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POST-STROKE DEPRESSION: CLINICAL FEATURES, RISK FACTORS, AND APPROACHES TO MANAGEMENT

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Abstract
Post-stroke depression (PSD) is one of the most common neuropsychiatric complications following cerebrovascular accidents and significantly affects functional recovery, quality of life, and mortality. The prevalence of PSD varies widely depending on diagnostic criteria and timing of assessment, ranging from 20% to 60%. This review aims to summarize current knowledge on the clinical manifestations, risk factors, pathophysiological mechanisms, and management strategies of post-stroke depression. Special attention is given to biological, psychological, and social determinants contributing to the development of depressive symptoms after stroke. Early recognition and appropriate treatment of PSD are essential for improving rehabilitation outcomes and reducing long-term disability. Understanding the multifactorial nature of PSD allows clinicians to implement comprehensive and individualized therapeutic approaches.

Keywords: Post-stroke depression; ischemic stroke; rehabilitation; mental health; neurology.

Introduction

Stroke remains a leading cause of disability and mortality worldwide. In addition to motor and cognitive impairments, emotional and psychological disorders frequently complicate the recovery process. Among these, post-stroke depression is the most prevalent and clinically significant condition. PSD negatively influences rehabilitation adherence, cognitive recovery, and social reintegration. Despite its high prevalence, depression after stroke is often underdiagnosed and undertreated. This review discusses the main aspects of PSD, including its clinical presentation, risk factors, underlying mechanisms, and treatment strategies.

Materials and Methods

This narrative review was conducted to analyze current scientific evidence regarding post-stroke depression. Relevant articles were identified through a comprehensive search of electronic databases including PubMed, Scopus, Web of Science, and Google Scholar. Publications written in English and published between 2000 and 2024 were considered for inclusion. The search strategy employed key terms such as “post-stroke depression,” “ischemic stroke,” “cerebrovascular disease,” “mental health,” and “stroke rehabilitation.” Original research articles, systematic reviews, meta-analyses, and clinical guidelines focusing on the epidemiology, clinical features, risk factors, pathophysiological mechanisms, and management of post-stroke depression were included. Studies were excluded if they

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were not directly related to post-stroke depression, lacked sufficient methodological detail, or were published in languages other than English. Data extraction focused on study design, sample characteristics, key findings, and clinical relevance. The selected studies were analyzed qualitatively and synthesized to provide an integrated overview of post-stroke depression.

Results

Analysis of the selected literature demonstrated that post-stroke depression (PSD) is a frequent neuropsychiatric complication affecting a substantial proportion of stroke survivors. According to the reviewed studies, the reported prevalence of PSD varied from 20% to 60%, depending on the time of assessment, diagnostic criteria, and study design. Higher prevalence rates were commonly observed within the first six months after stroke, although depressive symptoms were also documented in the chronic phase.

Clinical manifestations of PSD were consistently described across studies. The most frequently reported symptoms included persistent low mood, anhedonia, reduced motivation, fatigue, sleep disturbances, impaired concentration, and decreased appetite. Emotional lability, anxiety, and apathy were also commonly observed. Several studies emphasized that depressive symptoms often overlapped with neurological deficits, leading to underdiagnosis in routine clinical practice. Regarding risk factors, multiple studies identified stroke severity as a significant predictor of PSD. Patients with moderate to severe neurological impairment demonstrated a higher likelihood of developing depressive symptoms. Lesion location was also associated with PSD, with strokes involving the frontal lobe, basal ganglia, and left hemisphere showing stronger correlations. Additional biological risk factors included older age, female sex, and the presence of cognitive impairment.

Table 1. Key Findings on Post-Stroke Depression Based on Reviewed Studies

Aspect	Key Findings
Prevalence	Post-stroke depression was reported in 20–60% of stroke survivors, with higher prevalence observed within the first 6 months after stroke.
Time of onset	Depressive symptoms most frequently developed in the early post-stroke period but could persist or appear in the chronic phase.
Clinical manifestations	Common symptoms included persistent low mood, anhedonia, fatigue, sleep disturbances, impaired concentration, anxiety, emotional lability, and apathy.
Neurological factors	Greater stroke severity and lesions involving the frontal lobe, basal ganglia, and left hemisphere were associated with a higher risk of depression.
Demographic risk factors	Advanced age, female sex, and cognitive impairment were identified as significant predictors of post-stroke depression.
Psychosocial factors	Lack of social support, reduced functional independence, social isolation, and a history of depression prior to stroke increased vulnerability to PSD.
Pathophysiological mechanisms	Alterations in serotonergic and dopaminergic pathways, neuroinflammation, dysregulation of the hypothalamic–pituitary–adrenal axis, and structural brain damage were commonly reported.
Treatment outcomes	Antidepressant therapy, particularly selective serotonin reuptake inhibitors, along with psychological interventions and multidisciplinary rehabilitation, were associated with symptom improvement.

