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## POST-STROKE DEPRESSION: IMPACT ON ADHERENCE TO SECONDARY PREVENTION OF ISCHEMIC STROKE AND THE EFFECTIVENESS OF THERAPEUTIC INTERVENTIONS AT THE LEVEL OF PRIMARY HEALTH CARE. LITERATURE REVIEW

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### Abstract

**Introduction.** Post-stroke depression (PSD) is the most common psychiatric complication following a cerebrovascular event. In every second or third patient after a stroke, slowing recovery and worsening clinical outcomes. Despite the availability of validated screening instruments, PSD remains underdiagnosed and underestimated in routine practice.

**Objective.** To review current literature on post-stroke depression (PSD), its influence on adherence to secondary stroke prevention, and therapeutic interventions at the level of primary health care (PHC).

**Search strategy.** This review is based on the analysis of publications addressing the impact of PSD on adherence to secondary prevention of ischemic stroke, rehabilitation processes, and clinical outcomes, as well as screening and diagnostic methods applicable in primary health care. The literature search was conducted in PubMed, Scopus, eLibrary, and Cochrane Library databases for the period 2015–2025 using the following keywords: *stroke, post-stroke depression, PHQ-9, Beck Depression Inventory, primary care, screening, rehabilitation*.

**Results.** According to the literature, PSD is highly prevalent among stroke survivors. In post-Soviet countries, mental health problems remain stigmatized, leading to underrecognition and delayed diagnosis of PSD. Rehabilitation efforts are predominantly focused on motor and cognitive recovery, while depressive symptoms are often overlooked. PSD is associated with decreased treatment adherence, poorer rehabilitation outcomes, cognitive decline, and reduced quality of life.

**Conclusion.** Early detection and a comprehensive approach to PSD management at the PHC level improve adherence to secondary prevention and rehabilitation measures. Screening programs should be expanded and systematically integrated into routine follow-up care. Timely identification and treatment of PSD improve the quality of life for patients and caregivers engaged in the rehabilitation process.

**Keywords:** *stroke, depression, screening, secondary prevention, rehabilitation*.

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## Резюме

**ПОСТИНСУЛЬТНАЯ ДЕПРЕССИЯ: ВЛИЯНИЕ НА  
ПРИВЕРЖЕННОСТЬ К ВТОРИЧНОЙ ПРОФИЛАКТИКЕ  
ИШЕМИЧЕСКОГО ИНСУЛЬТА И ЭФФЕКТИВНОСТЬ  
ТЕРАПЕВТИЧЕСКОГО ВМЕШАТЕЛЬСТВА НА УРОВНЕ ПЕРВИЧНОЙ  
МЕДИКО-САНИТАРНОЙ ПОМОЩИ. ОБЗОР ЛИТЕРАТУРЫ**

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**Введение.** Постинсультная депрессия наиболее частое психическое нарушение после перенесенной мозговой катастрофы. По данным разных источников, постинсультная депрессия развивается у каждого второго-третьего пациента, пережившего инсульт, замедляя восстановление и утяжеляя исходы. Несмотря на наличие доступных валидизированных инструментов диагностики, постинсультная депрессия остается нераспознанным и недооцененным состоянием.

**Цель.** Обзор современных литературных данных посвященных постинсультной депрессии (ПИД), ее влиянию на приверженность к вторичной профилактике инсульта и терапевтическим вмешательствам на уровне первичной медико-санитарной помощи (ПМСП).

**Стратегия поиска.** Настоящий обзор основан на анализе публикаций, посвященных влиянию постинсультной депрессии (ПИД) на приверженность вторичной профилактике ишемического инсульта, реабилитационный период и клинические исходы, методам диагностики (инструментам скрининга) на уровне первичной медико-санитарной помощи (ПМСП). Поиск источников осуществлялся из баз данных PubMed Scopus e-Library, Cochrane Library за период с 2015-2025гг. с использованием ключевых слов: Stroke, post-stroke depression, PHQ-9, Beck Depression Inventory, primary care, screening, rehabilitation.

