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SPECIAL ARTICLE

Clinical practice guidelines for post-stroke depression in China

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Post-stroke depression (PSD) is a very common complication that leads to increased physical disability, poor functional outcome, and higher mortality. Therefore, early detection and treatment are very important. Since there are currently no specific guidelines for this disorder in China, the purpose of this study was to develop PSD guidelines and provide suggestions for clinicians and related workers.

Keywords: Post-stroke depression; diagnosis; treatment

Introduction

Post-stroke depression (PSD) is a common mood disorder characterized by a group of depressive symptoms in stroke survivors. PSD is similar to major depressive disorder (MDD), but differs in the frequency of certain symptoms.[1,2] For instance, PSD patients mostly present with mood fluctuations, retardation, irritability, or apathy, while anhedonia, pessimism, suicidal ideation, or attention deficits are more common in MDD. Importantly, it is difficult to determine whether retardation and apathy symptoms are due to depression or post-stroke neurological deficit. PSD diagnosis lacks specificity only in the symptoms common to MDD, such as self-guilt, suicidal ideation or behavior, weight loss, and early waking.[3] PSD reduces physical activity, quality of life[4] and substantially increases the risk of suicide.[5] Early antidepressant treatment for PSD appears to enhance both physical and cognitive recovery from stroke and might increase post-stroke survival up to 10 years.[6] Thus, it is both challenging and important to recognize and distinguish the depressive symptoms specific to PSD.

A 2014 meta-analysis found that the incidence of PSD was nearly 31%,[7] while a narrative review reported that PSD prevalence ranged from 5 to 67% among all types of stroke patients.[8] According to a correlative study in China, PSD prevalence is 45.79%, including 34.21% early-onset PSD and 11.58% late-onset PSD (occurring at least two weeks after a stroke).[9] In most studies, early-onset PSD is defined as depression occurring less than two weeks after a stroke, while late-onset PSD occurs more than two weeks after a stroke.[9-11] This result is inconsistent with the 25-79% found in another study.[12] This variation is influenced by many factors, the first of which is that different evaluation instruments yield different results: it was found that the rate of PSD diagnosis is higher when using adapted rating scales (e.g., the Hamilton Depression Rating Scale [HDRS]) than when using mood disorder diagnostic criteria (Structured Clinical Interview for DSM-IV).[13,14] Second, the stroke patients in these studies were assessed at different stages.[15-17] Most patients develop PSD in the acute phase of stroke, and newly diagnosed PSD often decreases in the subsequent follow-up.[10] Third, variations in study populations and environment (e.g., hospital vs. community) also lead to varied results, seeing that PSD in patients from neurological departments and rehabilitation centers is more prevalent than in outpatients.[18] Finally, stroke patients often present with aphasia, agnosia, and cognition impairments, which add to the complexity of recognizing and diagnosing PSD.

PSD should consist of post-stroke depressive symptoms (PSDS) and post-stroke depressive disorder (PSDD). PSDS develop in parallel with stroke, possibly due to direct brain injury or acute psychosocial response to stroke, while PSDD is an endogenous depression induced by stroke or stroke sequelae, usually occurring six months post-stroke.

PSDS have a relatively short duration (approximately 12 weeks), while PSDD lasts an average of 39 weeks.[19] Over 50% of new-onset PSD patients during the first six months after stroke can recover in an additional three to six months, but a considerable proportion of the patients will relapse within one year.[16] PSD affects activities of

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daily living and reduces the cognitive recovery of stroke survivors, and patients with cognitive impairment show worse neurological function.[20-22] A 2016 Chinese survey found that depression is associated with a five-year disability rate after stroke.[23] The incidence of suicidal ideation in PSD patients is reported to be 6.6 and 11.3% in the acute and late stages, respectively.[24] Furthermore, a recent nationwide study in China found that PSD is a possible risk factor for stroke-induced death.[25]

The Global Disease Burden Study reported that in 2013 there were almost 103 million new-onset strokes (67% ischemic), 6.5 million deaths from stroke (51% from ischemic stroke), and 11.3 million disability-adjusted life years (DALYs) due to stroke (58% to ischemic stroke).[26] Stroke caused 1.7 million deaths and 2.1 million DALYs in China in 2010.[27] The direct economic burden from stroke was almost 19.9 billion yuan, accounting for 3.79% of national medication expenses and 3.02% of national public health costs in 2005.[28] Although there have as yet been no assessments of the disease burden of PSD, depression does increase the post-stroke caring burden.[29] Considering the high prevalence, mortality, and significant influence of PSD on neurological recovery, it is apparent that depression contributes substantially to the disease burden after stroke.

