

## Depression and Stroke

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**Summary:** *The prevalence of depression is increasing. It is the number one cause of morbidity and disability in the world. 30 to 55% of patients with the initial stroke have depressive symptoms. Most studies find a prevalence after stroke, of major depression between 0 and 25% and of minor depression between 5 and 40%. The interactions between reactive depression and the effects of brain damage remain complex. Diagnosis is often difficult, especially in patients with cognitive impairment. Recent work has suggested that depression in the elderly may be associated with structural abnormalities in the brain parenchyma, as evidenced by the presence of hyperintensity abnormalities in brain imaging (with nuclear magnetic resonance [NMR] above all), in particular subcortical. Treatment should ideally combine psychological, social and medication.*

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### I. Introduction:

Depression, commonly seen after a stroke, is a now well-defined clinical entity and, like other stroke symptoms and sequelae, must be recognized and treated early.

Diagnosis is often difficult, especially in patients with cognitive impairment.

Treatment must combine antidepressant drugs, psychotherapy and social care.

The mechanisms involved in the development of depression after stroke are not clearly defined and must involve disabilities and their social consequences but also the direct effects of brain damage.

### II. History:

In 1843, Durand-Fardel reported for the first time a link between cerebrovascular pathology and depression.

In 1926, Guilarovsky suggested that in the elderly, the significant frequency of affective symptoms was explained by cerebral arteriosclerotic changes.

Since then, numerous physiopathological, diagnostic, epidemiological and therapeutic studies have been published on this morbid association, without however providing answers to all questions.

Despite everything, post-stroke depression (PADD) can now be recognized as a separate entity even if DSM-IV classifies it as "mood disorders linked to a general medical condition."

### III. Epidemiology:

30 to 55% of patients with the initial stroke have depressive symptoms. Most studies find a prevalence after stroke, of major depression between 0 and 25% and minor depression between 5 and 40%. Depression becomes chronic (more than 3 years after stroke) in a minority of patients. The prevalence of DPAVC with an early onset is 80% of cases and delayed for several months in 20% of cases. Most often, the spontaneous course of the DPAVC is towards regression in 6 to 12 months, with frequent relapses.

The risk of a major depressive episode seems to be increased by age, the presence of chronic pain, isolation and alcoholism. The other predisposing factors for a CVADP are the severity of the deficit, a neurotic premorbid personality and a personal or family history of psychiatric pathology. Non-fluent aphasia, cognitive impairment including memory and attention skills, and an increase in the ratio between the size of the ventricles and that of the brain could also be associated with a higher risk of developing CVAD. The reverse link between depression and stroke seems to exist, Ramasubbu reports the probable role of depression on the occurrence of a stroke and on the increase in vascular risk factors.

Men with depression and anxiety are more than three times more likely to die from a stroke than others. This is explained by changes in heart rate and contraction of blood vessels seen in depression, an increased propensity for platelet aggregation, and higher cholesterol.

#### **IV. Mechanisms:**

Several studies have recently looked at the links between the onset of post-stroke depression and the areas of the brain affected by stroke, mostly with conflicting results.

A neurochemical origin related to disturbances in the metabolism of serotonin induced by stroke would be more involved in early depression while late depression would be induced by residual disabilities whether related to physical, motor or sequelae. cognitive or with consequent social and family repercussions. A premorbid thymic state would promote early depression.

The interactions between reactive depression and the effects of brain damage remain complex. Indeed, if certain brain lesions can cause or promote the development of depression, they can also mask it or make the diagnosis difficult. The reaction component of the DPAVC is integrated into the classic phases of mourning that are incredulity, revolt, depression and acceptance. However, after the stroke, these 4 classic phases are most often incomplete and disorderly. The notion of acceptance of the handicap implies the mourning of several functions and statuses (walking, grasping, body diagram, language, leisure and professional activities, salary, social and family status, sexuality ...) and the serious and irreversible deterioration self-image that flows from it.

#### **V. Localization of the lesion and depression:**

Knowledge in the field of endogenous depression has led to the search for dysfunction of specific brain regions in the DPAVC, such as the limbic and temporal regions but also interconnected regions involved in emotional processes.

Regions such as the basal ganglia and premotor areas by the role they play in strengthening or inhibiting goal-directed behaviors could also be involved.

In endogenous depression, imaging studies have shown a tendency to reduce the size of the basal glands, hippocampus, and tonsil glands, cortical and subcortical atrophy, white matter hyperintensities, and an increase in the size of the ventricles.

#### **VI. Diagnosis:**

The clinical evaluation of these depressive tables is based on the clinical interview, but it can be supplemented by an evaluation of neuropsychological functions and by the use of scales specifically validated for the evaluation of the affective symptomatology occurring in the course of accidents. cerebral. The clinic for post-stroke depression can be viewed from three different angles

##### **Obvious depression on patent and documented stroke:**

Post-stroke depression appears to be relatively similar to that of major depressive episodes, not secondary to organic damage. The only symptom that seems to lose a good diagnostic value is that of early morning awakening when suicidal thoughts appear to be quite frequent in this context.

