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Management of post-stroke depression in elderly

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Abstract: Depression is one of the most common psychiatric manifestations in post-stroke patients. It affects approximately one third of all stroke survivors and is often undetected, undiagnosed, untreated or undertreated. These unsatisfactory situations are due to assessment complexities of mood disorders in disabled patients and patients with medical co-morbidities. Post-stroke depression (PSD) hampers rehabilitation and negatively alters the outcome of the therapy. The etiology of depression in post-stroke patients is complex and multi-factorial in origin. Family history or previous history of any depressive disorder distinctly makes them prone to be affected by depression. Depression followed by stroke is characterized by vegetative signs and symptoms and less likely to include dysphoria. Due to vague symptoms of depression that emerge as signs and symptoms of stroke, diagnosis of depression in elderly stroke patients is more challenging. Antidepressants are not generally used for mild depression because the balance of risk and benefit is not satisfactory in elderly stroke patients. Selective serotonin reuptake inhibitors (SSRI's) are used as the first line treatment for PSD in elderly patients. SSRI's has a lower potential for drug interaction and development of side effects whereas interactions and side effects are common for tricyclic antidepressants. Now stimulant medications have emerged to be a new promising therapeutic intervention for PSD. Other treatment methods like cognitive behavioural therapy and electroconvulsive therapy are also indicated for severe refractory PSD.

Keyword

Stroke, Post-stroke depression, Depression in stroke survivors, depression, antidepressant

Introduction

Stroke is an acute, focal or diffuse, dysfunction of the brain, originating from the vessels and lasting for a period longer than a day. It's a disease that predominantly occurs in adults and the elderly. The loss of blood supply to the brain is caused by thrombotic, embolic, or hemorrhagic events. Risk factors for recurrent stroke development include hypertension, hyperlipidemia, diabetes mellitus, sleep apnea, obesity, and cardiac diseases. Etiologically, stroke can be of two types, ischemic and hemorrhagic, where 85% of strokes are ischemic and hemorrhagic stroke accounts for 12%. The incidence of stroke varies drastically over the life course, with incidence rates of 10-20 per 10,000 individuals in the age range of 55–64, while incidence rates increase to 200 per 10,000 individuals for those aged over 85.

Depression occurs in roughly one third of stroke survivors and is associated with poor functional outcome and higher mortality. The consociation of neuropsychiatric disorders with cerebrovascular disease includes depression, anxiety disorder, apathy, psychosis, cognitive disorder, mania, catastrophic reactions, pathological affective display, fatigue, and anosognosia. The relationship between depression and stroke was first studied by Martin Roth. Later, Folstein reported that depression was the commonest psychiatric disorder in stroke survivors rather than patients with similar levels of motor disability caused by orthopedic problems. PSD is characterised by increased disability, reduced participation in rehabilitative programs, and worse rehabilitation results.

The diagnosis of post-stroke depression (PSD) is extremely hard due to concomitant focal cognitive disturbances or generalized intellectual impairment, and in certain cases, dissociation of mood and affective behaviour. The evaluation of depression in elderly patients is more complex. That is because of the influence of somatic conditions like cognitive, sensory, and language impairments. Some factors might overlap typical symptoms of depression from normal signs of aging and stroke disease, including reduced sleep, thoughts regarding death, fatigue, and loss of libido. The diagnosis has been methodized on the basis of psychiatric criteria in Diagnostic and Statistical Manual (DSM)-III, an approach that criticizes non-specific stroke-related somatic symptoms. Diagnosis of PSD by comparing the severity of depressive symptoms in stroke to that of non-specific symptoms influences the diagnosis of PSD. The prognosis, however, of early versus late diagnosed Pos-stroke depression and response to treatment of early versus late diagnosis of PSD differs, indicating that diagnosis of PSD by DSM-III criteria covers a heterogeneous group of etiologically different conditions.

Numerous therapeutic strategies are used for the treatment of post-stroke depression that have proven to be effective. This includes pharmacological and non-pharmacological interventions, psychotherapy, electroconvulsive therapy etc. Anti-depressant drugs, especially selective serotonin reuptake inhibitors (SSRI's), are mainly used by clinicians for the treatment of PSD. The SSRIs have fewer adverse effects than those of tricyclic antidepressants. There are a variety of psychotherapeutic methods used for depressive disorders, including cognitive behavioural therapy (CBT) and problem solving therapy (PST). The focus area of CBT is on changing cognitive patterns in order to change behavioural and emotional states. And PST focuses on improving patient skills and the ability to deal with their specific everyday problems.