Although PSD is a serious problem for both stroke survivors and medical workers, a standardized guideline for the clinical management of PSD has not yet been developed in China. PSD misdiagnosis and delayed or no treatment are frequent in clinical settings nationwide. Even if current antidepressant treatment can improve depressive symptoms, neither the optimal drug nor the optimal length of treatment have been identified.[30] Thus, a comprehensive introduction to PSD is urgently needed. This study aims to develop guidelines on PSD to provide suggestions for clinicians and related workers.

Risk factors for PSD

Previous studies have found that PSD is related to many factors, such as age,[31] sex,[32] ethnicity, cultural background,[33] and education level,[34] but the results are not always consistent.

The social-psychological factors of PSD mainly include neurological impairment after stroke, work disability, and change of status in society and the family.[35] It is reported that negative life events,[13] a lack of social support,[36] low family income,[37] premorbid neuroticism,[38] as well as type A behavior[39] are associated with an increased risk of PSD.

Patients with PSD are more likely to have a family history of depression.[17] Molecular genetic studies have demonstrated that variation in several genes is associated with PSD, including those for the serotonin transporter (SLC6A4),[40] 5-HT 1A[41] and 2A[42] receptors (HTR1A and HTR2A), brain-derived neurotrophic factor (BDNF),[43] N5, N10-methylenetetrahydrofolate reductase (MTHFR),[44] catechol-O-methyltransferase (COMT),[45] interleukin (IL)-10, IL-4,[46] and cAMP response element binding protein (CREB),[47] among which 5-HT associated genes are the most preferred candidates in genetic research. Moreover, gene-gene and gene-environment interactions are also involved in the occurrence of PSD.[43] In addition, microRNA is considered to play an important role in the development of PSD and antidepressant therapy.[48]

Researchers have been very interested in changes of neurotransmitter function in the pathophysiology of PSD. A significant decrease has been demonstrated in norepinephrine, 5-HT, and its metabolite 5-hydroxyindol acetic acid (5-HIAA) in the cerebrospinal fluid of PSD patients in the acute phase of stroke.[49,50] Abnormal glutamate metabolism in the prefrontal lobe can also be found by single voxel proton magnetic resonance spectroscopy.[51]

Previous studies indicate that inflammation may participate in the incidence of PSD. Inflammatory cytokines, such as IL-1, IL-6, IL-18, and tumor necrosis factor (TNF)-alpha have been found to be significantly increased after stroke,[52] and the serum level of C-reactive protein is reported to be an important predictor of PSD occurrence and severity.[53]

Neuroendocrine research suggests that there may be impaired function of the hypothalamus-pituitary-adrenal axis, the hypothalamus-pituitary-thyroid axis, and the hypothalamus-pituitary-gonad axis in PSD patients.[54,55] In addition, it has been demonstrated that PSD is associated with low BDNF[56] and ferritin,[57] as well as with elevated neopterin.[58]

Neuroanatomical abnormality may also play an important role. PSD is probably associated with damage of the frontal/temporal lobe-basal ganglia-ventral brainstem circuitry and deficits in corresponding chemical neurotransmission.[59] Stroke lesion location and white matter hyperintensities (WMHs) have been extensively investigated as risk factors for PSD. There is still a matter of debate on the association between the lesion location and the occurrence of depression due to contradictory findings. This might be explained by the fact that the clinical characteristics of PSD differ depending on its anatomical correlates. For instance, affective depression is associated with left frontal stroke, while apathetic depression is related to damage of the bilateral basal ganglia.[60] It has also been found that there is an association between the frequency and severity of PSD and the proximity of the lesion to the frontal pole, i.e., the closer the lesion is to the frontal pole, the more severe the depression is.[61] Subgroup analyses according to different stroke stages highlight that the risk of depression is associated with left hemisphere stroke in the acute phase (<1 month), but relates to right hemisphere stroke in the subacute stage (1-6 months).[62] Furthermore, WMHs have a relatively important influence on PSD in small infarctions, while the infarction itself plays a major part in PSD occurrence in patients with a large lesion.[63]

