

Post-stroke depression

Depresión después del infarto cerebral

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We all know that cerebral vascular disease is one of the leading causes of morbidity and mortality worldwide and that the costs of patient care can destabilize entire families and health systems, so it is important to seek early access to care and prevention. After a cerebral infarction, follow-up by the neurologist is oriented at finding the etiology and initiating timely secondary prevention. However, other comorbidities such as depression can have a major impact on a patient who has had a stroke.

Depression post-stroke is more prevalent in woman and stroke patients had a 55% greater risk for depression than patients with myocardial infarction¹. At least one third of stroke survivors present mood symptoms post-stroke^{2,3} with prevalence ranging from 20% to 60%⁴. In this edition, García-Valadez et al., report the prevalence of post-stroke depression in vascular neurology consultation. They found that although 36.6% of the patients evaluated had criteria for depression, only 1% of them had a previous diagnosis of depression.

Post-stroke depression often has a chronic course and is associated with a variety of adverse health outcomes, including increased disability, morbidity, and mortality. Depression persists after 20 months in 34% of elderly patients with acute stroke and has been associated with worse cognitive and physical outcomes. Post-stroke depression is associated with increased short-term (12 and 24 months) and long-term (10 years) mortality⁴.

Comorbid psychiatric conditions, especially anxiety, are associated with a greater risk of post-stroke depression. Older age, male sex, and non-white race were protective factors¹. Among the external factor contributing to post-stroke depression is the loss of social support^{4,3}.

There are several hypotheses about the genesis of post-stroke depression, such as the disconnection of the brain's prefrontal systems or its modulatory pathways⁴. Decreased serotonin, noradrenaline, and dopamine levels, abnormal neurotrophic response to stroke and dysregulation of the hypothalamic-pituitary-adrenal axis are factors that altered neurogenesis and neuroplasticity². Cerebral infarction lesions in the basal ganglia and left hemisphere have been related to post-stroke depression³. Inflammatory and hypoperfusion mechanisms impact brain function influencing depression⁴.

The period of adaptation after stroke can make it complicated to diagnose depression. Some patients may experience "grief", which can be considered a normal emotion in the period of adaptation to the new conditions after stroke, as the patient may be affected by autonomy loss and changes in personal relationships.

Halmiton Depression Rating Scale; 13 and the SF-36 questionnaire are useful to assess the severity of depressive symptoms and response to treatment during follow-up. The assessment can be performed by the neurologist, so the training of neurologists should be enriched with basic neuropsychiatric approaches, to be

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able to initiate treatment in necessary cases and to refer to a psychiatric specialist.

The screening for post-depression stroke could be considered during acute care stay in patient at high risk for depression, during transition points care and considered following discharge to the community⁵. Drug interactions with other drugs and adverse events should be considered in the choice of pharmacological treatment. The cognitive-behavioral therapy is a psychosocial intervention aimed at reducing symptoms of depression.

Remission of depression in the 1st month after stroke is associated with better recovery in activities of daily living⁴. In addition, social support is a protective factor against depression through emotional support, motivation for treatment, and support in daily functioning³.

In conclusion, we should consider that all patients with stroke have a high risk of developing depression, so we should include in their evaluation screening to search intentionally for depression and initiate timely treatment.

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