**Результаты.** Согласно литературным данным, ПИД имеет высокую распространенность среди пациентов, перенесших инсульт. В постсоветских странах проблема психического здоровья остается стигматизированной, что приводит к недооценке и поздней диагностике постинсультной депрессии. При этом реабилитационные мероприятия постинсультных пациентов направлены на восстановление утраченных физических функций и когнитивного дефицита, тогда как депрессивные расстройства часто игнорируются.

**Выводы:** Ранняя диагностика и комплексный подход в лечении ПИД на уровне ПМСП улучшает приверженность пациентов к вторичной профилактике и реабилитационным мероприятиям. Необходимо развивать программы скрининга и интегрировать их на уровень ПМСП. Чем раньше начато лечение ПИД, тем лучше качество жизни пациентов и их родных, вовлеченных в реабилитацию.

**Ключевые слова:** инсульт, депрессия, скрининг, вторичная профилактика, реабилитация.

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Түйіндеме

**ПОСТИНСУЛЬТТИК ДЕПРЕССИЯ: ИШЕМИЯЛЫҚ ИНСУЛЬТТИҢ  
ҚАЙТАЛАМА ПРОФИЛАКТИКАСЫНА БЕЙІЛДІЛІККЕ ӘСЕРІ ЖӘНЕ  
БАСТАПҚЫ МЕДИЦИНАЛЫҚ-САНИТАРИЯЛЫҚ КӨМЕК  
ДЕҢГЕЙІНДЕГІ ТЕРАПИЯЛЫҚ АРАЛАСУЛАРДЫҢ ТИІМДІЛІГІ.  
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**Кіріспе.** Постинсульттік депрессия – инсульттан кейін жиі кездесетін психикалық бұзылыс. Әдеби деректер бойынша, постинсульттік депрессия инсультті басынан өткерген науқастардың әр екінші-үшінші жағдайында дамиды, қалпына келу қарқынын баяулатып, клиникалық нәтижелерді нашарлатады. Қолжетімді және валидтелген скрининг құралдары бар болғанына қарамастан, постинсульттік депрессия әлі де жиі анықталмайды және жеткіліксіз бағаланады.

**Мақсат.** Постинсульттік депрессияның (ПИД) қазіргі ғылыми деректерін, оның инсульттің қайталама профилактикасына науқастардың бейілділігіне әсерін және бастапқы медициналық-санитариялық көмек (БМСК) деңгейіндегі терапиялық араласуларды талдау.

**Іздеу стратегиясы.** Бұл шолу PubMed, Scopus, e-Library және Cochrane Library деректер базаларындағы 2015–2025 жж. жарияланған материалдарды талдауға негізделді. Кілт сөздер: *stroke, post-stroke depression, PHQ-9, Beck Depression Inventory, primary care, screening, rehabilitation*. Талдауға ПИД-тің таралуы, оның клиникалық нәтижелерге және қайталама профилактикаға бейілділікке әсері, сондай-ақ БМСК деңгейінде скрининг әдістері жөніндегі деректер енгізілді.

**Нәтижелер.** Әдебиеттер көрсеткендей, ПИД инсульттан кейінгі науқастар арасында кең таралған. Посткеңестік елдерде психикалық денсаулық проблемалары жиі стигматизацияға ұшырайды, бұл ПИД-тің кеш анықталуына әкеледі. Реабилитациялық шаралар көбінесе қозғалыс функцияларын және когнитивтік тапшылықты қалпына келтіруге бағытталғанымен, депрессивті бұзылыстар көбіне назардан тыс қалады.

**Қорытынды.** ПИД-ті ерте кезеңде анықтау және БМСК деңгейінде кешенді емдеу науқастардың қайталама профилактикаға және реабилитациялық шараларға бейілділігін жақсартады. Скрининг бағдарламаларын дамыту және оларды БМСК жүйесіне енгізу қажет. ПИД-ті уақтылы емдеу пациенттердің және оларды күтетін жақындарының өмір сүру сапасын арттырады.