Clinical assessment of PSD

Early recognition, prevention, and treatment of PSD are vital to the recovery and prognosis of stroke survivors. Since there are many risk factors for PSD, an integrated assessment should cover general information, functional level after stroke, and depressive symptoms.

General information

Present history

It is very important to screen for depressive symptoms in the acute phase of stroke and record the time of onset, characteristics, severity, and accompanying psychological and somatic symptoms of depression. Considering the close relationship between PSD and stroke, an overall evaluation of the stroke is also essential, including its type, location, size, severity, and the functional state of the patient.

Prior history

Previous attacks of stroke, depression, and other psychotic disease are all significant determinants of PSD. The existence and treatment outcome of these disorders should be assessed. In addition, the assessment of vascular risk factors is also necessary, since hypertension and angina pectoris are independent predictors of PSD.[64,65]

Personal and family history

The factors associated with occurrence of PSD should be assessed, including age, gender, adverse life events, family income, living alone, social support, and personality traits.[66-68] Family history of depression should also be assessed, since a family history of depression is common in PSD patients.[17]

Functional level after stroke

Motor disability is one of the most severe outcomes of stroke. The National Institutes of Health Stroke Scale (NIHSS),[69] modified Rankin Scale (mRS),[70] and Barthel Index (BI) are common assessment instruments for neurological deficit.[71]

Depressive symptoms

The evaluation tools used in clinics are self-rating scales and observer-rating scales. Self-rating scales include the Self-Rating Depression Scale (SDS),[72] Beck Depression Inventory (BDI),[73] Patient Health Questionnaire-9 (PHQ-9),[74] Hospital Anxiety and Depression Scale (HADS),[75] and Center for Epidemiologic Studies Depression Scale (CES-D),[76] while the HDRS[77] and Montgomery-Asbery Depression Rating Scale (MADRS) are observer-rating scales.[78]

However, considering the different symptomatic distributions in MDD and PSD, the above-mentioned scales may have low specificity for PSD patients, given that they are all designed for MDD patients. Based on previous scales, Yue et al.[79] developed a new PSD scale (PSD-S), a self-evaluation scale with excellent reliability and validity (available in the original article as online supplementary material[79]). It can be used as an early screening tool for PSD in which scores of 6/24 and 15/24 are cut-points for mild and moderate/severe PSD respectively. The PHQ-9, which consists of nine symptomatic items derived from the DSM-5, is a screening instrument for depression, evaluating the frequency of depressive symptoms in the previous two weeks. Williams et al.[80] explored the performance of the PHQ-9 as a screening and diagnostic tool for depression in stroke survivors, finding 91% sensitivity and 89% specificity with a cutoff value of 10. The PHQ-9 is convenient, has good reliability and validity and is recommended for early screening of MDD and PSD.

Diagnosis and differential diagnosis of PSD

There are no accurate diagnostic criteria for PSD in the current generalized diagnosis and classification systems of mental disorders. Some studies have adopted the DSM-5's diagnostic criteria for MDD or merely relied on depression assessment scales to diagnose PSD. The disorder was merely defined as a "depressive disorder due to another medical condition" and classified into three subtypes – with depressive features (F06.31), with a major depressive-like episode (F06.32), or with mixed features (F06.34) – in the DSM-5, which was published in May 2013 (293.83).[81]

Generalized PSD should include PSDS and PSDD according to the diagnostic criteria. PSDS can be considered as a transition between normality and PSDD or the earlier stages of PSD, which can either progress to PSDD, maintain its original condition or recover spontaneously (Figure 1). Yue et al.[79] further proposed a set of diagnostic criteria for PSDS (Table 1), which can be used for diagnosis one week post-stroke. Meanwhile, PSDD is considered post-stroke MDD, and should first fulfill the PSDS criteria and then the DSM-5's MDD criteria.[82] To confirm this diagnosis, clinicians should discriminate PSD from post-stroke apathy, post-stroke anxiety (PSA), post-stroke fatigue (PSF) and post-stroke psychotic disorder (PSPD).