Epidemiology of post-stroke depression

Approximately around one third of stroke survivors develop post-stroke depression at some point in time after a stroke. The frequency of depression occurring in stroke survivors is higher in the first year, occurring in nearly 1 in 3 stroke survivors, and declines thereafter. The prevalence rates of PSD vary exponentially from 6% to 79%. These rates show considerable variation between different populations and different age groups. The rate of post-stroke depression depends dramatically on the location where the examination is done. Greater rates are shown among hospital inpatient-based settings like general hospital wards, acute stroke units or rehabilitation centres, whereas the rate decreases in community-based settings. The difference in evaluation instruments combined with different definitions of PSD and the mode of selection of patients alter the result at an enormous rate. During the acute period, which is one month after a stroke, depressive disorder was 30 percent and 36 percent in rehabilitation and hospital settings. The prevalence of depressive

disorders in adults over 55 has been estimated to be 18% for women and 11% for men. This also includes patients with dementia, impaired communication or patients with aphasia. Among the elderly population in the United States, a public survey on the prevalence of depression showed 42.9% for men and 64.1% for women, often the highest rate of stroke survivors. Linden et al showed that the frequency of depressive episodes in a series of stroke patients who had been hospitalized for 20 months after the onset of stroke was 34% in stroke patients and 13% in control.

Development of depressive in older vs younger adults

The occurrences of depressive disorders in younger adults are different from depression in older adults. When comparing the depression of elderly adult adults, there is a marked increase in the level of somatic symptoms, including lack of interest and other emotional symptoms, such as dysphoria, nausea, or guilt. Mostly, somatic symptoms often mask depression in the elderly, because of the somatic nature of the disorder or because of the increase in symptoms of existing physical illness. Older patients are very likely to have a declining mood, so often they don't meet the DSM-IV (Diagnostic and Statistical Manual of Mental Disorders) criteria for depression. Also, the symptoms of depression are masked by old age symptoms and other diseases that they are suffering. While comparing both these categories, elderly patients often hide their mood because of the thought that decreased life fulfillment is normal with aging and in a disease state. In all patients of depressive disorder, lack of concentration, loss of memory are some common symptoms. But symptoms like fatigue, sleep disturbances, mental retardation, and hopelessness about the future are more common among depressed younger adults.

The difference in the rate of development of depressive disorders in younger and older adults is given as follows, where + sign indicates severity.

	Younger adults	older adults
Cognitive impairment	+	+++
Depressed mood	+++	+(+)
Retardation	++	++
Anxiety	+(+)	+++
Somatic symptoms	+	+++
Psychotic symptoms	(+)	++
Hypochondria	+	++

Risk factors of post-stroke depression

Risk factors are the determining factors that are associated with an increased chance of the occurrence of a disease. They can be genetic or personal behavior, lifestyle or environmental exposure. Several factors have been identified that cause the risk of developing post-stroke depression. They are genetic factors: age, gender, type and severity of stroke, medical and psychiatric history, location of lesions, degree of disability, and social support. Some symptoms of PSD are the same as those of the risk factors, like reduced social activity, poor participation in the rehabilitation process and failure to return to work. Also, gender has lately been found to be mildly associated with PSD. Occurrence of PSD in women is twice as common as in men. And some studies indicate that older, physically disabled men along with inadequate social support were more likely to be diagnosed with PSD.

Common genetic mutations may provide a risk or ability to develop dementia when a person is exposed to an unfamiliar depressive challenge. A few genes have been evaluated as risk factors for PSD. 5-HTTLPR and STin2 VNTR polymorphisms of the serotonin transporter gene (SERT) have been linked to PSD in stroke survivors. Epigenetic modification of 5-HTTLPR is also affected by the onset and severity of PSD. Individuals with a 5-HTTLPR s/s genotype have 3 times higher chance of PSD compared with l/l or l/xl genotype carriers. Participants with the STin2 9/12 or 12/12 genotype have 4 times higher chance of PSD compared with the STin2 10/10 genotype. Thus, 5-HTTLPR and the STin2 VNTR polymorphisms of serotonin transport genes are associated with the development of post-stroke depression.

