# Cognitive functions after hemorrhagic stroke: follow-up study

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**Objectives**

There are scarce data on long-term cognitive outcomes after first-ever hemorrhagic stroke.

**Aim**

This study was carried out to determine the frequency of cognitive impairment, no dementia after an intracerebral hemorrhagic stroke and to study their evolution toward dementia (transitions in cognitive state) during a 2-year follow-up.

**Methods**

Thirty-five patients with first-ever hemorrhagic strokes and in an age-matched and sex-matched comparison group (nonstroke, $n=20$) were followed up for 2 years by three serial assessments. Stroke patients at 3, 12, 24 months after discharge were evaluated together with nonstrokes, by an extensive neuropsychological battery and clinical psychiatric interview based on Diagnostic and Statistical Manual of Mental Disease, 4th edition TR criteria. Rates of cognitive change were compared using repeated-measures analyses. Factors associated with incident dementia and cognitive impairment, no dementia at 2 years were determined by multinomial logistic regression.

**Results**

The majority of patients (71.4%) were cognitively stable. Fewer cases improved (11.5%) and 71.1% of the stroke cases worsened at the end of the 24-month follow-up. There was impaired cognitive function in nearly all cognitive domains of stroke cases compared with nonstroke cases. Overall, stroke cases showed a statistically significant decline in mini-mental state examination (MMSE), spatial ability, and in the executive function domain; however, there was no improvement in attention nonverbal memory domains. Age at stroke onset was independently associated with cognitive impairment at the 2-year follow-up $P=0.03$.

**Conclusion**

Cognitive evolution 2 years after hemorrhagic stroke is different among patients, but a substantial number of patients remain stable. Additional studies are required to reliably identify individuals at risk for cognitive decline for whom pharmacologic or other therapeutic interventions would be more suitable and who will probably spontaneously improve. Classification of stroke by etiology may be more useful to determine patients at a higher risk of stroke and for its prevention.

**Keywords:**

longitudinal study, neuropsychology, stroke, vascular cognitive impairment, vascular dementia

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## Introduction

Stroke remains a major healthcare problem. Approximately 795 000 people in the United States have stroke each year. About 610 000 are first events, and 6.4 million Americans are stroke survivors [1]. Stroke is also a leading cause of functional impairments, with 20% of survivors requiring institutional care after 3 months and 15–30% being permanently disabled. Effective prevention remains the best approach for reducing the burden of stroke. Primary prevention is particularly important because greater than 77% of strokes are first events [2]. Cognitive decline after stroke is more common than stroke recurrence. Stroke doubles the risk of dementia and is a major contributor to vascular cognitive impairment (VCI) and vascular dementia [3].

There are few prospective data on the long-term cognitive changes and predictors of incident dementia after a first stroke [4]. Stroke is not only a preventable but also a treatable disease. However, as the treatment is only safe and licensed within the first 3 h after stroke, advancements neurologic services are needed, with an emphasis on immediate care [5].

Fewer studies have focused on the behavioral and cognitive manifestations after stroke and these disorders are often overlooked in clinical practice. Frequently occurring symptoms are personality changes [6] and

neuropsychiatric disorders, such as poststroke depression, anxiety disorders, or apathy [7]. In addition, neuropsychological disorders, such as amnesia, executive dysfunction, or unilateral neglect, are common clinical manifestations after stroke and may be the single or dominant presenting features [8].

Most studies on the relation between stroke and cognitive impairment have reported on vascular dementia or poststroke dementia in general. The concept of vascular dementia has recently been discarded by most researchers because of the inconsistent criteria used to define vascular dementia [9] and because the level of cognitive impairment required for a diagnosis of dementia does not allow early identification of patients with less severe but seriously invalidating cognitive disturbances [10]. Subsequently, a range of conceptsthat include milder forms of cognitive impairment have been developed over the past few years, such as VCI [11], 'mild cognitive impairment' [12], or 'cognitive impairment, no dementia' (CIND) [13]. Although these concepts are preferable to that of vascular dementia, they will attempt to capture a very diverse phenomenon under one header, resulting in poor prognostic value and confusion in the literature. Moreover, these concepts do not provide information on the nature of the underlying cognitive disorder and the specific disability that might arise from these deficits. Nevertheless, early diagnosis of specific cognitive deficits such as amnesia or executive dysfunction could be very important to determine an appropriate discharge destination and in particular to facilitate rehabilitation. Also, interventions aimed at restoring specific cognitive functions can be initiated at an earlier stage, as studies have shown that the brain displays a heightened sensitivity to rehabilitation early after the stroke as compared with later stages [14,15].