Post-stroke apathy

Post-stroke apathy is so similar to PSD that it is difficult to distinguish them. The differentiation could depend on

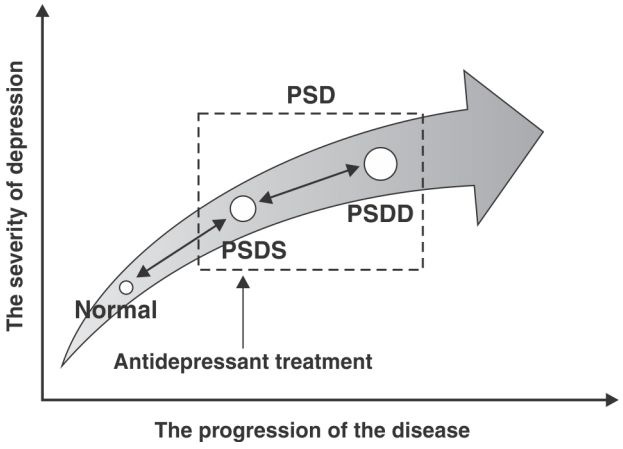

Figure 1 Diagram presenting the progression of PSD (cited from Yue et al.[82]). PSD = post-stroke depression; PSDD = post-stroke depressive disorder; PSDS = post-stroke depressive symptoms.

Table 1 Diagnostic criteria for post-stroke depressive symptoms

A. Three (or more) of the following symptoms have been present during the same one-week period and represent a change from previous functioning.
1. Uncommunicative (e.g., avoids speaking) most of the day, every day, or most of the week.
2. Fatigue or loss of energy every day, or most of the week.
3. Depressed mood persisting most of the day, every day, or most of the week, as indicated by either self-report or observation by others (e.g., feels sad, cries easily).
4. Insomnia, waking up early, or hypersomnia every day or most of the week.
5. Feelings of helplessness, worthlessness most of the day, every day, or most of the week.
6. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.
7. Feeling of hopelessness or despair (especially related to the stroke) most of the day, every day, or most of the week.
8. Unusually irritable every day or most of the week.
B. The symptoms cause clinically significant distress or impairment in social interaction, occupation, or other important areas of functioning.
C. The occurrence, development, and duration of these symptoms are closely related to cerebrovascular disease.
D. The occurrence of the major depressive episode could not be better explained by adjustment disorder with depressed mood, schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum or other psychotic disorders.
E. No manic or hypomanic episode reported.

varying psychiatric symptoms, emotional properties, or facial expressions. In psychiatric symptomatology, apathy is related to disinhibition, declining cognitive function, and aberrant motor behaviors, while depression is associated with anxiety, agitation, and irritability.[83] Regarding emotional properties, apathetic patients are indifferent, have a neutral mood, and are usually without suicidal ideation,[84] but depressed patients show a clearly negative mood. With respect to facial expression, apathetic patients often present a flat affect and lack of eye contact, while most depressed individuals have a typical expression of sadness, with emotion in their eyes.

Post-stroke anxiety (PSA)

PSA is usually seen in the chronic phase of stroke, with the incidence rising over time,[85] while most PSD occurs in the acute stage. Although the occurrence of PSD is loosely linked to prior history of depression, it is rather strongly influenced by the stroke itself; however, PSA is closely associated with prior anxiety.[86] PSD patients mostly show a constantly depressed mood and loss of interest, accompanied by somatic or mental anxieties such as worrying, tension, and palpitation, which are all attributable to depressive mood. In comparison, PSA patients present fear, tension, worry, irritability, or restlessness.

Post-stroke fatigue (PSF)

PSF is a subjective feeling of physical or mental weariness and lack of energy independent of exercise or prior activity, with abnormal, transitional, and chronic characteristics that lead to difficulty maintaining even routine activities.[87] It is necessary to differentiate PSF from PSD when depressed mood presents in fatigue and when symptoms such as fatigue and loss of energy accompany PSD.