Some major cardiovascular risk factors like high blood pressure and hypercholesterolemia are seen to be unrelated with the development of PSD. But a history of diabetes mellitus may be present in patients with PSD. A personal history of depression or anxiety or both was identified as a risk factor for PSD. The location of lesions has been extensively studied as a risk factor for PSD. Two studies led by Robert G. Robinson in 1984 and 1987 reported that severe stroke patients with left or right basal ganglia lesions had a much higher frequency of major or minor depression. In extension to that, both studies show significant correlation between the anterior margin of the ischemic lesion and the anterior left ventricle in the severity of depression in both the cortical and sub cortical regions. But this is still important only during the first 6 months after a stroke. As a result of the studies conducted by Robert G Robinson, reports show an association between PSD and anterior or left basal ganglia lesions within 2 months of the first clinical stroke. Both depression and cognitive impairment are connected with worse outcomes of stroke. The extent of post-stroke disability in daily life activities is a factor that is strongly related with PSD. The strength of the relationship between PSD and the deterioration in daily life activities is weak, accounting for only about 10% of PSD severity variability. Preliminary studies showed that patients with high-stroke had significantly lower points in the Mini-Mental State Examination than non-depressed patients with similar features compared to both the site of the lesion and the volume of the lesion. This finding was repeated in an independent study of stroke patients with left hemisphere lesions that were evaluated in the first year after a stroke.

The available evidence regarding PSD and community support is contradictory, probably due to significant differences in the definition and evaluation of community support. For example, although the value of public relations was shown to be negatively correlated with the size of PSD, living conditions and marital status were not statistically correlated with PSD. However, a prospective study found that the lack of accepted social support was associated with the onset of PSD.

Clinical manifestations of post-stroke depression

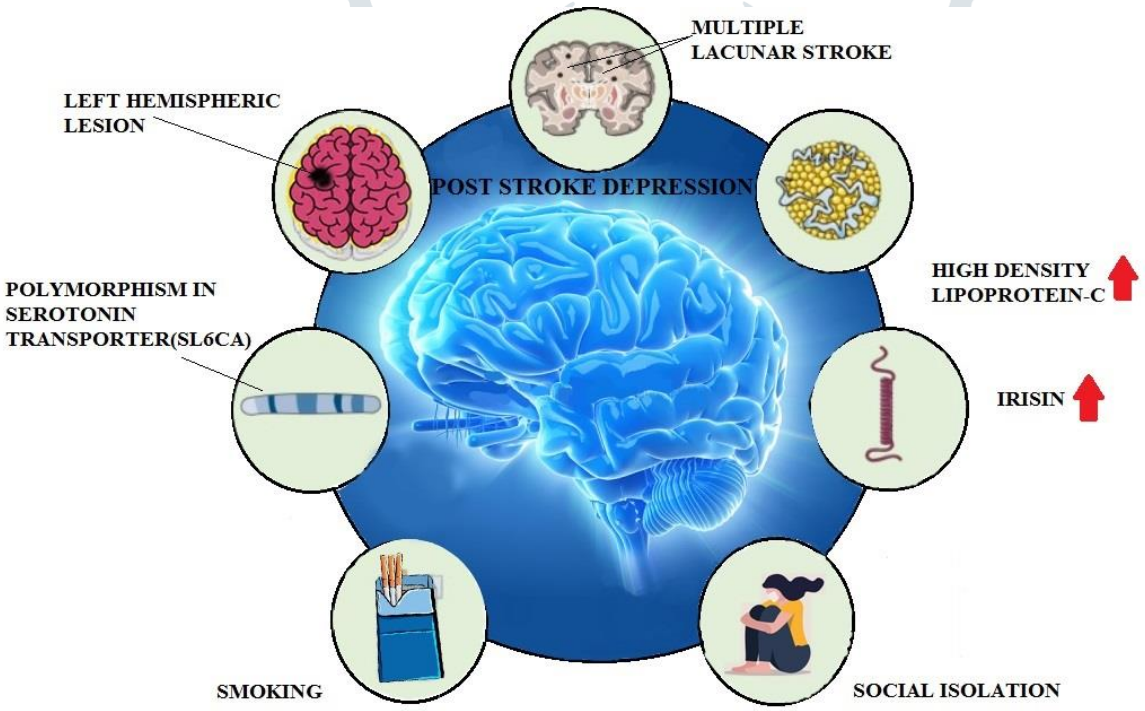
The post-stroke depression that occurs within the first three months is said to be early and when the symptoms occur after three months, they are said to be late. PSD may appear soon after a stroke but usually begins a few months after a brain event. High incidence and severe depression usually occur between 6 months and 2 years after a stroke. Early PSD patients have more depressive symptoms compared to psychological symptoms and often show earlier symptoms of melancholy, vegetative symptoms, and psychiatric disorders. Symptoms of depression at any level of severity can be seen in 18% -30% of patients within 3-5 years after a stroke. Depression occurs differently among patients with neurological syndromes. Depression following stroke, especially right hemisphere damage, is less likely to cause dysphoria and is more likely to show signs of symptoms and symptoms compared with other types of recent depression. Patients with a stroke are more likely to show withdrawal symptoms and are less likely to show anxiety compared with adult depression associated with other medical conditions, although there is no difference in the state of depression or strength between them. Somatic symptoms are also more common among patients with a stroke where a depressive disorder is present. In this case, physicians are more sensitive to the similarities between somatic features, which are more common in older patients, and are the commonest clinical features of depression. For a more accurate diagnosis, doctors should depend more on non-somatic symptoms rather than somatic symptoms. Other symptoms are similar in both stroke and depression, such as sleep disturbances, difficulty concentrating, and decreased appetite. For older people with dementia due to old age and dementia, additional mental retardation caused by stress may be more severe. About 2% –3% of patients without a stroke show hidden depression, which is a symptom of depression other than depression and manifestations of biological rhythm disturbances such as insomnia or hypersomnia and allergies, especially autonomic vascular dystonia, vertigo, or various other events.