Although the brain is capable of reorganization and significant cognitive recovery may occur in the first months after stroke, many patients do not show improvement at all or even show deterioration in the long term [16], resulting in poststroke dementia [17].

Clinical criteria for VCI as well as vascular CIND are still lacking, and major discussions are ongoing in this field. Detection of cognitive impairment has two difficulties: a neuropsychological battery fitted to the VCI profile is yet to be established and limits from normal cognition are still undefined [18]. Although clinical criteria are still undefined, considerable work has been carried out to determine the frequency, characteristics, and evolution of VCI and V-CIND. Importantly, there is a growing interest in V-CIND, because patients with only mild cognitive deficits also have significant disability, may be at an increased risk of cognitive deterioration, and have more opportunities for treatment and prevention [19]. There are few prospective data from population-based studies on the long-term cognitive changes and predictors of incident dementia after a first stroke. It is also uncertain whether early poststroke cognitive status is associated with a high risk of future incident dementia. The aim of this study was to determine the frequency of CIND after intracerebral hemorrhagic stroke and to study their evolution toward dementia (transitions in cognitive state) during a 2-year follow-up.

## Patients and methods

This study was carried out in Mansoura University Hospitals from February 2007 to September 2009.

The study included 35 patients from the neurology department. All patients had a diagnosis of first-ever incident hemorrhagic stroke (with no history of prior stroke). The patient group comprised 28 men and seven women who completed the assessment tools during the scheduled follow-up visits. They were included in the study on the basis of certain inclusion and exclusion criteria. Inclusion criteria included fully conscious patients. The exclusion criteria were as follows: (a) patients above the age of 75 years, (b) other areas of lesions such as subarachnoid hemorrhage, cerebral infarction, or tumor, (c) major neurological diseases for example, Alzheimer's disease, other dementias, Parkinson's disease, or epilepsy, (d) history of any psychiatric diagnosis, (e) history of psychoactive substance use disorders or current use of sedating medications, (f) medical conditions such as hepatic, renal, or pulmonary disease, and (g) patients with dysphasia or inadequate vision or hearing.

The control group included 20 participants and were selected to match the patient group in terms of age, sex, and level of education. None of the control participants had a history suggestive of any psychiatric, neurological diseases, head injury, substance use, history of previous stroke, transient ischemia attack, or medical conditions such as hepatic, renal, or pulmonary diseases.

### Methods

Clinical assessment included neurological examination and collection of data on demographic distribution, hypertension diabetes mellitus (DM), myocardial infarction, and smoking. Moreover, computed tomography or MRI were carried out for all participants to confirm the diagnoses. The psychiatric assessment included a clinical psychiatric interview along with a semistructured psychiatric interview on the basis of Diagnostic and Statistical Manual of Mental Disease, 4th edition TR criteria [20] for the diagnosis of dementia.

A neuropsychological battery was designed to cover the main cognitive functions and to detect early or minimal cognitive impairment with tests applicable to low-educated patients with eventual sensorimotor deficits. The battery included the MMSE scale [21], which is a 5-min screening test that is designed to evaluate basic mental functions in a number of different areas. Selected subtests from the Wechsler memory scale were used to assess memory [22]: the logical memory A–B test to assess verbal memory, digit forward, and digit backward to assess attention and short-term verbal memory, visual reproduction I, II to assess nonverbal memory and word association to assess executive functioning, which includes verbal attention (fluency), working memory, abstract

reasoning, and planning. Moreover, the trail making test was used in its two forms (trail A and B) [23] to assess executive functions. The Wechsler adult intelligence scale [24] was used to detect visuo-spatial function (performance part): The test comprises 11 subtests made up of six verbal subtests and five performance subtests. The Bender–Gestalt test (BG) was used also to assess short-term visual memory and visuo-spatial/visual construction functions.