Post-stroke psychotic disorder (PSPD)

PSPD refers to many types of psychiatric syndromes in the acute, rehabilitation, and sequelae stages of stroke. It is reportedly a complex of many symptoms, including hallucination, delusion, and delirium, which hinders functional outcome and quality of life.[88] Although usually with a slow and fluctuating course that may rapidly worsen when aggravated by a stroke or improve due to compensating collateral circulation, PSPD will generally develop into dementia despite its various clinical presentations.

PSD treatment

Drug therapy

Timely and reasonable antidepressant treatment is not only helpful for relieving depression, but also benefits neurological outcome and long-term prognosis. PSD treatment depends on a good doctor-patient relationship, establishing a therapeutic alliance consisting of the patients and their family members, who are encouraged to cooperate with clinicians for the patient's recovery. Antidepressant treatment should begin as soon as stroke survivors have been diagnosed with PSDS, including medication, physical therapy, psychological therapy, etc.

Antidepressants are the preferred treatment of PSD.[89] The choice of antidepressant depends on the patient's depressive symptoms and the side effects, especially for the elderly. Meanwhile, doctors should be aware of the interaction of the chosen drug with other medications and its influence on the disease. Clinicians should closely observe and assess the response to therapy, constantly focus on the probable side effects and thoroughly communicate with patients to maintain their compliance, since antidepressants are not as well tolerated due to certain adverse events.[89] Furthermore, there is also the risk of a manic attack during PSD treatment. If this occurs, the antidepressant dose should be reduced or stopped immediately and replaced with a mood stabilizer.

Regarding the efficacy of antidepressants, a multicenter randomized controlled trial (RCT) with 788 PSD patients demonstrated that the effectiveness of an

eight-week treatment with paroxetine is 93.1%.[90] A small number of clinical studies have had success with citalopram,[91] escitalopram,[92] sertraline,[93] fluvoxamine,[94] and mirtazapine.[95] A meta-analysis of 11 RCTs indicated that fluoxetine has superior effects as a PSD treatment,[96] whereas another meta-analysis of 32 RCTs reported that venlafaxine is more effective than selective serotonin reuptake inhibitors (SSRIs).[97] A meta-analysis on the effectiveness of citalopram shows there may be no significant difference in the efficacy of different SSRIs.[98] However, the validity of evidence in many of these studies is weak due to a small sample size or a non-RCT design.

Traditional Chinese medicine has also been used in PSD treatment. In clinical studies, traditional Chinese medicine has been used alone for mild depression or combined with antidepressants for moderate and severe depression. The results showed that the Wuling capsule and the Shuganjieyu capsule are effective for PSD patients.[99,100]

However, it is still unclear when to start antidepressant treatment and how long it should continue. A longitudinal follow-up study by Fruehwald et al.[101] found that three-months of fluoxetine treatment in PSD patients can significantly increase emotional and functional recovery at 18 months post-stroke, although no significant effects were observed in the early stages of drug administration. A single-blind RCT in a Chinese population also found that the effects of a three-month citalopram treatment program were more efficient six months post-stroke.[102] These studies indicate that the drugs' effects might be masked by other clinical factors, and further studies should be conducted to determine whether PSD treatment should follow the acute, consolidation, or maintenance phases, as with MDD. In the 2015 update of the Canadian Stroke Best Practice Recommendations, it was advised that drug therapy should continue for at least period is 6 to 12 months if the chosen drugs are effective.[103] With respect to PSDS, the maintenance stage of treatment could be reduced, continuing 2 to 3 months after the depressive symptoms disappear.

Physical therapy

Repetitive transcranial magnetic stimulation (rTMS) might be an effective and safe treatment method for refractory PSD patients.[104] However, the method presents certain complications, such as headaches, gastrointestinal reaction, dry mouth, tinnitus, and even seizures.[105] It is appropriate to carry out rTMS six months post-stroke. More clinical evidence is needed to confirm the best frequency and stimulation intensity for PSD patients. Electroconvulsive therapy (ECT) is an alternative for nonacute PSD patients with severe suicidal ideation, refractoriness, or intolerance to drugs. However, ECT often induces or aggravates cognitive impairment and ECT-associated complications, thus it is not preferred for PSD treatment.[106] It has been indicated that early treatment is favorable for the long-term prognosis of PSD patients. However, attention should also be paid to adverse events in rTMS or ECT, since many stroke survivors, especially those with cerebral stent or hemorrhage, cannot tolerate rTMS or ECT.