Pathogenesis of post-stroke depression

The pathogenesis of PSD is complex and involves a combination of biological and psychosocial factors. Exact pathogenesis of PSD is not yet identified so multifactorial reasons are proposed. This involves biological and psychosocial components, which may vary depending on the timing after the event.

Understanding the pathogenesis of PSD may be helpful in its management; for example, PSD-induced biological causes may respond better to medication, while PSD-induced psychiatric and social interventions may respond better to psychotherapy and social support interventions. Some proposed biological factors that contribute to the development of PSD include lesion location, genetic susceptibility, inflammation, alterations in neurotrophic factors, neurogenesis in response to ischemia, and alterations in serotonergic, noradrenergic, and dopaminergic pathways, leading to changes in amine levels. However, there are not many hypotheses that are strong enough to identify them as the only neurobiological pathogenesis of PSD. Conversely, PSD may be caused by a variety of biological factors that may exist and interact in a complex way. In addition, PSD can be classified as different subtypes depending on the symptoms or the initial time. There are already studies showing that certain pathogenesis may be different from the different subtypes of PSD. The primary biological mechanism is considered as the cause of PSD, where ischemic shock directly affects the neural regions involved in the regulation of emotions. Damage to the catecholamine pathway of the brain reduces the release of neurotransmitters from possible stress as a result. Decreased cortical biogenic amines are found after pre-cortical circulatory disorders after stroke. However, there are also issues with the PSD psychological basis presented by researchers. This is related to the similarity of the symptoms and profiles of treatment response between PSD and active depression. Carson et al conducted a meta-analysis in which they noted that the risk of depression was not related to the area of cerebral lesion. However, some stroke survivors may only have a natural PSD, while others are only psychological.

Figure 01 causes of PSD



Diagnosis of post-stroke depression

Stroke patients present unique challenges in diagnosing depression. Symptoms of a stroke related to a stroke such as aprosodic speech, abulia, or flatulence may prevent doctors from diagnosing PSD, and aphasia may lead to undiagnosed and inadequate treatment for depression. The symptoms of PSD can be subtle, such as refusal to participate in treatment. Patients may experience an emotional obligation or a pseudo bulbar is affected after a stroke, which often causes the team to mistakenly diagnose a patient with PSD. Emotional bonds can frustrate the patient and the family; however, symptoms usually subside over time and do not require treatment for depression. There are no accurate diagnostic methods for PSD in the current generalized diagnosis and classification systems of mental disorders. Some studies have adopted the DSM-5 diagnostic method or simply relied on a stress test scale to diagnose PSD. The disease was simply described as ‘‘depressive disorder ’’ and was divided into three subtypes: with depressive features, with a major depressive-like episode, or with mixed features.

