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A ONE-YEAR FOLLOW-UP STUDY INTO THE COURSE OF DEPRESSION AFTER STROKE

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Abstract: *Background :* Stroke patients commonly suffer from neuropsychiatric disorders, such as depression, that negatively influence stroke outcome. Diagnosis, treatment and prevention of post-stroke psychiatric disorders including depression are under debate. *Objective:* To study the course of depression after stroke. *Methods:* One hundred and ninety first-ever stroke patients were screened for depressive symptoms at 1, 3, 6, 9, and 12 months after stroke. Diagnosis of depression was made according to the DSM-IV criteria of major and minor depression. *Results:* Follow-up was completed in 138 patients. The cumulative incidence of post-stroke depression (PSD) in 1 year was 36.2%. One month after stroke the prevalence of PSD was 18.8%. Thirty percent of patients who were depressed in the first three months did not reach cut-off levels on depression screening instruments at the following assessments. In 44% of these patients symptoms recurred. Recurrent cases were older than patients with limited disease. In 40% of PSD patients depression persisted for at least two consecutive following follow-up visits. Persistent cases were more disabled and suffered more often from major depression. *Conclusion:* Half of PSD patients become depressed within the first month after stroke. Although most patients recover, a clinician has to be aware that symptoms can recur especially in older patients and that in patients with major depression symptoms may be persistent. In these patients treatment should be considered, whereas in patients with limited disease an observational approach may suffice.

Key words: Depression, stroke.

Introduction

Not only does stroke cause physical inability but it also has essential emotional and cognitive consequences which can be severely debilitating. Depression is among the most commonly reported neuropsychiatric complications of stroke. In the long term, depressive symptoms are highly prevalent in stroke survivors (1-4). Depression has a negative impact on stroke mortality, recovery, and quality of life in stroke survivors (5).

Identification of patients at risk focuses on bio-psychosocial risk factors such as female sex, living alone, older age, level of handicap, and a neurotic personality, but study findings are inconsistent (6-10). The anatomical influence of lesion size or location as a risk factor is controversial (11, 12). For the optimal identification and treatment of PSD patients knowledge about the course of depressive symptoms after stroke is of necessity. How PSD evolves over time remains to be explored more extensively. Despite much research into PSD only a few studies focus on the natural course and reported results are inconsistent. Most of these studies comprise long follow-up periods with long intervals between assessments. Some studies state that PSD most frequently develops shortly after stroke but that it also can appear months or years after the event. Patients who develop PSD shortly after the event may differ in characteristics and symptomatology from patients in whom PSD develops later. Tateno et al. state that early symptoms of PSD are associated with the direct consequences of stroke, whereas late symptoms are more related to psycho-social functioning.(13)] Some studies demonstrate that most cases of PSD recover spontaneously within months without any treatment whereas other studies report that depression also may take a chronic course (6, 8, 14, 15). Knowledge of risk factors enables a clinician to identify patients who are at risk of developing a more chronic course of PSD and who would benefit from intervention. However, for efficient management of PSD, knowledge about the natural course of symptoms is warranted, especially since the effects of treatment of PSD with anti-depressive drugs and non-pharmacological interventions are debatable (16, 17). The aim of this study is to describe the natural course of depression after stroke in first-ever stroke patients. We hypothesized that patients in whom depression takes a chronic course are more severely affected and have more risk factors for developing PSD than patients in whom PSD recovers spontaneously in the short term. Furthermore, we were interested whether patients with early PSD differed from patients who develop PSD later in the course of stroke recovery.

Patients

In this study 190 consecutive patients with a first-ever hemispheric cerebral infarction and without depression in the weeks prior to the infarction were included. The patients were recruited from the Emergency Department of the University Hospital of Maastricht, The Netherlands. Patients and study design have been described previously (18). In short, first-ever ischaemic stroke patients were examined every three months after stroke in a prospective longitudinal study design during

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the first year after stroke.

Stroke was diagnosed by a neurologist according to the WHO criteria (19). Computed tomography (CT) differentiated between hemispheric infarction and other causes and types of stroke. If patients had another type of stroke (e.g. recurrent stroke, brainstem stroke or haemorrhage) they were not included in order to increase homogeneity of groups. Patient data were entered into a prospective stroke registry, described elsewhere (20).