**Түйінді сөздер:** инсульт, депрессия, скрининг, қайталама профилактика, реабилитация.

**Дәйексөз үшін:**

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### Introduction

According to data provided by the World Health Organization (WHO), mortality from cardiovascular diseases continues to rise, increasing from 14 million deaths in 2000 to 17.9 million in 2019 [6]. Stroke and myocardial infarction together account for 85% of all cardiovascular deaths [6], with stroke remaining the second leading cause of mortality globally.

Post-stroke depression (PSD) is an affective disorder that develops at various time points after a stroke and is characterized by anhedonia (loss of the ability to experience pleasure), apathy (loss of interest and emotional indifference), anergia (reduced or lost ability to engage in purposeful activity), as well as cognitive and somatic symptoms [45]. According to ICD-10, this condition is coded under F06.3 "Organic mood (affective) disorders" in the Republic of Kazakhstan [5].

PSD is the most common and clinically significant neuropsychiatric complication of stroke [8]. Its prevalence varies widely, from 20% to 60% across different studies [27]. Underrecognition and delayed diagnosis contribute to reduced adherence to treatment and rehabilitation programs, thereby worsening clinical outcomes [14]. PSD is also associated with more pronounced cognitive impairment and a significantly reduced quality of life [22]. High-risk groups include women and patients with severe motor and cognitive deficits [8,14,15,27]. Approximately one in three stroke survivors develops depressive symptoms within three months to two years after the event [27], and PSD is considered a potential predictor of mortality.

Despite the availability of validated depression assessment tools, early detection of PSD remains challenging [20]. This is largely due to symptom overlap with post-stroke cognitive impairment and the absence of epidemiological data specific to Kazakhstan.

**Aim:** to analyze current literature to assess the impact of post-stroke depression on the rehabilitation process, adherence to secondary prevention, and outcomes of ischemic stroke, as well as to determine the feasibility of integrating early screening instruments into the practice of primary health care (PHC).

### Materials and methods

**Study design:** literature review.

A comprehensive analysis of scientific publications in Russian and English was conducted using international databases, including PubMed, Scopus, eLibrary, and the Cochrane Library, as well as official resources of the World Health Organization (WHO). Particular emphasis was placed on studies published within the past ten years that provide contemporary evidence on post-stroke depression (PSD) and its impact on the outcomes of ischemic stroke.

**Inclusion criteria:** peer-reviewed publications presenting data on the epidemiology, pathophysiology, and clinical impact of PSD on rehabilitation processes and outcomes of ischemic stroke; studies describing validated tools for early PSD detection and therapeutic interventions applicable in primary health care (PHC). The analysis included major systematic reviews, clinical trials, and recommendations issued by leading international organizations.

**Exclusion criteria:** studies lacking clinical data, publications with low levels of evidence, duplicates, articles

without full-text access, and research focused on hemorrhagic stroke.

### Search strategy:

The following keyword combinations were used:

- ("Post-stroke depression" OR "PSD" OR "Post-stroke rehabilitation" OR "Secondary prevention") AND

- ("Stroke" OR "Ischemic stroke") AND

- ("Diagnostic scales" OR "Depression scales")

Boolean operators **AND**, **OR**, and **NOT** were applied.

**Additional filters** (English and Russian):

- **Type of publication:** review, meta-analysis, clinical trial, guideline, systematic review

- **Topic areas:** neurology, psychiatry, rehabilitation, public health

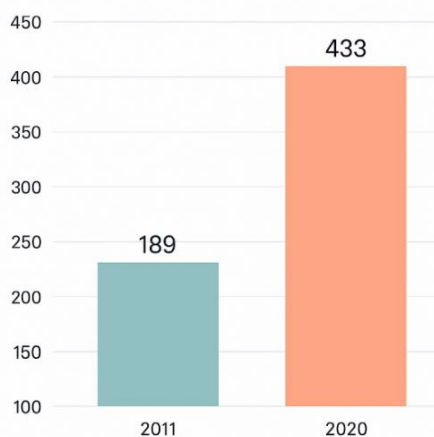
- **Age group:** adult, older adult

### Results of the review

Despite the high prevalence and clinical significance of this complication, no official statistical data on post-stroke depression (PSD) are available in the Republic of Kazakhstan. The average estimated frequency of PSD in Kazakhstan is presumed to be comparable to global rates; however, objective assessment of its true prevalence requires data from national stroke registries.