The neurobiological pathogenesis of PSD has been studied extensively. The Studies of pathogenesis of PSD have provided various results about the different neurobiological hypotheses of PSD involving lesion characteristics, neurotransmitters, inflammatory factors, hormones, and neurotrophins. However, none of the multiple hypotheses is dominant enough to be identified as the only or main neurobiological pathogenesis of PSD. The most advanced diagnostic method in the clinical setting is a structured psychological examination, which results in the diagnosis being achieved to a certain extent, such as the Diagnostic and statistical Manual of Mental Disorders [DSM] or the International Disease Separation criteria. DSM diagnostic procedures for depressive disorder include paralysis, fatigue, and sleep and appetite disorders, all of which may be the result of the stroke itself and without emotions. Behavioral disorders, facial expressions, and verbal communication caused by a stroke may mask or mimic symptoms of depression. In addition, slow-paced mental conversations are not only time-consuming but also weighty in response to words. Emotional scores and questionnaires are the preferred method for assessing the burden of depressive symptoms in general clinical care. Certain patients with dysphasia, impaired writing ability and visual impairment, are unable to compile a questionnaire and therefore patient feedback and visual cues are used to complete it.

Management of post-stroke depression

There is no reason to reject any form of treatment for elderly patients with PSD based on age alone, as many older people had a higher basic life expectancy and a life expectancy that would last for many years. The main problem, which can be cited as the leading cause of PSD treatment, is that the patient and the physician generally do not accept the condition as a treatable disease. The most troubling problem is that when examining an older patient, the doctor focuses on other aspects of the patient and dismisses the symptoms of depression. Unfortunately, it is estimated that 80 percent of PSD patients may miss out on psychiatrists. At the same time, early diagnosis of PSD is critical to developing an effective treatment plan for the patient. The importance of early recognition and diagnosis of PSD is widely agreed upon to improve performance and psychological outcomes. All treatments should be tailored to individual needs based on patient needs, including cost, accessibility, and availability of treatment. Active treatment will usually include family participation and other support networks. In all cases, it is recommended that a physician refers a person who is showing depression at least weekly for the first 6 weeks to assess mood swings, suicidal thoughts, physical safety, social life, and side effects of any drugs used.

The focus point of the treatment is to eliminate or reduce all symptoms of depression and thereby improve your mood and quality of life. Another accompanying aim is to reduce the risk of complications including relapses, and facilitate the use of health care services. The intended outcome of the treatment is complete relaxation and well being for a long time. The effective treatment methods used for the management of PSD are pharmacotherapy, psychotherapy, and electroconvulsive therapy, which are all used primarily for patients with severe depressive disorders.

Pharmacotherapy

Even after studies on pharmacotherapy of post-stroke depression were done more than 100 years ago, there are still several challenges regarding the therapy or choice of drugs for post-stroke depression and its effect. Pharmacotherapy can be especially difficult for older people with PSD, who often have high levels of medical comorbidity, associated polypharmacy, and are at high risk for adverse effects of antidepressants (ADs). It is important that the antidepressants used should not only be effective in controlling emotional disturbances but also have no adverse effects on mental functions, especially for patients who have had a stroke and more or less severe mental retardation. There are many variables involved in pharmacotherapy for elderly patients, including pharmacokinetic changes related to aging, drug interactions with other drugs, existing diseases, and adverse drug reactions in the elderly due to their increased risk.

Antidepressant drugs in post-stroke depression

Antidepressants can be effective in many moderate and severe depressive disorders but they are often not indicated in mild conditions, because the balance of benefit and risk is unsatisfactory in elderly patients with stroke. Even when depression is well tolerated in older patients, they may not get the medication they need, and because of the potential adverse effects of antidepressants in elderly patients, doctors often avoid

prescribing such medications. Unfortunately, even when antidepressants Treatment is given to an older patient, it is mostly in inadequate doses and for shorter durations than recommended.