Tests were grouped into six cognitive domains: (a) orientation (temporal and spatial orientation from MMSE), (b) executive ability (trail making A, B, and word association), (c) verbal memory [logical memory A, B, and BG (recall)], (d) nonverbal memory visual reproduction I and II, (e) spatial ability (Wechsler adult intelligence and BG), and (f) attention (digit forward and backward).

The clinical dementia rating (CDR) scale [25] was used to characterize and track the level of impairment/dementia (0 = normal, 0.5 = cognitive decline without dementia, 1 = mild dementia, 2 = moderate dementia, and 3 = severe dementia). An overall CDR scale score may be calculated using an algorithm.

### Diagnosis of dementia and cognitive impairment

*Diagnostic criteria for dementia*

Patients were diagnosed as having dementia or not having dementia according to the Diagnostic and Statistical Manual of Mental disorders, 4th edition DSM IV TR, on the basis of a progressive decline in memory and at least one other cognitive domain from baseline, the cognitive changes recorded in the CDR scale, and the neuropsychological battery scores. The final diagnosis was always made after a thorough assessment of the clinical and neuropsychological data and controlling for the effects of sensory-motor defects.

*Diagnostic criteria for cognitive impairment, no dementia*

CIND was diagnosed in the absence of dementia when there was cognitive impairment without clinically evident progression from the previous assessment established with the CDR (0.5 = cognitive decline without dementia). Neuropsychological examination defined CIND as failure in at least one cognitive domain. Missing values, attributable to the inability of the patient to perform a test, were considered as failure on the test.

*Follow-up*

After obtaining informed consent, patients were evaluated at admission and reexamined 3, 12, and 24 months after stroke. In these visits, neurological examinations, functional psychiatric assessment, neuropsychological battery, and the diagnosis of dementia were performed. Patients were classified at each follow-up as having dementia, CIND, or being noncognitively impaired. The control group was interviewed at the same intervals to be comparable with stroke cases.

The statistical analysis of data carried out using an excel program for graphs and Statistical Package for Social Science version 17 (SPSS, New York, New York, USA). The data were in the form of mean ± standard deviation for quantitative data and frequency and proportion for qualitative data. The data were analyzed to determine statistically significant differences between groups. The one-way analysis of variance test was used to compare more than two groups, followed by the post-hoc test least significant difference for intergroup comparisons. For quantitative data, the student *t*-test was used to compare between two groups. A paired-sample *t*-test was used to compare one group at different times. The $\chi^2$ test was used for qualitative data. Correlation coefficiency was calculated to detect an association between variables. Multivariate regression analysis was done for significant factors. *P* was considered significant if less than or equal to 0.05 at a confidence interval of 95%.

## Results

Table 1 shows that in the patient group, most of the patients were men, half of them had no education, and one-fourth had more than 10 years of education. Stroke cases were more likely to have a history of hypertension, DM, and smoking (Table 2). The site of hematoma was in subcortical structures in 70.4% of the cases, $n = 25$ (51.4% thalamic and 20% basal ganglia), and lobar structures in 28.6% ($n = 10$) of the cases. Table 3 shows that at baseline, 14 patients were cognitively normal (CDR = 0, 40%) and 21 patients showed a cognitive decline without dementia (CDR = 0.5, 60%). Among the controls, 16 participants were cognitively normal (CDR = 0, 80%) versus four participants, who showed a cognitive decline without dementia (CDR = 0.5, 20%). During the entire study period, CIND was diagnosed in 18 stroke cases (51.4%) and four nonstroke cases (20%). A new incidence

**Table 1 Demographic data of the total sample**

| | Stroke group ($n$=35) Mean ± SD | Nonstroke ($n$=20) Mean ± SD | Significant test | *P* value |
|---|---|---|---|---|
| Age | 58.46 ± 8.037 | 57.85 ± 8.41 | $t$=0.265 | 0.792 |
| Sex | | | | |
| Male, *n* (%) | 28 (80%) | 15 (75%) | $\chi^2$=0.187 | 0.666 |
| Female, *n* (%) | 7 (20%) | 5 (25%) | | |
| Education | | | | |
| Illiterate | 19 (54.3%) | 9 (45%) | $\chi^2$=4.95 | 0.17 |
| ≤6 years | 5 (14.3%) | 3 (15%) | | |
| 7–9 years | 3 (8.6%) | 6 (30%) | | |
| ≥10 years | 8 (22.9%) | 2 (10%) | | |

SD, standard deviation.