Exclusion criteria were any major psychiatric disorder other than a depressive disorder (e.g., schizophrenia or a current psychotic episode), the onset of a current depressive episode before stroke, co-morbid intracerebral disease (e.g., a brain tumour or Parkinson's disease), and inability to understand the informed consent procedure (e.g., because of severe aphasia or cognitive dysfunction). Furthermore,in order to obtain a homogenous population patients with other stroke types such as haemorrhage, recurrent stroke, or brainstem infarction were excluded from the study. All participants gave written informed consent and the study was approved by the local Medical Ethics committee.

Assessments

One month follow-up after stroke all patients had a psychiatric assessment by a well-trained research physician (IA) using the Structured Clinical Interview of DSM-IV (SCID-I-R), to diagnose them as non-depressed, major or minor depressed. To determine severity of depression and symptoms, the 17-item Hamilton Depression Rating Scale (Ham-D) was administered (21). Furthermore, patients were asked to fill out three depression self-rating scales, i.e. the Beck Depression Inventory (BDI), (22) the 90-item Symptom Checklist (SCL-90), (23) and the Hospital Anxiety and Depression Scale (HADS-A and HADS-D) (24). Cut-off values for these rating scales and their sensitivities have been defined previously (25). For the BDI the cut-off value was 9/10, for the HADS 7/8 for both subscales, and for the SCL-90 the cut-off was 22/23 for men and 22/28 for women.

At 3, 6, 9, and 12 months after stroke, patients were screened for depressive symptoms using the previously mentioned self-rating scales. Patients who exceeded the cut-off values on one of these scales were re-interviewed by the physician using the SCID-I-R and Ham-D for diagnosis.

Neuroticism was evaluated 1 month after stroke using the Dutch translation of the Neo Five Factor Inventory (NEO-FFI) (26, 27). This is a self-report questionnaire consisting of 60 statements comprising the five main domains of personality: neuroticism, extraversion, openness to new experiences, agreeableness, and conscientiousness. Each statement is rated on a 5-point scale ranging from 'strongly disagree' to 'strongly agree', resulting in a total dimension score of 12-60. Neuroticism is defined as the tendency to experience negative emotions and to have poor coping skills and it is highly associated with psychopathology, including depression (28).

Disability and handicap were measured at baseline using the Barthel Index and Rankin Score. Global cognitive functioning and aphasia were determined at baseline by the MMSE and FAST.

Patients who were excluded from the study or who refused participation were not assessed.

Statistical analyses

For demographic variables descriptive analyses were administered. Survival techniques were used to study the cumulative incidence.

Cases with persistent depression were defined as having a diagnosis of depression during at least 2 consecutive follow-up visits and who reported symptoms of depression between these visits. Cases were defined as transient if they were depressed at one follow-up. Recurrent cases were diagnosed with depression during more than one visit, with sub-threshold symptom levels in between.

Early onset depression was defined as a diagnosis of depression at 1 month. Late onset depression was defined as a depression that debuted at subsequent follow-up visits after 3, 6, 9, or 12 months.

To compare descriptive characteristics Student's t tests were applied in the case of continuous variables and Chi-square tests were used in the case of dichotomous variables. For non-parametrically distributed characteristics Mann-Whitney U tests were applied.

For the comparison of group variances between non-depressed, persistent, transient, and recurrent cases one-way ANOVA was applied with post-hoc Bonferroni and Scheffé correction in the case of significance in between-group variances. In the case of not -normally distributed characteristics, Kruskall-Wallis tests were applied. In the case of dichotomous variables Chi-square tests were applied.

To study the relationships between cognitive and functional deficits and depression, correlation coefficients were estimated for the MMSE, FAST, Barthel Index, and Rankin score.

All statistical analyses were performed using the Statistical Package for Social Sciences version 15 (SPSS15).

Results

Patients were selected from 444 consecutive patients who visited the emergency department with a first-ever hemispheral infarction. One hundred and ninety-three patients were excluded (37 died within 1 month after stroke, 54 patients had severe aphasia or other cognitive deficits, 38 had severe somatic co-morbidity, 12 had combined somatic and cognitive co-morbidity, 12 patients had a current psychiatric disorder including depression, 40 were excluded because of miscellaneous reasons such as living too far from the hospital, not native Dutch speaking or untraceability), and 61 patients refused participation. Of the 190 patients who entered the study

at baseline, 52 were lost to follow-up during the 1 year follow-up period (11 died, 27 refused further participation, 14 were incapable of further participation or untraceable). Table 1 shows the population characteristics. Patients lost to follow-up were older, lower educated, had lower MMSE scores and were more handicapped. There was no difference in prevalence of depression.