Some common side effects caused by antidepressants include dry mouth, slight blurring of vision, constipation, problem passing urine, drowsiness, dizziness, weight gain, excessive sweating, heart rhythm problems etc. It is widely accepted that all antidepressants work in the same way, so agent selection is based primarily on antidepressant tolerance and adverse effects profile. Starting with low doses such as half the normal active dose and gradually increasing the dose after one to two weeks until normal dose is reached which results in improved tolerance and fewer side effects. Patients who do not have at least a partial 4- to 6-week response to a standard adult treatment dose are less likely to respond to that treatment. In such conditions the drug should be switched to a different anti-depressant medication. In patients who experience significant clinical improvement within four weeks of treatment dose, it is reasonable to extend the initial treatment if the current antidepressant is tolerated. Nutrition treatment may be needed in patients at high risk for recurrent episodes of depression. The care phase begins when the doctor checks that the patient is well but is at risk of recurrence, which can last for many years and perhaps forever.

Various studies have been performed on antidepressants that have found its efficiency to reduce symptoms of depression. However, when examining the Diagnostic and Statistical Manual of Mental Disorders (DSM) or the Hamilton Depression Rating Scale (HAMD), there is no clear evidence that antidepressants are effective in the complete remission of PSD. The best-known agents are fuoxetine, sertraline, citalopram, and nortriptyline. The main goals of PSD treatment include reducing symptoms of depression and complete relief. The complete relief means the patient no longer meeting the baseline criteria for depression.

The antidepressant selection for the treatment of adult should e done with high optimisation. they should be a unaltered drug handling in old age, Interaction free, Safe in frail subjects with comorbid illnesses, Simple dose regimen, Well tolerated, Rapid onset of antidepressant action. During antidepressant therapy is followed the following criteria's should be adopted:

•The choice of antidepressant is based on profiles of side effects, history of previous response, simultaneous illness, simultaneous medications, and antidepressant costs.

• Low initial doses should be considered and gradually increased to a full dose of antidepressant for older people, who is at high risk of side effects.

• Before discontinuing antidepressant medication as a failure, providers should ensure that the appropriate dose and target dose range is reached and that a reasonable response time is allowed (minimum of four to six weeks).

• Discontinuation of antidepressant therapy should be done with a slow taper, as it may cause severe withdrawal symptoms or recurrence of true depressive symptoms. Tapering should be directed at the elimination half-life of the parent compound and metabolites and carefully monitor the symptoms of depression.

Selective serotonin reuptake inhibitors (SSRIS)

Selective serotonin reuptake inhibitors (SSRIs) are the first line choice of PSD treatment in elderly patients due to their lower potential of drug interaction and development of side effects. The SSRIs or Selective Serotonin Reuptake Inhibitors act at the level of synapse, the exhibit their action by blocking serotonin reuptake. The SSRIs are usually considered as the first line drugs for the treatment of PSD because of its high level of tolerability in the population. These categories of drugs are often accompanied by nausea and mild headache. Other observed side effects are decreased libido and difficulty achieving orgasm, which are noted as persistent problems. The drug paroxetine also has same adverse effects and also capable of ant cholinergic side effects. Most importantly, SSRIs can increase the risk of bleeding in the elderly and may cause hyponatremia, which requires monitoring sodium levels. The risk of bleeding results in inhibition of platelet function, and the disorder may range from injury to intestinal bleeding. Therefore, caution should be

exercised with respect to hemorrhagic strokes. Other possible side effects include anorexia at the beginning of treatment; intestinal disorders such as nausea, vomiting, and diarrhea; sedation or insomnia; and serotonin syndrome. Sertraline seems to have the worst effects of GI.

The patients who fail or are intolerant to any one SSRI can be switched to another SSRI or to another first-line antidepressant. Following a second failure with a different SSRI, the patient should be switched to another class of antidepressant

Advantages of SSRIS over TCA (Tricyclic Antidepressants)

• Faster onset of action compared with TCAs

• At least as effective as the TCAs

• SSRIs have a more favourable safety profile

• SSRIs are generally not affected by age-related alterations in drug metabolism

• SSRIs can be administered in a simple, once-daily regimen, less confusing for elderly patients

• Improved tolerability of the SSRIs makes them a more appropriate choice for elderly patients

• SSRIs do not cause the ant cholinergic effects like TCAs

• TCAs are potentially fatal in overdose (great concern in elderly PSD patients who are at increased risk of suicide)

• SSRIs are associated with a lower potential of drug interaction compared with TCAs

• A safer side-effect profile compared with TCAs

Nonpharmacological treatment of post-stroke depression

A number of factors have been suggested in favour of non-medical treatment, including the complications which may occur on treating older adults who are depressed by the adverse effects of AD. Their inability to reverse severe environmental stressors and lack of social support would often respond to psychological trauma. Non-medical interventions in PSD included psychotherapy, ECT, and transcranial magnetic stimulation. Psychological interventions are the preferred method of treating central nervous system disorders and are reserved for those with AD. In many psychiatric therapies, part of behavioural activation is often involved, which deals with the problem of functional limitations. Although some of these approaches are more focused on job understanding during treatment, it is more likely to focus on stress and strengthening and maintaining understanding has been done. Psychiatric treatment includes behavioural therapy, psychotherapy (CBT), problem-solving therapy, and lifestyle review therapy.

