Poststroke depression

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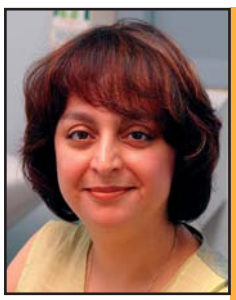

BACKGROUND

Poststroke depression (PSD) is common and often unrecognised. The diagnosis can be difficult due to deficits of stroke such as impaired self reporting and cognition, poor insight and dysphasia. Untreated PSD can interfere with recovery and adversely affect functional and social outcomes.

OBJECTIVE

This article outlines the diagnosis, pathophysiology and treatment for PSD.

DISCUSSION

The natural history of PSD suggests that most PSD is not immediate but develops over months with peak prevalence between 6 and 24 months, and in some cases persists up to 3 years following stroke. General practitioners and treating specialists need to actively monitor patients for PSD. While antidepressant medication is the mainstay of treatment for PSD, psychotherapeutic interventions are important. Treatment should include patient and family education, re-establishment of sleep pattern, addressing functional difficulties, increasing community participation, improving diet and regular exercise.

Stroke is the leading cause of disability in adults and is frequently associated with neuropsychiatric symptoms such as depressed mood, generalised anxiety and apathy. The prevalence of poststroke depression (PSD) varies from 25–79% due to the differences in various study diagnostic criteria, selection of patients, and the time elapsed since the stroke.

Clinical depression is a common complication, and in some long term studies was shown to persist up to 3 years following stroke.[1,2] Depression decreases patients' participation in rehabilitation and impairs functional recovery, community reintegration and long term outcomes.[3] It increases the cost of treatment and burden of care to families.

The diagnosis of PSD can be difficult, and is reported to have been missed in 50–80% of cases by nonpsychiatric physicians.[4] Longitudinal studies on the natural history of PSD progression show that most depression is not immediate but develops over months with peak prevalence between 6–24 months poststroke.[5] Most major depression remits in the 1–2 years following stroke, either spontaneously having run its course or through treatment – it is rarely chronic.[6] Depression also occurs in 40% of primary care givers of patients with stroke and is easily missed.[7]

PSD and functional recovery

Poststroke depression is associated with poor functional and psychosocial outcome.[8] Although there is no long term data on the direct effect of PSD on cost of care there are reports of:

- prolonged inpatient hospital length of stay[4]
- greater disability with activities of daily living[9]
- severe physical impairment[10]
- poor cognitive function[11]
- poor participation in rehabilitation[11]
- reduced social activity[1]
- poor language function[1,2]
- failure to return to work[8], and
- a higher mortality at 10 years.[12]

What causes PSD?

The aetiology of PSD is not well understood. Different mechanisms for PSD may be involved in the aetiology of stroke over time and this has implications for treatment. Depression may be a result of:

- a biologic effect of brain damage
- a reaction to the losses caused by stroke, or
- a combination of these factors.[13]

Initially PSD may be caused by a neurophysiologic imbalance and depression that develops later may be caused by psychological factors.[1]

Biological effects of stroke

During the acute brain infarction there is decreased monoamine synthesis (enzyme inhibition during ischaemia) resulting in decreased 5-HT levels. These have been implicated in altered mood,[14] sleep, and appetite.[15] The use of serotonergic agents has therefore been suggested to augment stroke recovery.[16] Stroke patients with depression may also have:

- altered cortical receptor activity[17]
- altered concentration of cerebrospinal fluid neurotransmitter metabolites[18]
- electrophysiological abnormalities[19], and
- decreased cerebral blood flow.[20]

Lesions in the left frontal lobe or basal ganglia are reported to cause more PSD than other brain areas.[21] There is no clear association between the volume of the lesion[22] and cortical/subcortical atrophy[23] with the type or severity of depressive symptoms.

Psychosocial effects of stroke

Despite successful rehabilitation for mobility and self care skills, social reintegration and life satisfaction remain an issue for many stroke patients. Stroke is associated with significant psychosocial difficulties that can impact on the development of depression including:

- grief at loss of function, loss of independence or loss of employment
- financial difficulties
- social isolation
- poor self esteem, and
- relationship or sexual difficulties.