**Table 2 Risk factors among the total sample**

| | No. (%) | |
|---|---|---|
| Item | Stroke group | Nonstroke group |
| Hypertension | 15 (42.9) | 6 (3) |
| DM | 7 (20) | 2 (10) |
| Ischemic heart | 3 (8.6) | 2 (10) |
| MI | 1 (2.9) | – |
| Smoking | 16 (45.7) | 4 (20) |

DM, diabetes mellitus; MI, myocardial infarction.

**Table 3 Transitions of cognitive state over 2 years after first-ever stroke**

| | | | Second-year follow-up | | |
|---|---|---|---|---|---|
| Cognition at the 3-month interview | First-year follow-up diagnosis | | Normal | CIND | Dementia |
| Stroke group | | | | | |
| Normal ($n=14$) | Normal | 11 | 8 | – | – |
| | CIND | 3 | – | 5 | – |
| | Dementia | – | – | – | 1 |
| Impaired ($n=21$) | Normal | 2 | 4 | – | – |
| | CIND | 15 | – | 13 | – |
| | Dementia | 4 | – | – | 4 |
| Nonstroke group | | | | | |
| Normal ($n=16$) | Normal | 15 | 14 | – | – |
| | CIND | 1 | – | 2 | – |
| | Dementia | – | – | – | – |
| Impaired ($n=4$) | – | – | – | – | – |
| | Normal | 1 | 1 | – | – |
| | CIND | 3 | – | 2 | – |
| | Dementia | – | – | – | 1 |

CIND, cognitive impairment no dementia.

**Table 4 Comparison of cognitive function between the stroke and the nonstroke group at baseline (after 3 months)**

| | Baseline time stroke | | | |
|---|---|---|---|---|
| Cognitive ability | Stroke group Mean ± SD | Nonstroke group Mean ± SD | *t*-test | Significance |
| MMSE | 22.3 ± 4.2 | 25.6 ± 5.1 | −2.59 | 0.01 |
| Verbal memory | | | | |
| Logical memory A, B | 11 ± 6.69 | 17.6 ± 5.86 | −3.7 | 0.001 |
| BG (recall) | 6.08 ± 4.37 | 14.9 ± 4.38 | 7.19 | 0.000 |
| Nonverbal memory | | | | |
| Visual reproduction | 1.94 ± 1.8 | 3.25 ± 1.1 | −2.89 | 0.006 |
| Visual reproduction II | 2.14 ± 1.98 | 4.2 ± 1.28 | −4.15 | 0.000 |
| Spatial ability | | | | |
| WAIS (performance part) | 72.3 ± 20.60 | 88.8 ± 16.56 | −3.06 | 0.004 |
| BG (copy) | 9.31 ± 6.63 | 16 ± 4.77 | −3.951 | 0.000 |
| Attention | | | | |
| Digit forward | 4.97 ± 1.75 | 6.55 ± 1.57 | −3.32 | 0.002 |
| Digit backward | 2.54 ± 1.00 | 3.85 ± 1.38 | −2.96 | 0.005 |
| Executive function | | | | |
| Trail making A | 237.67 ± 97.01 | 600.59 ± 61.15 | −0.164 | 0.870 |
| Trail making B | 600.89 ± 6.40 | 568.85 ± 11.13 | −0.498 | 0.621 |
| Word association | 3.8 ± 3.81 | 10.05 ± 2.72 | −6.408 | 0.000 |

BG, Bender–Gestalt test; MMSE, mini-mental state examination; SD, standard deviation; WAIS, Wechsler adult intelligence scale.