Table 1
Demographic characteristics

	Total cohort (N=190)	Patients with complete follow-up (N=138)	Drop-outs (N=52)
Sex (F)	46.8%	48.1%	55.1%
Age mean (SD)	68.6 (11.7)	66.1 (11.4)	75.1 (9.7)*
Education (high)	32.0%	37.7%	17%*
MMSE median (range)	26.0 (16-30)	27.0 (19-30)	25.0 (16-30) *
FAST median (range)	27.0 (9-30)	26.0 (4.1)	23.8 (4.8)*
Barthel median (range)	20.0 (0-20)	18.0 (0-20)	20.0 (1-20)*
Rankin mean (SD)	2.4 (1.2)	2.2	2.9*
Hamilton score mean (SD)	9.5 (6.5)	8.7 (6.4)	11.5 (6.4)
PSD	35.8%	36.2%	36.2%
Neuroticism mean (SD)	30.1 (7.3)	30.0 (6.8)	30.4 (9.1)
Cortical infarction	44.9%	44.9%	45.1%
Left-sided lesion	46.8%	44.9%	51.9%
History of depression	21.5%	21.3%	22.0%
Family history of depression	20.1%	19.4%	22.2%

MMSE: Mini Mental State Examination (minimum score: 0, maximum score: 30); FAST: Frenchay Aphasia Screening Test (minimum score: 0, maximum score: 30); Barthel Index (completely dependent in Activities of Daily Living: 20, no disabilities: 0); Rankin (no handicaps: 0, bedridden: 5). *p<0.05: for comparisons between patients who completed the follow-up and drop-outs.

Table 2 shows the cumulative incidence and prevalence rates of PSD at each assessment during the follow-up period of 1 year.

In total, 50 patients were diagnosed with depression during the follow-up (1 year cumulative incidence of 36.2%). Thirty patients were diagnosed suffering from major depression and 20 patients were diagnosed suffering from minor depression.

In 52% of PSD patients depression was already apparent after 1 month. In 14 patients (28%) depression was recurrent.

Table 3 shows the characteristics of non-depressed patients and patients with persistent, transient and recurrent depression.

Persistently depressed patients had higher Rankin scores (Kruskall Wallis X^2=11.58, df=3, p=0.009). Recurrent cases were older than transient cases and persistent cases (73.4 , 64.1, and 63.1 years respectively), albeit not significantly (F=2.49, df=3, p=0.06). Non-depressed patients more often were highly educated (X^2 = 8.00, df=3, p=0.04). Non-depressed patients had lower neuroticism scores than recurrent cases (F=4.5, df=3, p=0.004). There were no differences in severity of depression, location of the lesion, cognition, and sex. Recurrent cases were prescribed antidepressants more often albeit not significantly. There was no significant correlation between cognitive and functional deficits and persistent, transient or recurrent depression. Four patients received an intravenous thrombolytic treatment. These patients were distributed equally among the non-depressed, incidentally, persistently and recurrently depressed patient groups.

Table 4 shows the characteristics of patients diagnosed as depressed at the first follow-up visit or later.

Patients suffering from late-onset PSD were more highly educated (X^2= 3.82, df=1, p=0.05). They more often were men albeit not significantly. Early and late PSD patients did not differ in level of neuroticism.

Discussion

In this study of PSD in first-ever stroke patients during a one year prospective follow-up, we examined the persistency of PSD. The cumulative incidence of PSD was 36.2%. Half of these patients were diagnosed as depressed within 1 month after stroke and incidence rates declined after the acute phase after stroke. The prevalence rates at subsequent follow-up times initially tended to decrease and at 6 months to increase but all in all remained comparable to baseline throughout the follow-up period. In approximately 30% of patients who were depressed within the first three months the duration of disease was restricted to 1 follow-up visit. In 44% of these patients,

Table 2
Cumulative incidence of PSD

Month	Number at risk	Incident cases (major / minor depression)	Cumulative incidence of PSD	Prevalence of PSD	Presence at one assessment N	Persistence at 2 following assessments N (major/minor depression)	Persistence at 3 or more following assessments N (major/minor depression)	Recurrent cases* N (major / minor depression)
1	138	26 (17/9)	18.8%	18.8%	8 (4/4)	2 (1/1)	5 (5/0)	11 (7/4)
3	112	6 (2/4)	23.1%	10.9%	1 (0/1)	1 (0/1)	1 (1/0)	3 (1/2)
6	106	5 (3/2)	26.7%	13.0%	1 (0/1)	2 (1/1)	2 (2/0)	0
9	101	6 (4/2)	31.0%	15.2%	5 (3/2)	1 (1/0)	n.a.	n.a.
12	95	7 (4/3)	36.2%	14.6%	7 (4/3)	n.a.	n.a.	n.a.