Psychotherapy

Supportive psychotherapy and cognitive behavior therapy (CBT) have been found effective in PSD treatment.[107,108] Social support interventions are very helpful for returning patients to society and reestablishing the patients' relationships with others. PSD can be avoided or decreased in severity with social support from family, friends, and colleagues, who are advised to visit more frequently, providing more company and encouraging the patient to accept the therapy and rehabilitation exercises positively. CBT aims to change the cognitive activity of stroke patients, pointing out inappropriate modes of thought and their counterproductive ideations, inspiring patients to adopt reasonable thoughts and abandon self-destructive ideas and emotions, and then re-constructing neuronal circuits to correct cognition and behavior. Balancing psychotherapy (BPT) is a treatment approach based on eastern philosophical systems that combines different psychological schools. Initially, psychosomatic balancing theory and methods are used to clear away the causes of PSD and mental blockage, which imbalance internal homeostasis. The patients are then provided with clues toward an overall and intrinsic knowledge of their problems. Afterwards, patients learn to observe themselves resolutely and reconstruct their cognition, obtaining inner balance by comparing their experiences with similar events in the lives of others. Psychotherapy is preferable as a supplementary tool in the treatment of mild and moderate depression.

Other therapy

Music therapy, acupuncture therapy,[109] Taijiquan, and hyperbaric oxygen therapy[110] have all been tried in PSD treatment, but, unfortunately, they lack well-designed RCTs to support their efficacy at present.

Preventive treatment of PSD

It has been found that early prevention can reduce the occurrence of PSD and induce effective recovery of neurological function.[111] Both antidepressants and psychotherapy have been shown to improve activities of daily living and cognitive function, as well as to decrease the mortality of stroke patients.[112]

Preventive application of antidepressants, especially SSRIs, can significantly reduce the incidence of PSD and improve the prognosis of stroke patients.[113]

Traditional Chinese medicine, such as the Wuling capsule, can also have a preventive effect, reducing incidence rates, delaying occurrence, and alleviating PSD symptoms.[114]

A motor relearning program (MRP) is a type of physical therapy with task-oriented strategies that rehabilitate motor function after damage to the central nervous system as a relearning or retraining process. Early combined MRP with regular stroke treatment can greatly decrease the prevalence of PSD.[115]

It has been demonstrated that services performed by community health professionals can effectively reduce the

severity of PSD and improve health-related quality of life. Intervention methods include telephone interviews, obtaining written information, home visits, contacting health professionals, and health service referrals.[116]

The occurrence of PSD has been associated with physiological, psychological, and social factors. CBT, problem-solving therapy, and home-based therapies have all been found efficient in stroke patients and beneficial to cognitive function and the quality of life of PSD patients.[117]

Overall, early prophylaxis is important for decreasing the incidence of PSD and helping regain function. However, there is still controversy about the strategies and timing of the prophylaxis. It has been suggested that early nondrug preventive methods, including lifestyle education, psychological counseling, and intervention should be used.[116,117]

PSD nursing

Hospital nursing staff and family caregivers play an important role in the recovery of PSD patients, who are in need of specific care measures.[118] Caregiver understanding of the disorder can have a powerful influence on its development and prognosis,[119] and caregiver workload and mental health are also associated with PSD patient outcomes.[120,121] Therefore, nursing care and psychological intervention are very important for PSD patients in both the acute and recovery phases.

Appropriate nursing care should be based on comprehensive nursing assessments, including physical conditions, mental status, and associated risk factors. Affective nursing support is necessary to help patients adapt to unfamiliar environments and reduce their emotional insecurity as quickly as possible. A family-based nursing strategy should be introduced in the early phase of PSD.