**Table 5 Cognitive change from baseline to the first-year and second-year follow-up in the stroke group**

| | First year[a] | | | Second year[b] | | |
|---|---|---|---|---|---|---|
| Cognitive ability | Mean ± SD | *t*-test | Significance | Mean ± SD | *t*-test | Significance |
| MMSE | 21.06 ± 5.18 | 2.47 | 0.019 | 20.71 ± 5.09 | 2.42 | 0.02 |
| Verbal memory | | | | | | |
| Logical memory A, B | 10.08 ± 6.87 | 1.96 | 0.05 | 9.74 ± 7.09 | 1.81 | 0.08 |
| BG (recall) | 6.08 ± 4.37 | 0.000 | 1.00 | 5.91 ± 4.41 | 0.322 | 0.75 |
| Nonverbal memory | | | | | | |
| Visual reproduction I | 1.88 ± 1.081 | 0.466 | 0.644 | 2.00 ± 1.93 | −0.291 | 0.77 |
| Visual reproduction II | 2.06 ± 1.86 | 0.517 | 0.609 | 2.2 ± 1.95 | −0.91 | 0.77 |
| Spatial ability | | | | | | |
| WAIS (performance) | 68.57 ± 21.65 | 1.97 | 0.057 | 66.97 ± 20.92 | 2.183 | 0.04 |
| BG (copy) | 9.23 ± 6.39 | 0.131 | 0.896 | 9.05 ± 6.59 | 0.325 | 0.74 |
| Attention | | | | | | |
| Digit forward | 4.68 ± 1.66 | 1.378 | 0.177 | 4.66 ± 1.7 | 0.123 | 0.23 |
| Digit backward | 2.31 ± 1.65 | 1.276 | 0.211 | 2.26 ± 1.74 | 1.51 | 0.14 |
| Executive function | | | | | | |
| Trail making A | 254.55 ± 93.9 | −2.139 | 0.040 | 256.7 ± 94.9 | −2.13 | 0.04 |
| Trail making B | 631.87 ± 25.3 | −1.823 | 0.077 | 643.9 ± 25.7 | −2.19 | 0.03 |
| Word association | 3.74 ± 3.57 | 0.367 | 0.716 | 3.71 ± 3.44 | 0.260 | 0.79 |

BG, Bender–Gestalt test; MMSE, mini-mental state examination; SD, standard deviation.
[a]Comparison of the first year with 3 months (baseline).
[b]Comparison of the second year with 3 months (baseline).

**Table 6 Cognitive change from baseline to the first-year and the second-year follow-up of the nonstroke group**

| | First year | | | Second year | | |
|---|---|---|---|---|---|---|
| Cognitive ability | Mean ± SD | *t*-test | Significance | Mean ± SD | *t*-test | Significance |
| MMSE | 25.75 ± 4.89 | −0.125 | 0.902 | 25 ± 5.803 | 0.656 | 0.519 |
| Verbal memory | | | | | | |
| Logical memory A, B | 17.65 ± 5.9 | 0.000 | 1.00 | 17.00 ± 6.46 | 0.712 | 0.485 |
| BG (recall) | 15.1 ± 4.38 | −0.300 | 0.768 | 14.5 ± 4.77 | 0.487 | 0.632 |
| Nonverbal memory | | | | | | |
| Visual reproduction I | 3.25 ± 1.12 | 0.000 | 1.00 | 3.1 ± 1.2 | 0.825 | 0.419 |
| Visual reproduction II | 4.20 ± 1.28 | 0.000 | 1.00 | 4.05 ± 1.43 | 0.616 | 0.545 |
| Spatial ability | | | | | | |
| WAIS | 89.25 ± 16.1 | −0.240 | 0.813 | 87.400 ± 18 | 6.00 | 0.556 |
| BG (copy) | 16 ± 4.78 | −0.000 | 1.00 | 15.40 ± 5.18 | 0.940 | 0.359 |
| Attention | | | | | | |
| Digit forward | 6.55 ± 1.57 | 0.000 | 1.00 | 6.35 ± 1.76 | 0.748 | 0.464 |
| Digit backward | 3.75 ± 1.52 | 0.623 | 0.541 | 3.55 ± 1.73 | 1.37 | 0.186 |
| Executive fugitive | | | | | | |
| Trail making A | 239.8 ± 60.1 | 1.339 | 0.739 | 244.24 ± 60.9 | −0.377 | 0.710 |
| Trail making B | 576.9 ± 12.9 | −0.563 | 0.580 | 599.21 ± 15.6 | −1.46 | 0.158 |
| Word association | 10 ± 2.55 | 0.092 | 0.928 | 9.55 ± 2.98 | 0.768 | 0.452 |

BG, Bender–Gestalt test; MMSE, mini-mental state examination; SD, standard deviation; WAIS, Wechsler adult intelligence scale.