More than 49,000 stroke cases are recorded annually in the country, of which 80% are ischemic [2]. According to epidemiological data reported by *Kairatova G.K. et al.* (2022), the incidence of stroke in Kazakhstan has been steadily increasing, rising more than twofold - from 189 per 100,000 population in 2011 to 433 per 100,000 in 2020 [1]. Mortality has reached 180 deaths per 100,000 population per year.

**Incidence of Stroke in Kazakhstan  
(per 100.000 population)**



**Figure 1. Stroke incidence in the Republic of Kazakhstan**

Approximately 29% of all stroke patients are younger than 60 years, representing individuals of active working age. High disability rates (unofficially estimated at 49–80%) in Kazakhstan create a substantial socio-economic burden for the state [1].

Given these data, assessing the true prevalence of PSD remains challenging. The absence of official statistics on the occurrence and impact of PSD on post-stroke recovery in the Republic of Kazakhstan underscores the need for a systematic analysis of contemporary literature and the

implementation of validated early screening tools in the acute and subacute post-stroke period.

### **Pathophysiological mechanisms of post-stroke depression**

The pathophysiological mechanisms of post-stroke depression (PSD) are multifactorial and arise from a combination of ischemic brain injury, psychological stress, and disrupted neurobiological processes [45].

#### **1. Neuroanatomical factors: lesion location and infarct volume**

Earlier hypotheses suggested an association between PSD and lesions of the left frontal lobe or basal ganglia, regions responsible for emotional regulation in healthy individuals [16,46]. However, studies by Caeiro [17] and Aben [11] did not confirm a consistent relationship. At the same time, several investigations have demonstrated that larger infarct volume is associated with an increased risk of PSD development [49,51].

#### **2. Monoaminergic neurotransmitter imbalance**

Reduced levels of monoaminergic neurotransmitters - serotonin (5-HT), dopamine (DA), and norepinephrine (NE) [43] - result from ischemic injury to ascending brainstem pathways [39,40], forming a core neurobiological basis of PSD.

Experimental PSD models frequently employ middle cerebral artery occlusion (MCAO) [30,41]. MCAO-induced activation of calcium-activated potassium channels (SK channels) in the ventral tegmental area suppresses dopaminergic neuron activity and promotes depression-like behavior, whereas pharmacological inhibition of these channels produces an antipsychotic effect [50].

#### **3. Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis**

Excessive production of pro-inflammatory cytokines following stroke leads to hyperactivation of the HPA axis and persistent hypercortisolemia [52]. This contributes to impaired neuroplasticity and reduced neurogenesis in emotion-regulating structures, including the hippocampus and prefrontal cortex [37]. Chronic hypercortisolemia is considered a significant risk factor for PSD, particularly when sustained for several months [23].

Prolonged HPA axis activation also disrupts the Keap1-Nrf2 signaling pathway, a key component of the antioxidant defense system, further contributing to PSD development [38].

#### **4. Glutamate-mediated neurotoxicity**

Acute elevations in extracellular glutamate - the primary excitatory neurotransmitter [44] - trigger excitotoxic cascades involving cellular swelling, activation of calcium-dependent enzymes, apoptosis, and neuronal death [56]. Elevated serum glutamate concentrations in patients with PSD correlate with greater severity of depressive symptoms [19].

#### **5. Neuroinflammation**

Neuroinflammation is a central mechanism of neuronal injury in the post-stroke period [18]. It triggers the release of pro-inflammatory mediators - chemokines, cytokines (IL-6, TNF- $\alpha$ ), adhesion molecules, and reactive oxygen species (ROS) - which, despite their initial protective role in ischemic tolerance, ultimately worsen tissue damage [13].