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Psychosocial factors played a substantial role in the development of PSD. Lack of social support, reduced functional independence, and social isolation were repeatedly reported as important contributors. A history of depression prior to stroke significantly increased the risk of post-stroke depressive disorders. Several studies highlighted the interaction between biological vulnerability and psychosocial stressors in the onset of PSD. Pathophysiological findings reported in the reviewed literature suggested that PSD is associated with neurochemical and inflammatory changes following stroke. Alterations in serotonergic and dopaminergic neurotransmission, increased pro-inflammatory cytokine activity, and dysregulation of the hypothalamic–pituitary–adrenal axis were commonly described mechanisms. Structural damage to mood-regulating neural networks further contributed to depressive symptomatology.

In terms of management outcomes, studies evaluating therapeutic interventions indicated that pharmacological treatment with antidepressants, particularly selective serotonin reuptake inhibitors, was associated with a reduction in depressive symptoms. Non-pharmacological interventions, including psychotherapy and structured rehabilitation programs with psychological support, demonstrated additional benefits. Early screening and integrated multidisciplinary care were associated with improved emotional well-being and functional recovery.

Discussion

The findings of this review confirm that post-stroke depression is a highly prevalent and clinically significant complication of cerebrovascular disease. The reported prevalence rates ranging from 20% to 60% are consistent with previously published data, underscoring the substantial psychological burden experienced by stroke survivors. Variability in prevalence across studies may be attributed to differences in diagnostic criteria, timing of assessment, and study design. Clinical manifestations of post-stroke depression were found to be heterogeneous and frequently overlapped with neurological impairments. This overlap complicates clinical recognition and contributes to underdiagnosis, particularly in routine rehabilitation settings. The presence of apathy and emotional lability, in addition to classical depressive symptoms, highlights the need for structured screening tools specifically adapted for stroke patients.

The analysis of risk factors emphasizes the multifactorial nature of post-stroke depression. Neurological factors, including stroke severity and lesion location, play a critical role in mood regulation through disruption of frontal–subcortical circuits. However, psychosocial determinants such as social isolation, reduced functional independence, and lack of emotional support were equally influential. These findings support a biopsychosocial model of post-stroke depression, in which biological vulnerability interacts with environmental and psychological stressors. Pathophysiological mechanisms described in the reviewed literature further explain the complex relationship between stroke and depression. Neurochemical imbalances involving serotonergic and dopaminergic pathways, combined with inflammatory responses and hypothalamic–pituitary–adrenal axis dysregulation, appear to contribute to the development of depressive symptoms. Structural brain damage affecting mood-regulating networks may exacerbate these processes, leading to persistent emotional disturbances.

Regarding treatment outcomes, the reviewed evidence indicates that pharmacological interventions, particularly selective serotonin reuptake inhibitors, are effective in reducing depressive symptoms in

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stroke survivors. However, optimal management requires a multidisciplinary approach that integrates psychological support, rehabilitation, and family involvement. Early screening and timely intervention were consistently associated with improved emotional well-being and functional recovery. Several limitations should be acknowledged. As a narrative review, this study is subject to potential selection bias and does not provide quantitative synthesis of data. Additionally, heterogeneity among included studies limits direct comparison of outcomes. Nevertheless, the findings provide a comprehensive overview of current evidence and highlight key areas for future research.

Conclusion

Post-stroke depression is a common and clinically important complication that significantly affects recovery, functional outcomes, and quality of life in stroke survivors. The findings of this review highlight the complex and multifactorial nature of post-stroke depression, involving neurological damage, neurochemical and inflammatory processes, as well as psychosocial factors. Early identification and systematic screening for depressive symptoms should be considered an essential component of post-stroke care. Timely and individualized management strategies, combining pharmacological treatment with psychological support and multidisciplinary rehabilitation, can substantially improve emotional well-being and rehabilitation outcomes. Integrating mental health assessment into standard stroke rehabilitation programs may contribute to reduced long-term disability and improved overall prognosis. Further research is needed to develop standardized diagnostic tools and optimize therapeutic approaches for post-stroke depression.

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