General nursing objectives include providing a comfortable environment, appropriate personal care (including nutrition supplements), and creating a relaxing sleeping environment to ensure adequate rest. Since post-stroke apathy and hypersomnia are among the leading obstacles to rehabilitation,[122] the nursing staff should help patients overcome these problems and guide them through the rehabilitation process as soon as possible. Effective bedside exercise can improve the emotional and physical functioning of stroke patients and thus elevate their quality of life.[123]

The role of psychological nursing includes promoting health, reducing stress and increasing coping skills.[124] PSD patients inevitably feel failure when faced with physical dysfunction.[125] Therefore, the nursing staff should respect and care for the patients, educate them about the disease, rebuild their self-confidence, and properly conduct the supportive psychotherapy.

A health education course can reduce the depressive symptoms of PSD patients.[126] The full course contains three phases: the acute phase (1-2 weeks post-stroke) involves supportive psychotherapy; the stable phase (3-4 weeks post-stroke) involves collective multi-media instruction; during the recovery phase (4-6 weeks post-stroke), caregivers should consolidate achievements and prevent relapse, encouraging the patients to return to society and their work and family responsibilities.[126]

Nurses should work out a personalized and complete nursing program depending on the specific needs and conditions of patients. Patients should receive health guidance prior to discharge and be interviewed by telephone one to two weeks after discharge to resolve any further questions. The health education course enables patients to voluntarily follow a healthy lifestyle, eliminate negative emotions, and increase their trust in the treatment program. As a result, they can fully engage themselves in treatment and rehabilitation and find relief from their PSD symptoms.

Diagnostic and treatment procedures of PSD

Figure 2 shows the diagnostic and treatment procedures used for PSD.

Prevention and management of PSD

Medical staff training

It is very important for sustainable PSD prevention that medical staff be actively trained under the auspices of the health department. Major training bases should be set up in psychiatric organizations nationwide. Systematic training in PSD diagnosis and treatment should be developed for psychiatrists, nonpsychiatrists, psychological consultants, psychological therapists, and medical nursing staff in general hospitals, special hospitals, health centers, and nursing homes of different scales, so that a basic mental service team composed of available human resources can be drawn from medical organizations on different levels for extensive and effective PSD prevention.

Education on mental health

Different types of media should be mobilized to promote public awareness of PSD and facilitate identification and treatment, maximizing the effects of such campaigns through conferences, speeches, family interviews, posters, popular science articles, and broadcasts.

Formation of an assessment and monitoring system

Medical workers should comprehensively evaluate the mental and physical conditions, dysfunction, and quality of life of their stroke patients to find high risk populations and carry out psychosocial interventions in time. Psychiatric units should be available to give advice on PSD diagnosis and treatment for inpatients in neurological and rehabilitation wards.

Building up favorable doctor-patient relationship and three-grade prevention

A good doctor-patient relationship based on appropriate communication will result in better patient compliance,