**Table 7 Odds ratios of factors predicting executive dysfunction derived from logistic regression analysis**

| Variable | Odds ratio | 95% Confidence interval | Significant |
|---|---|---|---|
| Age | 0.1 | 0.79–1.05 | 0.185 |
| Sex | 8.89 | 0.001–21.3 | 0.05 |
| Diagnosis of hypertension | 1.86 | 0.6–68.5 | 0.122 |
| Site of lesion | | | |
| Thalamic | 2.29 | 0.43–226 | 0.11 |
| Lobar | 2.123 | 0.61–181 | 0.10 |
| Basal ganglia | 0.02 | 0.03–28 | 0.99 |

**Table 8 Predictors of cognitive impairment at the second-year follow-up: multivariate logistic regression**

| | *B* | SE | 95% CI | *P* |
|---|---|---|---|---|
| Age at stroke onset | 0.55 | 0.26 | 1.04–2.9 | 0.03 |
| Baseline cognition | −0.91 | 1.5 | 0.02–7.8 | 0.54 |
| Site of lesion | −1.6 | 1.9 | 0.004–9.18 | 0.4 |

*B*, beta standardized coefficient; CI, confidence interval; SE, standard error.

of CIND was diagnosed in three cases of stroke group (CDR = 0.5) and one case of the nonstroke group between the first-year and the second-year follow-up interviews. Dementia was diagnosed in five stroke cases and one nonstroke case. Of these, four stroke cases had developed a new incidence of dementia (two cases CDR = 1, one case CDR = 2, one case CDR = 3) between the first-year and the second-year follow-up interviews, whereas the other two cases developed dementia (CDR = 1) at the 2-year follow-up. However, by the end of the follow-up, the majority of the patients (25, 70.1%) and (17, 85%) controls were cognitively stable. Fewer stroke cases than nonstroke cases showed an improvement from being cognitively impaired at baseline to being unimpaired at 2 years [19.4% (4/21) and 25% (1/4), respectively]. Also, 42.8% (6/14) of stroke cases worsened from being cognitively unimpaired at baseline to being impaired at 2 years versus 12.5% (2/16) of nonstroke cases. Table 4 shows that there is impaired cognitive function in nearly all domains with a significant difference between stroke and nonstroke groups. Overall stroke cases declined the most in MMSE, spatial ability, and in executive function between baseline and the first and the second year of follow-up with a statistically significant difference and to a lesser extent in other domains with no statistically significant difference. There was also a lack of improvement in attention and nonverbal memory (Table 5). Nonstroke cases, in contrast, showed stable performance in all cognitive domains (Table 6). Table 7 shows that sex was a significant predictor of executive dysfunction when a multivariate regression analysis was carried out for significant factors in univariate analysis. Also, in multivariable analysis, only the age at stroke onset was independently associated with cognitive impairment at the second year of follow-up (Table 8).

## Discussion

In the early phase of stroke, a cognitive disorder is usually related to the direct local effects of the lesion, indicating that the afflicted brain area is an essential component in a network subserving that specific cognitive function. In case of ischemic stroke, these local effects are related to the core area of the infarct and the ischemic zone surrounding the infarct, whereas an intracerebral hemorrhage usually causes symptoms by exerting pressure on the surrounding brain tissue. Indirect effects may also play a role in causing cognitive impairment, such as (a) 'diaschisis', in which the lesion cuts off neural input to a remote area of the brain, causing a dysfunction of that remote area [8], (b) 'hypoperfusion', in which neural dysfunction is caused by a decreased cerebral blood flow in case of occlusion of one or more cerebropetal arteries [26], or (c) neuronal metabolic abnormalities throughout the entire brain beyond the effects of the stroke lesion [27]. In patients with stroke, CIND is associated with disability and a rate of progression to dementia, but its operational definition is not straightforward.

The main objective of this longitudinal study was to determine the cognitive evolution over 2 years of patients with first-ever hemorrhagic stroke. A remarkable finding of this study is the evidence of multiple evolutionary trends in the sample. There were patients with stable, improving, or worsening cognitive status at every evolutionary time frame and in every cognitive stage. In agreement with this study, the study of Tham *et al.* [28] found a 33% overall rate of change from the cognitive baseline state after a 1-year follow-up both in improving and deteriorating cases. Several studies have reported that cognitive impairment after stroke may improve over time, although the recovery rates were very diverse because of the different basal characteristics of the samples. In this study, six patients (19.4%) improved during the follow-up and, in agreement with previous reports [29], patients who had dementia showed a high improvement rate, (13.9%).