Overactivation of perivascular astrocytes and excessive secretion of cytokines such as IL-6, TNF- $\alpha$ , and MMP-9 disrupt the integrity of the blood–brain barrier [21] and

exacerbate necrotic processes. Collectively, these mechanisms contribute to the clinical expression and severity of PSD [28].

### **Mechanisms by which PSD influences adherence to secondary prevention**

The key psychopathological manifestations of post-stroke depression (PSD) that affect patient adherence to secondary prevention measures are outlined below.

#### **1) Anhedonia and apathy (impaired motivation and goal-directed behavior)**

PSD is frequently accompanied by anhedonia, apathy, sleep disturbances, and increased fatigability. These symptoms reduce motivation and impair the performance of essential self-management behaviors such as medication intake, participation in physical training, and regular self-monitoring [35]. Notably, antidepressants are not always effective in treating apathy [25].

#### **2) Cognitive and communicative deficits /depressive–executive dysfunction syndrome**

PSD is associated with executive dysfunction, aphasia, and low health literacy, which impair planning and complicate adherence to complex treatment regimens. These difficulties often lead to the need for external assistance [34]. Clear explanations and educational materials have been shown to improve adherence to preventive measures.

#### **3) Low patient activation and reduced self-efficacy**

PSD diminishes patients' confidence in their ability to manage their condition. Lower engagement in rehabilitation and preventive strategies, as well as negative attitudes toward medical treatment, are independently associated with poorer medication adherence among stroke survivors [48]. Additional barriers - including anxiety, inconvenience of treatment regimens, and concerns about medication side effects - further reduce adherence, often to a greater extent than sociodemographic factors [34].

#### **4) Social isolation and lack of support**

Depression correlates with reduced social activity. Research demonstrates a strong association between depression, social support, and the effectiveness of secondary prevention, as well as the benefits of caregiver and family involvement [55]. Living alone and polypharmacy are also linked to lower adherence, amplifying the negative impact of PSD [47].

### **Impact of post-stroke depression on adherence to secondary prevention of ischemic stroke**

Given that PSD occurs in approximately one-third of stroke survivors, the AHA/ASA recommends routine depression screening for all patients following a stroke. A study conducted among older adults ( $n = 102$ ) demonstrated that higher PHQ-9 scores - indicating more severe depressive symptoms - were associated with lower medication adherence at the 6-month follow-up [47].

Furthermore, a 2019 meta-analysis conducted by a group of Chinese researchers showed that the presence of PSD was associated with an increased risk of recurrent stroke (relative risk  $\approx 1.48$ ) [54].

### **PSD and medication adherence (adherence to anticoagulants, antiplatelet agents, statins, and antihypertensive therapy)**

A multicenter cohort study conducted in France demonstrated that PSD identified using the HADS scale (8–



10 points) was associated with incomplete adherence to the full range of secondary prevention measures: OR 1.90; 95% CI 1.05–3.44 [29]. The lowest adherence was observed for statin therapy, which led to poorer vascular outcomes [12].

#### **Influence of PSD on lifestyle and rehabilitation**

Large-scale international studies indicate that up to 90% of strokes are attributable to ten major risk factors, either individually or in combination [42].

Key modifiable risk factors include healthy diet, regular physical activity, maintaining a normal BMI, moderate alcohol consumption, and smoking cessation.

PSD significantly reduces adherence to secondary prevention and impairs effective management of modifiable risk factors. The presence of two or more uncontrolled risk factors substantially worsens prognosis [33].

#### **Diagnosis of post-stroke depression at the primary healthcare level**

Early detection of PSD within primary healthcare settings is of critical importance. Short, validated, and easy-to-administer screening tools are particularly relevant, as they allow timely identification of at-risk patients without requiring mandatory involvement of a psychiatrist.