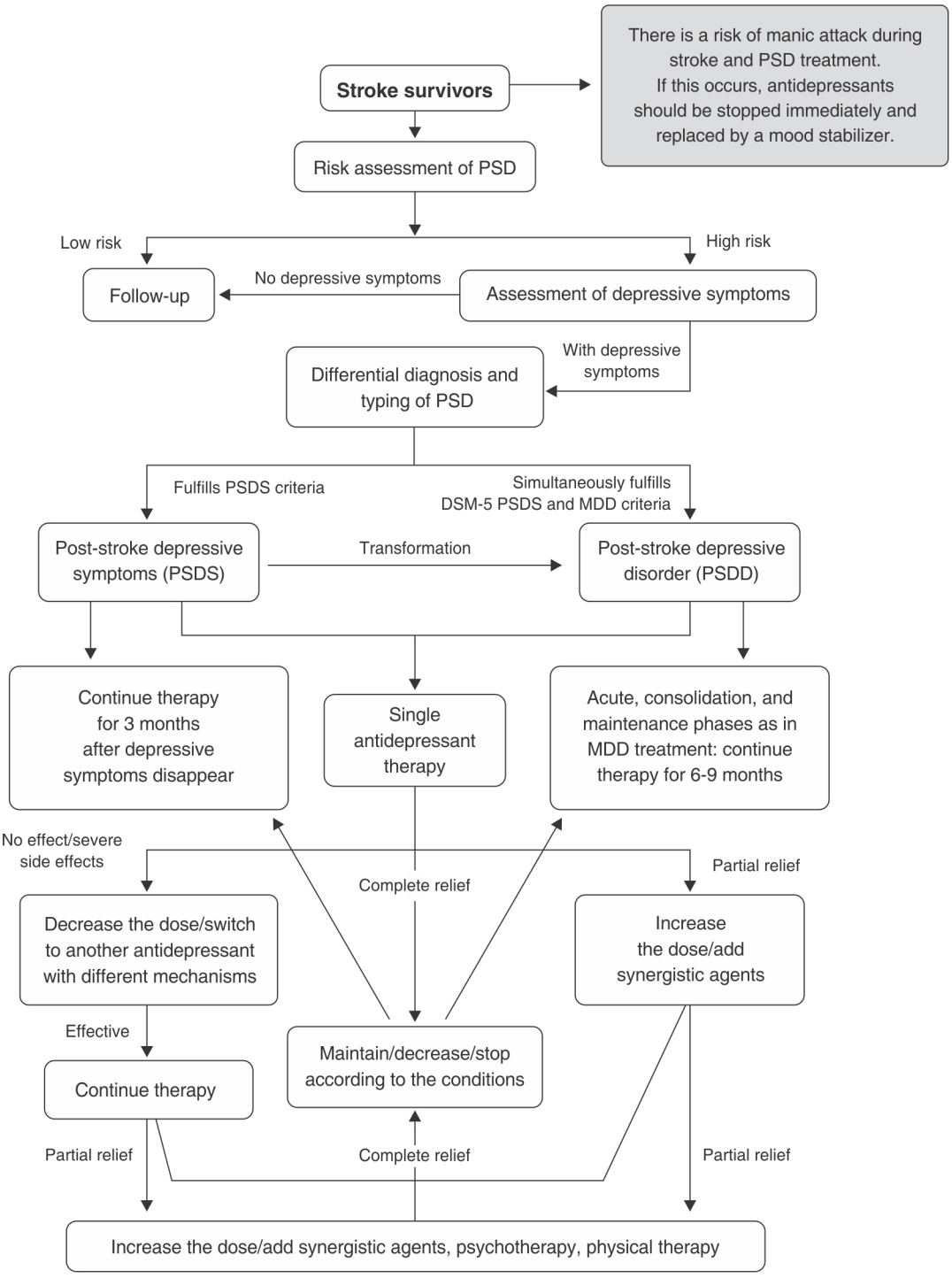

Figure 2 The diagnostic and treatment procedures of PSD.MDD = major depressive disorder; PSD = post-stroke depression.

which can improve therapy outcomes and cure rates. In addition, due to the limited therapeutic modes available for mental disorders, successful implementation of a three-step prevention program is an efficient strategy for decreasing the burden of disability and related mental and behavioral disorders on the family and society. The first step in prevention focuses on etiologies, reducing the prevalence of PSD with the most active measures. The second step emphasizes early identification, diagnosis, and treatment to obtain a favorable prognosis. The third step is to prevent relapse, which requires good rehabilitation to thoroughly promote functional recovery and quality of life.

Conclusion

Since PSD is a frequent phenomenon in stroke survivors and strongly impacts physical and cognitive outcomes, diagnosis and treatment are essential for the management of stroke patients. However, the etiology and pathogenesis of PSD is not clear. Comprehensive assessment of stroke patients can help recognize individuals at high risk. Thus, the present set of PSD classification and diagnostic criteria have been proposed. Antidepressant treatment is required as soon as the patients are diagnosed with PSD. The treatment choice depends on the patient's characteristics and overall condition, the severity of the symptoms, and adverse effects. Identifying the mechanism of PSD is important for future research, since it may lead to a specific therapeutic interventions. In conclusion, clinicians and related workers should be aware of PSD and be able to recognize the disease in a timely manner to help patients out of their depressive mood as quickly as possible.

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Disclosure

The authors report no conflicts of interest.

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