Early factors predictive of PSD include:

- aphasia at 3–12 months poststroke
- older age
- limited social supports
- living alone, and
- a previous history of psychiatric problems.[24]

There is conflicting evidence regarding gender and PSD. In one study, women were found to be more depressed after stroke,[25] but another found otherwise.[26] When followed up, the male PSD patients had a poorer prognosis compared with women patients.[27] Although older patients were found to be more depressed overall,[2] younger patients tended to be depressed in the acute early stages following stroke.[28]

Diagnosis

The diagnosis of PSD may be difficult due to deficits in limited patient self report, impaired cognition, poor insight and aphasia. Features such as anosognosia, fatigue, emotionalism, apathy and intellectual decline can also limit a patient's ability to express themself. This may result in discrepancy between self rated depression and observer rated depression.

Distinguishing between cognitive decline due to stroke and poor cognitive function secondary to depression can be difficult in this group of patients. Stroke is followed by decline in cognitive function that appears to be independent of the presence of depression. However, depressed patients perform poorly in areas of cognition[2] and have poorer verbal abilities.[29] Apathy is also seen in stroke patients and may coexist with emotional and cognitive poststroke disturbances.

Symptoms of depression may be due to an underlying medical condition or cognitive deficits rather than an underlying mood disorder. Differential diagnoses include organic brain syndrome, side effects of medications, sepsis and hypothyroidism.

A number of standardised tests are used to screen patients for depression or to monitor their response to treatment, but these should not be used for diagnosis in isolation (*Table 1*). The Hospital Anxiety and Depression Scale (HADS) and the General

Table 1. Screening tools for depression and recommendations[33]

Hospital Anxiety and Depression Scale

Seven items each measuring depression and anxiety. Originally used in 100 patients 16–65 years of age in outpatient settings. It has high reliability and validity, and only 1% false positives and negatives. Correlations with psychiatric ratings were 0.79 for depression and 0.54 for anxiety. Recommended by the British Stroke Research group

Beck Depression Inventory

Recommended scale in integrated pathway of PSD. Twenty-one item self report instrument with low reliance on somatic items. Respondents are asked how they have been feeling over the past 2 weeks. Each item is rated on a 4 point scale, ranging from 0–3. A cut off of 14/15 is indicative of depression. It has good reliability and validity and has a positive predictive value of 0.54 and negative predictive value of 0.99, and few false negatives. It has high internal consistency. Factor analysis can discriminate between those with cognitive and noncognitive dimensions. The advantage in stroke patients is the low demand on memory

General Health Questionnaire-28 (GHQ-28)

Uses 28 items for somatic symptoms, anxiety, insomnia, social dysfunction and severe depression. It is widely used, acceptable for the elderly patients, sensitive to the effects of intervention and recommended by the British Stroke Research group. It has good reliability and validity. Coefficient correlations between GHQ-28 and interview measures are 0.67 and 0.83 with a median of 0.76. The sensitivity ranged from 0.44–1.0 and specificity ranged from 0.7–0.93

Depression, Anxiety, Stress Scale (DASS)

This dimensional scale contains 42 questions, with 14 items subdivided into 2–5 items. It assesses self reports on depression, hopelessness, anxiety and stress levels. DASS has high internal consistency and yields meaningful discriminations in a variety of settings

Other depression screening tools include: Hospital Stroke Aphasic Depression Questionnaire, Brief Assessment Schedule for Depression Cards, Geriatric Depression Scale, Signs of Depression Scale, and Visual Analogue Mood Scale

Health Questionnaire (GHQ) are the best validated scales in patients without communication problems and are probably the most useful for screening stroke patients without communication difficulties in the rehabilitation or community setting. Those who score more than eight for depression on the HADS should be further assessed.

Full history, examination, and patient and family reports are important. The DSM IV criteria for major depression are listed in *Table 2*. The normal exclusion of symptoms due to an underlying medical condition is waived in PSD.[30] Older patients may experience depression without sadness, but they may present with anxiety and a general sense of hopelessness.