##### **Minor depression on patent and documented stroke:**

Dysthymic tables or minor depressions have also been described in the aftermath of stroke If major depression is defined according to DSM IV criteria as the association of a depressed mood (criterion A of DSM IV) and four of the other seven symptoms of the depressed line, diagnosing minor depression requires only two to four of the seven major symptoms of depression. As in patients with a major depressive episode, the complaint of a depressed mood may be absent in patients with minor depressive symptoms.

##### **Obvious depression on silent cerebrovascular lesions:**

Recent work has suggested that depression in the elderly may be associated with structural abnormalities in the brain parenchyma, as evidenced by the presence of hyperintensity abnormalities in brain imaging (with nuclear magnetic resonance [NMR] above all), in particular subcortical.

Three types of confounding factors must be taken into account in order not to overlook or, on the contrary, overvalue a CVADP.

**Indirect factors:** Are stillness (potential confusion with apathy), dysphagia (interfering with eating habits), articulation difficulties with related communication difficulties.

**Direct factors:** in connection with cerebrovascular pathology are also major confounding factors; aphasia, amnesia, cognitive deficit, anosognosia and denial of depressive symptoms, aprosody, apragmatism, drunkenness, apathy, loss of psychic self-activation, frontal syndrome, post-stroke fatigue, emotional lability or "sentimentalism". There is a painful or exhausting experience of activities of daily life such as washing, clothing ...

## VII. Influence of the DPAVC on the functional prognosis:

Failure to participate in the rehabilitation program Social and family relationships are also frequently altered, Greater functional deterioration, Greater dependence on activities of daily living The vital prognosis is also altered, by suicidal risks but also by the refusal of care, in particular poor compliance with treatments aimed at preventing the recurrence of a vascular event.

## VIII. Differential diagnostics:

Chronic fatigue: Fatigue after stroke is a symptom of multifactorial origin It can be physical, linked to effort, somatic in relation to the disease, mental appearing during the execution of mental tasks, and psychological due to a loss of interest and motivation.

The frontal syndrome, observed after frontal lesion but also in the event of multiple lesions of small size, is marked by a withdrawal, a pragmatic, verbal and motor spontaneousness, but associated with normal superior functions. In this case, the identification of depression is difficult and a complete neuropsychological assessment must often be offered.

Symptoms directly related to the stroke, without associated depression can be observed, in particular in case of vegetative involvement: inactivity and modification of appetite, sleep and libido.

## IX. Treatment:

Treatment should ideally combine psychological, social and medication. All the means which will be implemented to facilitate the patient's life will be as many elements which can enable him not to pass from a normal stage of fear and anxiety to a stage of depression. Psychological care must also be provided by a psychologist and by the entire medical team, within coordinated multidisciplinary work. Finally, psychotherapeutic approaches have also been shown to be effective in the management of post-stroke depression although none of them has been the subject of controlled studies to date. Antidepressants clearly improved symptoms related to mood disorders but had no obvious effect on preventing CVDD or improving the functional prognosis For tolerance reasons only, the most commonly prescribed antidepressants are serotonin reuptake inhibitors. A treatment which must be prescribed for a period of at least 3 months, most often for a period between 6 and 9 months. The cessation will most often be gradual, choosing an appropriate time, away from any potentially destabilizing event such as resumption of employment, the anniversary of the stroke, an upsurge in financial difficulties. Extreme caution will be required at the start of treatment as with any other depression due to the possible anxiety resurgence and psychomotor deinhibition which can lead to a transition to suicidal acts. An anxiolytic treatment should be combined for a short period in patients with suicidal thoughts. Among other techniques, seismotherapy seems to have an interest in the treatment of major depressions resistant to a well-conducted drug treatment. It must be performed at a distance from the stroke,) despite an increased risk of the occurrence of confounding episodes which is mitigated by performing unilateral ECT, on the side of the non-dominant hemisphere. In the event of proven depressive syndrome, it is necessary to ensure good compliance with preventive treatments for a new vascular event.

## X. Conclusion:

Depression is the most common affective disorder seen after a stroke. The origins of this depression are probably multiple, making its etiopathogenic and therapeutic approach complex. The various disabilities (loss of autonomy, deterioration of the body image, desocialization, loss of role in the family) consecutive to the stroke play a primordial role but other elements such as the neurochemical modifications consecutive to the various brain lesions seem to be involved. . The diagnostic process is often complex, hampered by the cognitive disorders frequently associated (aphasia, anosognosia, unilateral spatial neglect, etc.). The DPAVC seems to decrease the effectiveness of rehabilitation, disrupt the patient's social and family reintegration and increase the excess mortality observed in the first years after the stroke.

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