*Recurrent cases were diagnosed depressed at two or more assessments during the follow-up period with episodes with no or sub-threshold symptoms in between. n.a. = non-applicable

Table 3
Demographic variables of persistent, transient and recurrent cases of PSD

	Non-depressed (N=90)	Persistent PSD (N=14)	Transient PSD (N=22)	Recurrent (N=14)
Sex (F)	62.2%	30.8%	47.6%	42.9%
Age (SD)	65.8 (11.2)	63.1 (11.4)	64.1 (12.9)	73.4%
Education (high)	44.4%*	7.7%	28.6%	28.6%
MMSE (median, range)	27.0 (17-30)	27.0 (24-30)	27.0 (10-30)	25.5 (23-29)
Rankin (mean, SD)	2,0 (1,0)*	3.0 (1.0)	2.1 (1.2)	2.6 (0.8)
Barthel (median, range)	20 (5-20)	19 (0-20)	20 (0-20)	18 (5-20)
Hamilton score (mean, SD)	6.1 (3.9)*	16.1 (8.0)	11.0 (7.3)	13.9 (6.3)
Major depression	n.a.	76.9%	52.4%	57.1%
Neuroticism mean (SD)	28.5 (6.4)*	32.1 (10.3)	31.9 (6.3)	34.6 (5.0)
Cortical infarction	46.6%	38.5%	42.9%	42.9%
Left sided lesion	46.6%	46.2%	33.3%	50%
Antidepressants	4.0%	40%	36.8%	61.5%
History of depression	15.6%	33.3%	25.0%	42.9%
Family history of depression	14.9%	20%	35.7%	30.8%

Comparison of group variances between non-depressed, persistent, transient and recurrent depression using one-way ANOVA and Kruskall-Wallis in case of either normally distributed or non-normally distributed continuous variables. For dichotomous variables Chi-square tests are attributed. * p<0.05

PSD recurred within the first year after stroke. Patients in whom disease recurred were older than patients in whom disease was either restricted to 1 visit or persisted over a longer period. Patients with persistent symptoms of PSD more often met criteria for an episode of major depression somewhere in their disease course. This is in accordance with the findings of Morris et al.(29). Moreover, these patients were more severely handicapped than patients with a transient disease episode. In our study, non-depressed patients were more often highly educated (44%), whereas 7.7% of patients in whom disease persisted were highly educated. Also, patients with a late onset of PSD were more often highly educated compared to patients with an early onset of PSD.

Table 4
Demographic variables of early and late PSD patients

	Early (N=26)	Late (N=24)
Sex (v)	51.9%	30.4%
Age (mean, SD)	66.2 (12.2)	66.3 (11.8)
Education (high)	14.8%	39.1%*
MMSE (median, range)	26.0 (19-29)	27.0 (18-30)
Barthel (median, range)	18.0 (0-20)	20 (0-20)
Rankin (mean, SD)	2.74 (1.1)	2.13 (1.1)
Hamilton score (mean, SD)	16.7 (7.0)	8.8 (5.4)*
Major depression	66.7%	52.2%
Neuroticism mean (SD)	33.2 (6.3)	32.6 (7.4)
Cortical infarction	51.9%	34.8%
Left sided lesion	40.7%	47.8%
Antidepressants	40.0%	41.7%
History of depression	56.5%	28.6%
Family history of depression	37.0%	13.6%

*p<0.05

In the literature, PSD is often studied in longitudinal prevalence studies of several years with long time periods between follow-up visits (30). A strong point of our study is that patients were examined every 3 months, providing insight into incident depressive cases and the dynamic character of depression in the course of PSD during the first year. In a longitudinal study performed by Astrom et al., in 80 stroke patients followed over 3 years the investigators found a prevalence of 25% at the acute stage, and that 60% of these patients recovered within 1 year (6). Berg et al. found that 54% of 100 stroke patients were at least mildly depressed during an 18 month follow-up and approximately half of the patients with depressive symptoms within the first 2 months also had depressive symptoms after 12 and 18 months (8). In our study we found that most patients diagnosed with PSD in the acute stage recovered. However, in our study patients were screened for the DSM-IV diagnosis of minor and major depression. Thus our study comprised only the more severely affected patients and patients with mild depressive symptoms were excluded. It is conceivable that the incidence of patients with persistent depressive symptoms is actually higher. Two studies reported that patients with less severe depression suffered longer (30, 31). Moreover, in our study PSD recurred in 28% of patients. Patients with recurrence of disease were older. There was no difference in frequency of antidepressive drug treatment between patients who recovered or in whom disease persisted or recurred.