Treatment

Treatment for PSD involves a combination of patient education, antidepressant medication, behavioural strategies and psychotherapy.

A number of psychosocial interventions have been studied. These interventions included controlled trials of counselling, occupational therapy for leisure, and social work support; they have not demonstrated any clear benefit on mood state.[31] Cognitive behaviour therapy (CBT) results are encouraging but these treatments are not always accessible. Language limitations and cognition issues in stroke patients can interfere with psychotherapy interventions including CBT. In the weeks and months after the stroke, the residual cognitive and behavioural deficits are common causes for failure to return to premorbid levels of interpersonal, vocational and recreational functioning. Patients with behavioural problems that are difficult to manage may be treated with success in specialised care where behaviour is monitored, assessed and subjected to positive or negative reinforcers. In such patients, giving feedback and support assists in engaging the patient in rehabilitation treatment and improves compliance.

In practice, antidepressants are often the most pragmatic solution, with more involved psychotherapy reserved for antidepressant resistant cases or those who are unable to tolerate medication. Patients still benefit from pyschoeducation and learning new coping skills as detailed below.

Table 2. DSM IV criteria for major depression

The American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders (4th edn) (DSM-IV) list the following criteria for diagnosis of depression:

Five or more of the following symptoms persisting over a 2 week period causing clinically important distress or impairing work, social or personal functioning (with depressed mood or decreased interest or pleasure as one of the five):

- depressed mood most of the day, occurring most days (subjective or observed)
- markedly diminished interest or pleasure most of the day, nearly every day
- significant weight or appetite change
- insomnia or hypersomnia
- psychomotor agitation or retardation (observable by others)
- fatigue or loss of energy
- feelings of worthlessness or inappropriate guilt
- diminished ability to concentrate or make decisions
- recurring thoughts of death or suicide plans

Note: the normal exclusion of symptoms due to an underlying medical condition is waived in PSD[30]

Pharmacological management

Selective serotonin reuptake inhibitors (SSRIs) are the most commonly used drugs for PSD. Tricyclic antidepressants (TCAs) are less commonly used and inhibit the uptake of both serotonin and noradrenaline. There is no high level evidence to suggest SSRIs are superior to TCAs. In a double blind, placebo controlled study, fluoxetine was compared with nortriptyline – the response was adequate with both drugs, but only the nortriptyline group improved functional independence.[32] However, SSRIs have a safer side effect profile, a relatively quick onset of action of 7–10 days, and good anxiolytic effects making them first line antidepressant, especially in the elderly.

Within a class, there is no hard data to recommend one drug over another for PSD, however, a recent review[32] suggested an optimum regimen for sertraline with a starting dose of 50 mg per day, which can be increased to 100 mg per day in 2 weeks. There is little benefit from a higher dosage, and if there is no benefit after 4–6 weeks on 100 mg, then it should be discontinued. It should be continued for at least 6 months and slowly weaned but can continue if symptoms recur. Sertraline is also effective in the treatment of emotionalism following stroke. Electroconvulsive therapy has been used to treat refractory severe PSD but is limited due to side effects.

Nonpharmacological treatment

Psychosocial management strategies include:

- education about depression to the patient, carer and family
- managing sleep-wake cycles
- improving nutrition
- anxiety management techniques
- problem solving techniques
- behavioural programs to target specific issues (eg. apathy, aggression)
- cognitive remediation (*Table 1*)
- compensatory rehabilitation strategies for specific physical problems (eg. spas-

ticity, pain)
- addressing relationship difficulties (eg. altered family role, intimacy issues, self image)
- provision of aids and adaptive equipment to optimise physical fitness and maximise function and community participation
- daily activity planning and reduction of social isolation
- improving patient and carer support, and
- fatigue management and energy conservation strategies.

Conclusion

General practitioners play a pivotal role in the ongoing care and management of patients recovering from stroke – and their families. Early screening, monitoring and treatment for depression after stroke can facilitate a patient's functional recovery, social reintegration and improve their quality of life.

Conflict of interest: none declared.

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