Most of our patients with and without cognitive impairment (70.1%) had stable cognition during follow-up. Del Ser *et al.* [4] also found that most of the dementia and nondementia cases (78.2%) had stable cognition during follow-up. A neuropsychologic battery exploring different cognitive domains is an objective and more reliable tool and therefore all studies on poststroke cognitive impairment have used this for diagnosis. However, there is no standard battery validated for this purpose and every group has selected a different set of tests and different cutoff points.

Three months after stroke, 60% of our patients with stroke were classified as CIND. Previous series have reported a poststroke CIND frequency that ranges between 35 and 71% [17,28,29]. These different figures can be explained by differences in the design of neuropsychological batteries, cutoff values, inclusion–exclusion criteria (such as exclusion of cases with previous stroke), and very importantly, the time points chosen for poststroke baseline. Because most cognitive recovery occurs within 3 weeks to 3 months after stroke [30], a short interval for poststroke baseline can overestimate cognitive impairment.

We found an increased risk of incident dementia at 2 years in patients with stroke who were cognitively impaired at baseline. Also, a single stroke was strongly associated with CIND at the first follow-up. A study carried out by Ballard *et al.* [31] confirmed our finding. This suggests that the impact of a single stroke on dementia is unlikely to be a delayed phenomenon. It is possible that a single stroke, because of its early cognitive effects, may lower the threshold for future clinical expression of preexistent neurodegenerative disease. We speculate that CIND in patients with stroke is likely to represent a mixture of the effects of cerebrovascular disease and neurodegenerative disease, but this requires further investigation.

In the next 2 years, patients undergo individual changes but the overall frequency of CIND remains fairly stable. Del Ser [4] published data on the stability of cognitive status 2 years after stroke assessed by changes in the CDR score.

Actual transitions in diagnostic state have been reported in two hospital-based studies [4,28]. In a Singapore sample [28], 31% of patients with CIND at 6 months were cognitively stable at 1 year. High rates of improvement from CIND (44%) and dementia (19%) were also found at 2 years after stroke in a Spanish study [4]. However, in this study, there is low rates of improvement from CIND (19%), while there is no improvement from dementia. Definitional differences may partly explain the varying results between these studies and our study. Our criteria of dementia most likely identified people with an Alzheimer-type dementia, given the primacy of memory impairment and the requirement for progression. This therefore constrains the possibility of recovery from dementia. Also, the small size of our study sample may explain these results. In addition, as acknowledged by the Spanish investigators [32], aphasic individuals may have been misclassified as cases of dementia; thus, improvements in language may have led to a perception of recovery from dementia. However, we excluded people with significant aphasia, thus eliminating the possibility of a misdiagnosis.

Hajat *et al.* [33] found differences between the subtypes of hemorrhagic stroke, with a higher prevalence of hypertension, DM for primary intracerebral hemorrhage, and higher rates of smoking for subarachnoid hemorrhage. In agreement with this study, we found a higher incidence of hypertension and DM among the stroke group than the nonstroke group.

We found that first-ever hemorrhagic stroke was associated with reduced spatial ability, executive function, and a decline in the MMSE score between baseline and the first-year follow-up and baseline and the second-year follow-up. These results are in agreement with another study [34], in which they found that a single stroke was associated with reduced spatial ability, attention, and mental speed. However, executive function impairment found in our results could be explained by the fact that mostly our patients had a hematoma in subcortical structures (basal ganglia and thalamus) and the frontal lobe. This is in keeping with recent concepts of the neurobiology of executive functions, which emphasize the importance of frontal–subcortical circuits [35]. It has been postulated that the clinical dysexecutive syndromes observed with frontal lobe injures may also result from strategic lesions in subcortical components of specific frontal–subcortical circuits. However, a recent study [36] assessed cognitive disability changes after hemorrhagic stroke in 62 patients, with evaluation after 1, 3, and 6 months. It was found that achieved problem solving and safety and social behavior recovery were lower than those who showed attention communication and memory recovery.

There was no relation between the site of hemorrhage and cognitive functions and this may be because of the small number of cases.