Cognitive behavioral therapy

CBT focuses on changing cognitive patterns in order to change behaviour and mood. CBT has a strong and positive effect on patients because it not only improves and builds self-esteem but also improves patients' daily lives through a series of activities. Treatment requires trained health professionals to regularly assess participants, making it easier to meet a variety of individual needs. CBT is based on providing information on psycho education, collaborative empiricism, effective problem solving, quality assurance and quality support, and improving adaptation to a new lifestyle after a stroke. Patients will discover how their thoughts can affect these affected symptoms and feelings and how they can change them. Generally, approximately 6-

8 times should be given to patients within 10-12 weeks, most people experience a improvement in mood or a decrease in symptoms after 2 months of treatment. The treatment response should be reviewed after 8 sessions. A period of 6 months of treatment is considered necessary for someone with multiple problems or serious illness. Psychotherapy should be combined with AD to reduce the remaining symptoms and the risk of relapse in patients with severe depression and in those with moderate or severe depression, who refused to take AD.

Electroconvulsive therapy

Electroconvulsive therapy should only be used to achieve rapid and short-term improvement of severe symptoms of after other therapies appear to be ineffective. It is not recommended as a primary or enduring treatment for PSD. It is more commonly used in older adults compared to any other age groups. Its main symptom is a serious depressive disorder or when the disorder or its symptoms are considered dangerous to health. ECT involves a brief passage of electrical energy from the brain to induce a generalized seizure. The main symptom is a serious depressive disorder, or when the disorders, or your symptoms, are considered dangerous to health. Electroconvulsive therapy should be administered only by properly trained health care professionals under approved guidelines and should be used to achieve rapid and short-term improvement of severe symptoms after other treatments appear to be ineffective. Since the long-term benefits and risks of electroconvulsive therapy are consistent, ECT are considered as an effective mode of non pharmacological treatment method of post-stroke depression. However, a series of cases suggest that the presence of a stroke does not increase the risk of the procedure or treatment in patients with severe depression. Electroconvulsive therapy is therefore not a recommended treatment for depressed patients, and adverse events such as heart problems, memory loss, and delirium raise awareness about the use of ECT in older PSD patients.

Conclusion

Depression is common after stroke, affecting one-third of stroke survivors at any given time. The natural history of PSD is strong; however, symptoms usually appear in the first year. The neurobiological pathogenesis of PSD is far from clear. The PSD study yielded mixed results regarding different neurobiological perceptions of the PSD including wound characteristics, neurotransmitters, inflammatory factors, hormones, and neurotrophins. However, there are not many hypotheses that are strong enough to identify as the only neurobiological pathogenesis of PSD. In contrast, PSD may be caused by a variety of biological factors such as genetic predisposition, inflammation, neurotrophic mutations, disruption of neural networks, and serotonergic, noradrenergic, and dopaminergic pathways. Stroke patients present unique challenges in diagnosing depression. The most advanced diagnostic method in the clinical setting is a systematic psychological examination, which leads to the diagnosis being achieved to some degree such as the Mental Diagnosis and Statistics Manual [DSM] or the International Disease Separation criteria. Antidepressant treatment is required as soon as patients are diagnosed with PSD. The choice of treatment depends on the patient's condition and overall condition, severity of symptoms, and side effects. Identifying the PSD method is important for future research, as it may lead to certain therapeutic interventions. Antidepressants are beneficial for those with chronic depression, and treatment with antidepressants should be continued for at least 6 months in those who respond to treatment. Considering the possible side effects of medication and difficulty with the diagnosis of major depressive disorder, psychiatric treatment has been recommended as an alternative. Along with medical treatment, non-pharmacological therapies are also included that include behavioural psychotherapy, electroconvulsive therapy, antidepressant therapy, and lifestyle rehabilitation therapy.

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