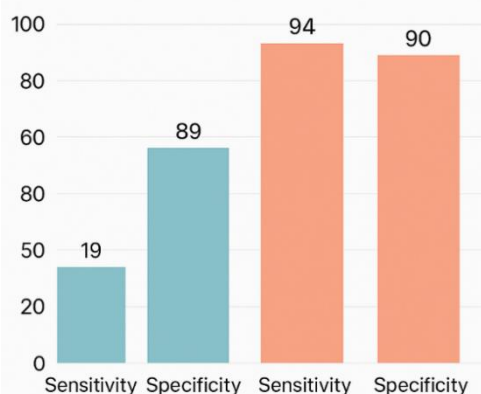
Table 1.

**Diagnostic tools for post-stroke depression.**

Scale	Advantages	Threshold	Sensitivity / Specificity
PHQ-9	Brief, easy to administer (including self-administration), and readily integrable into primary healthcare workflows	≥ 10 (AUC=0,96)	91%/89% (Williams L.S., 2005)
BDI-FS	Complements the PHQ-9 and excludes stroke-related somatic symptoms	≥ 10 (AUC=0,93)	94%/90% (Furlanetto LM, 2005)

These scales are recommended for screening and dynamic monitoring of groups at high risk for developing post-stroke depression.

**Comparison of Diagnostic Characteristics of the PHQ-9 and BDI-FS Scales**



**Figure 2. Comparison of Diagnostic Scales**

#### **Therapeutic interventions for post-stroke depression at the primary healthcare level**

The primary goals of PSD therapy include reducing depressive symptoms, strengthening self-management and

motivation, and improving adherence to secondary prevention of ischemic stroke.

#### **Management strategy for patients with PSD in primary healthcare (PHC):**

1. Screening for PSD in PHC settings is recommended for all stroke survivors without pronounced cognitive impairment [32].

2. During the initial visit, screening is performed by neurologists and general practitioners using the PHQ-9 and BDI-FS scales. Screening may also be conducted by appropriately trained mid-level healthcare personnel.

3. Repeat assessment of PSD and evaluation of medication adherence (using the MMAS scale) should be performed at 4–6 weeks and subsequently at 3, 6, and 12 months of follow-up [32].

4. All post-stroke patients and their family members must be informed about the risk of developing PSD, which includes patient education during consultations and the provision of informational materials.

5. Prescribing antidepressants is permitted for neurologists and general practitioners in accordance with the Ministry of Health Order № ҚР ДСМ-224/2020

6. “On the Approval of the Standard for Medical and Social Care in the Area of Mental Health for the Population of the Republic of Kazakhstan” [7].

Table 2.

**Management strategy for patients with post-stroke depression at the primary healthcare level.**

Scale	Score range	Degree of depression	Management strategy	Level of evidence
PHQ-9	5-9	Mild	Psychological counseling, cognitive-behavioral therapy (CBT) monotherapy	B
BDI FS	10-15			
PHQ-9	10-14	Moderate	CBT, with antidepressants if required	B
BDI FS	16-19			
PHQ-9	15-19	Severe	Psychiatric consultation, mandatory initiation of antidepressant therapy, and hospitalization in a specialized unit if necessary	A
BDI FS	20-29			
PHQ-9	20-27	Very severe (high suicide risk)	Urgent psychiatric consultation and hospitalization in a psychiatric/neurological facility with selection of combined therapy: pharmacotherapy + psychotherapy.	A
BDI FS	30-39			

Note: Levels of evidence (A, B, C) correspond to the Canadian Stroke Recommendations system (2021) [10].

**Key Therapeutic approaches to post-stroke depression in accordance with the AHA/ASA Clinical Guidelines (2017) [8] and Cochrane systematic reviews (2021) [9], include the following components:**

1. The primary therapeutic objective is to achieve a clinically significant reduction in depressive symptoms, defined as a decrease of more than 5–6 points on the PHQ-9 and BDI-FS scales within 6–8 weeks of treatment.

2. Completion of all stages of post-stroke rehabilitation, including timely initiation of PSD treatment, contributes to a reduction in depressive severity and accelerates functional recovery.