## Conclusion

In this prospective design with high follow-up rates, cognitive evolution 2 years after hemorrhagic stroke is different among patients, but a substantial number of patients remain stable. Additional population-based studies are required to reliably identify individuals at risk of cognitive decline for whom pharmacologic or other therapeutic interventions would be more suitable and those who will probably show spontaneous improvement. The determinants of progression of cognitive impairment are still poorly known. In our sample, age at stroke onset was found to be a determinant of progression of cognitive impairment. It was also found in this study that sex can be considered as a predicting factor for executive dysfunction. Further Egyptian studies are essential with a larger sample size. Classification of stroke by etiology may be more useful for explaining the excess risk of stroke and the scope for its prevention.

## Acknowledgements

### Conflicts of interest

There is no conflict of interest to declare.

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# الوظائف المعرفية بعد السكتة الدماغية النزفية الأولى: دراسة متابعة

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**قسم الأمراض النفسية – جامعة المنصورة قسم الأمراض العصبية – جامعة المنصورة**

هناك ندرة في الأبحاث المتعلقة بتأثير السكتة الدماغية النزفية الأولى على الوظائف المعرفية على المدى الطويل ومعرفة نسب التحسن والتدهور. والهدف من هذا البحث دراسة معدل التغير في الوظائف المعرفية على المدى الطويل وعوامل الخطر المتعلقة بحدوث العته والقصور في الوظائف المعرفية بدون عته، عامين بعد الإصابة بالسكتة الدماغية النزفية الأولى. وقد أجريت الدراسة على 35 مريض مصاب بالسكتة الدماغية النزفية للمرة الأولى من قسم الأمراض العصبية بمستشفى المنصورة الجامعي وعدد 20 شخص من الأصحاء متوافقين مع المرضى من حيث السن والنوع. وقد أجريت لهم متابعة لمدة سنتين خلال 3 فترات تقييم متتالية 3 ، 12 ، 24 شهر. وقد خضعت عينة المرضى في كل متابعة لفحص إكلينيكي ونفسي وعصبي وكامل وأشعة مقطعية بالإضافة إلي قياسات نفسعصبية شاملة لتقييم الوظائف المعرفية المختلفة وقد تم مقارنة معدلات التغير في الوظائف المعرفية من خلال إحصاء تحليلي متكرر وكذلك لتحديد العوامل المرتبطة بحدوث العته والاضطراب في الوظائف المعرفية بدون عته. وقد خلصت النتائج إلي أن حوالي 71.4% من المرضى بالسكتة كانوا مستقرين من حيث الوظائف المعرفية، وحوالي 71.5% تحسنوا، بينما 17.1% ساءت وظائفهم المعرفية وذلك في نهاية سنتين المتابعة. وقد شخص التدهور في الوظائف المعرفية بدون عته في 18 حالة من المرضى (51.4%) وشخص العته في 5 حالات من المرضى (14.2%) وبصفة عامة وجد أن هناك تدهور في اختبار الفحص العقلي المختصر وفي الوظائف الحيزية (المكانية) والوظائف التنفيذية. بينما هناك ضعف في التحسن في وظائف الانتباه والذاكرة الغير لفظية. وقد وجد أن القصور في الوظائف المعرفية بعد سنتين كان متعلق بالسن عند حدوث السكتة الدماغية النزفية الأولى. وقد أسفرت الدراسة أيضا وجود نسبة أكبر من الإصابة بارتفاع ضغط الدم والسكر والتدخين بين المرضى عنه بين العينية الضابطة. وقد خلصت الدراسة إلي أن التقييم المعرفي بعد سنتين من حدوث السكتة الدماغية النزفية غير متجانس على الرغم من أن هناك عدد وافي من المرضى كان في حالة مستقرة. وأنه يلزم وجود دراسات إضافية لتحديد أو تقييم الأشخاص المعرضين لحدوث قصور في الوظائف المعرفية، الملائمين للعلاج الدوائي أو وسائل العلاج الأخرى أو هؤلاء الذين من المتوقع تحسنهم تلقائيا. وتوصي الدراسة بتصنيف مرضى السكتة الدماغية تبعا لسبب السكتة، لأنه وجد ذلك أفضل لتفسير زيادة الخطر لحدوث السكتة الدماغية وأملا في منع حدوثها.