Thus far the causality of PSD has not been fully elucidated. Although the literature reports no association between the vulnerability for PSD and level of education we found that PSD patients were less often highly educated than non-depressed stroke patients (9). Furthermore patients suffering from late onset PSD were often more highly educated than patients suffering from early onset PSD. However, due to small numbers, a conclusion should be taken with caution. Bio-psychosocial and social factors all account for the occurrence of PSD at all stages in stroke rehabilitation (7, 9, 32, 33). Some

authors report a time-dependent causality (13). In the early disease stage especially the course of symptoms is dynamic. Whereas most recovery is seen in the first weeks after stroke this is also the period in which the incidence of PSD is highest (34, 35). Tateno et al reported a time-related change in symptomatology and concluded that early depressive symptoms are related to biological causality provoked by the brain damage itself, whereas in late onset symptoms social and psychological markers may attribute more importantly to the cause of depression (13). Berg et al. found that early symptoms were associated with older age and late onset symptoms were associated with stroke severity and male sex.[8] Astrom found that in the early stage, dependence in activities of daily living predicted PSD, whereas in the long term having few social contacts was more predictive (6).

Our study has some limitations. First of all since we had a limited follow-up period of one year we cannot be specific about the persistency of PSD in patients who were diagnosed depressed at follow-up visits later than the first 3 months. Secondly, we used screening instruments to identify patients at risk of suffering from PSD. Although our screening method was shown to be highly sensitive, some patients with (major or minor) depression using the SCID-I on the previous measurement, may falsely have scored below threshold on the next screening since patients were dichotomized depressed or non-depressed (25). It is conceivable that analysis of the severity of depression on a continuous scale would have increased the sensitivity of our methods. Small numbers limited the detection of significant relationships between the diagnosis of depression and demographic variables, especially between subgroups. A study into the causality and risk factors for depression, however, would require a different study design and was beyond the scope of this study. Additionally, the prescription of antidepressant agents may have influenced the conclusions drawn about the persistency and recovery of PSD. However, there were no differences in the number of patients who were prescribed antidepressants in the disease course between persistent and recovered PSD patients.

Furthermore, since we excluded patients with most severe cognitive deficits and severe aphasia or somatic morbidity we cannot rule out bias. Since excluded patients were not assessed we cannot determine how this affected our results. However, it is conceivable that patients with more handicap and cognitive deficits more often suffer from depression and that the incidence of PSD in our study is actually higher. The informed consent warranted that patients would understand the procedure. This generally applies to studies warranting a long term cooperation. In a previous study we reported that patients with lower MMSE scores experienced more problems fulfilling the rating scales and had higher drop-out rates (25). The course of depression in such patients may differ from those studied. In literature these patients often are excluded due to the lack of sufficient diagnostic measures. Nowadays, there are screening measurements to define the severity of depression in aphasic patients (36). More insight into PSD in these patients is of great necessity. It is important to know how cognitive and functional deficits influence the course of PSD. In our study patients who did not exceed the cut-off scores of the attributed screening measures for depression were not assessed. Therefore, we cannot determine how these deficits evolve over time and how they influenced our results over time. However, there were no significant correlations between baseline cognitive or functional deficits and the subgroups of PSD in our study. Finally, we had a substantial amount of drop-outs. These patients however did not differ in frequency or severity of depressive symptoms so it is not likely that this affected our results.

A better understanding of the spontaneous course of PSD may assist in deciding if and when a patient should be treated with antidepressants or non-pharmacological interventions.

To date there is little evidence to support routine use of antidepressants in the prevention and treatment of PSD, only a few studies report a beneficial effect of antidepressants on depressive symptoms and stroke outcome (17, 37, 38).

In our study, we found that PSD is most frequent in the acute phase after stroke. Prevalence rates remained more or less stable throughout the first year after stroke. Most patients recovered spontaneously within 3 months. Since a treatment effect of antidepressants is not obvious until weeks after initiation, drug treatment will not be attributive to recovery. So, in most cases it will be acceptable to wait and see. However, in a substantial number of patients depression recurred. These patients were older. Furthermore, chronic diseased patients more often suffered from major depression. These patients may benefit from antidepressants and non-pharmaceutical interventions if symptoms persist for 3 months. More research into the treatment of PSD is warranted.

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