause neurons in the limbic and cortical parts of the brain to die. Neurotrophic factors that influence restoration and brain rehabilitation following a stroke include insulin-like growth factor-1 (IGF-1) and brain-derived neurotrophic factor (BDNF).[32] Serum BDNF concentrations in PSD patients are lower than in the control group. Sertraline and desipramine, 2 common traditional antidepressants, have been shown to increase BDNF expression, which in turn boosts neuron survival and function. On other hand, low levels of brain-derived neurotrophic factor (BDNF) might exacerbate depression symptoms. Consequently, serum BDNF may be used as a PSD biomarker. [33]

TREATMENT

Pharmacotherapy

In comparison to patients receiving a placebo, those randomly assigned to receive nortriptyline (50-100 mg/day) experienced a considerably higher reduction in HAM-D scores throughout the course of six weeks of treatment.[34] Over the course of six weeks, the HAM-D scores of 33 post-stroke patients treated with citalopram (10 mg-20 mg/day) decreased considerably more than those of 33 similarly depressed individuals treated with a placebo. The usage of SSRIs has been linked to an increased risk of hemorrhagic complications and falls in the elderly. Lastly, the American heart association suggests that patients with PSD take antidepressants for a minimum of six months following their recovery. The first-choice medications for treating PSD are SSRIs, with tricyclics being second-line treatments.[35]

Psychotherapy

An equally significant strategy for managing PSD is psychological therapy. It is thought that depression can be effectively treated using cognitive behavioral therapy (CBT). On the other hand, a later Cochrane review that reviewed multiple trials on psychological therapy for depression revealed that CBT might be beneficial for older individuals with depression.[36]

Transcranial magnetic stimulation (TMS)

Moreover, a recent non-invasive experimental method for changing brain physiology is repetitive TMS. While 0.5 Hz low frequency rTMS can alleviate depression symptoms in PSD patients.[37]

Physical exercise

Physical activity and exercise programs, including aerobic exercises and strength training, have been found to improve mood and reduce depressive symptoms in stroke survivors.[38]

Electroconvulsive therapy (ECT)

ECT may be considered for severe and treatment-resistant PSD, especially when pharmacotherapy and psychotherapy have failed.[39]

Mindfulness-based interventions

Mindfulness-based stress reduction (MBSR) or mindfulness-based cognitive therapy (MBCT), have shown promise in reducing depressive symptoms and improving overall well-being in stroke survivors.[40]

Social support and rehabilitation programs

Engaging in social support networks and participating in stroke rehabilitation programs can provide emotional support, encouragement, and a sense of belonging, which may help alleviate symptoms of depression.[11]

Nutritional interventions

Adequate nutrition, including consumption of omega-3 fatty acids, antioxidants, and a balanced diet, may play a role in preventing or alleviating PSD.[41]

Sleep management

Addressing sleep disturbances through sleep hygiene practices, relaxation techniques, and, if necessary, pharmacological interventions for sleep disorders can improve mood and overall well-being in stroke survivors with depression.[42]

Complementary and alternative medicine

Some individuals may benefit from complementary and alternative medicine approaches such as acupuncture, yoga, or herbal supplements; however, evidence supporting their efficacy in treating post-stroke depression is limited and requires further research.[43]

CONCLUSION

Because of the potential harm to patients' recuperation, depression is a common and dangerous post-stroke consequence. Progressive PSD research has advanced significantly. As a result of various diagnostic standards, sample sizes, regional variations, stroke kinds, etc., the incidence of PSD varies from 11 to 41%. It is believed that the most dependable risk factors for PSD include lesion location, degree of stroke, and depressive history. A variety of scales, including the BDI, PHQ-9, HDRS, and CES-D, can be used to screen the PSD. The HPA axis dysfunction, increased inflammation, neurotransmitter shift, neurotrophic hypothesis, and neuroplasticity are currently thought to be pathophysiological processes of PSD. The two ways to manage PSD are medication and psychosocial therapies. Antidepressant use can ultimately significantly prevent and lower the occurrence of PSD. It would seem that stroke units should be organized with the use of therapeutic and preventive measures to lower the likelihood of aberrant mood and enhance the results of rehabilitation.

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