3. High adherence to rehabilitation measures and secondary prevention strategies is essential for effective PSD management and is associated with improved cognitive and emotional outcomes.

4. Developing self-management skills plays a critical role. These include maintaining a blood pressure diary, adhering to dietary recommendations, and optimizing physical activity. Such measures help achieve target blood pressure levels and lipid profiles (LDL < 1.7 mmol/L), thereby reducing the risk of recurrent ischemic events.

5. Cognitive-behavioral therapy (CBT) is effective both as a treatment modality and as a preventive strategy for PSD in high-risk individuals (those with a history of depressive episodes, significant cognitive or motor impairments, or social isolation).

6. Antidepressants may be prescribed, considering safety and tolerability, for patients with moderate PSD who do not respond sufficiently to CBT. First-line agents include selective serotonin reuptake inhibitors (SSRIs) [8,9].

7. Severe forms of PSD require multimodal therapeutic approaches, incorporating long-term pharmacotherapy with antidepressants, CBT, and additional non-pharmacological interventions (motivational programs, physical activity, and comprehensive psychoeducation).

8. Gradual tapering of antidepressants over 1–2 months is recommended to prevent withdrawal syndrome and minimize the risk of relapse of depressive symptoms.

Thus, early diagnosis and timely initiation of treatment for post-stroke depression lead to improved functional and clinical outcomes following ischemic stroke.

### Discussion

The prevalence of post-stroke depression (PSD) ranges from 18% to 33%, yet its true occurrence remains difficult to estimate due to insufficient diagnostic practices and low clinical awareness among healthcare providers. These factors contribute to delays in initiating treatment, which negatively affects stroke outcomes.

Established risk factors include female sex, a history of depression, extensive or recurrent strokes, a stroke occurring within the past year, severe disability, and limited social support. These factors should be considered during early risk stratification at the level of primary healthcare (PHC).

The pathophysiological mechanisms of PSD are multifactorial, reflecting the variability of clinical presentations and the heterogeneity of treatment response.

Evidence regarding the effectiveness of preventive interventions remains limited and inconsistent, preventing the formulation of a universal prevention strategy for all

stroke survivors. Further research is needed to establish standardized approaches to PSD prevention.

According to current AHA/ASA guidelines and Cochrane reviews, optimal management of PSD includes cognitive-behavioral therapy (CBT) and psychological counseling, either as standalone interventions in mild cases or as part of combined therapy. Selective serotonin reuptake inhibitors (SSRIs) are considered first-line pharmacological agents for moderate and severe depression.

Timely identification of PSD at the primary care level using validated screening tools is of particular importance. For Kazakhstan, key priorities include implementing standardized PSD management protocols within the PHC system, training general practitioners and mid-level healthcare personnel in the use of screening instruments, and integrating depression assessment into the clinical pathway for post-stroke patients.

### Study limitations

1. Lack of unified methodological approaches for diagnosing post-stroke depression, including variability in diagnostic criteria, assessment scales, and timing of evaluation.

2. The chronological interval following the stroke is a key factor influencing the clinical course of PSD; its variability complicates comparison across studies.

3. Insufficient research on the diagnosis and management of PSD in outpatient settings (PHC) and in the post-stroke population of Kazakhstan.

4. Limited number of studies evaluating the effectiveness of comprehensive intervention programs (pharmacotherapy + psychotherapy + motivational interventions) within primary healthcare.

5. Lack of national epidemiological data on PSD and its impact on adherence to secondary prevention in the Republic of Kazakhstan.

### Conclusions

1. Post-stroke depression is a common complication, occurring in approximately one-third of stroke survivors and significantly impairing functional recovery.

2. PSD is an independent predictor of adverse outcomes, including reduced adherence to secondary prevention, worsening cognitive status, and increased risk of mortality.

3. Early diagnosis and timely treatment of PSD at the PHC level are crucial for successful rehabilitation, as they enhance patient engagement in secondary prevention programs and improve overall prognosis.

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