Eur Arch Psychiatry Clin Neurosci (1993) 243:11–15

European Archives of Psychiatry and Clinical Neuroscience

Cognitive impairment after acute supratentorial stroke: a 6-month follow-up clinical and computed tomographic study

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Received January 11, 1993

Summary. To document the occurrence, time course, and predictors of global cognitive impairment following a supratentorial stroke, we prospectively studied 41 consecutive patients with acute cerebral ischemia and no evidence of pre-existing intellectual disturbances. The Graded Neurologic Scale and Mattis Dementia Rating Scale were used to assess neurologic and cognitive deficits within the first week, 3 weeks and 6 months after the onset of symptoms. CT was performed at each examination and semiquantitative measurements of infarct volumes and brain atrophy were obtained. Sixty-one percent of patients were found to be cognitively impaired within the first week. After 6 months this deficit had resolved in 24%, but was still present in 37% of individuals. Initial findings associated with a high risk of long-term intellectual dysfunction were: 1. moderately severe cognitive impairment, 2. diminished alertness in the acute stroke stage, 3. infarction involving the temporal lobe, 4. evidence of multiple brain infarcts and 5. pronounced ventricular enlargement. Logistic regression analysis revealed temporal infarcts and evidence of multiple ischemic lesions as the most powerful predictors of persistent cognitive impairment. By these two factors alone, 85.4% of study participants could be correctly classified regarding their cognitive outcome. These results suggest cognitive dysfunction to be a frequent sequela of supratentorial stroke. Its long-term persistence may be predicted on the basis of certain features.

Key words: Cognitive impairment – Stroke – Computed tomography

Introduction

Cerebrovascular disease is generally considered the second most common cause of cognitive impairment in the elderly [4, 6, 19, 20, 27]. The risk factors and radiographical correlates of cognitive dysfunction due to cerebral ischemic injury are still a matter of debate [24]. The controversy partly stems from difficulties in establishing a causal relationship between strokes and intellectual deficits. Both disorders commonly occur with aging and therefore may co-exist merely by chance. The most directed approach in addressing the problem of causality is probably the long-term follow-up of acute stroke patients free of preceding cognitive deficits, since this could enable the temporal linkage of mental deterioration to the onset of cerebrovascular attacks.

We here present a longitudinal study over 6 months in a first-ever stroke population involving neuropsychologic testing and computed tomography (CT). Our goals were to document the occurrence and time course of stroke-related cognitive decline as assessed by a global dementia rating scale and to identify clinical and CT findings associated with a high risk for persistent cognitive alterations.

Patients and methods

The study cohort consisted of all first-ever stroke patients admitted to the Dent Neurologic Institue over an 8-month period who met the following criteria:

- clinial findings compatible with a supratentorial infarct
- hemorrhage ruled out by CT
- completion of first evaluation within 7 days of stroke onset
- ability of patients to undergo psychometric testing. Individuals with profoundly impaired consciousness and severe aphasia were therefore excluded
- no evidence of pre-existing cognitive disturbances as assessed by the Geriatric Evaluation by Relatives Rating Instrument [22] which was administered to a close relative living in the patient's household
- no history or signs of concomitant disease known to be potential causes of dementia such as psychiatric disorders, history of substance abuse, head injuries, multiple sclerosis or metabolic disease. There was no historical evidence of Alzheimer's disease in any study participant.
- scores < 8 on the Selfcare (D) scale [5] to exclude patients with pronounced depression.

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All patients satisfying these criteria were examined within 1 week, 3 weeks and 5–7 months (mean 6 months) after the ictus. At each examination they underwent repeated neurologic and neuropsychologic testing and CT. Examinations and tests were always performed by the same neurologist (R.S.).

Diagnosis of cerebrovascular risk factors relied on the patient's history and the laboratory results during the period of evaluation. Accepted risk factors were arterial hypertension (> 160 mm Hg systolic or 95 mmHg diastolic), diabetes mellitus (> 160 mg/dl fasting blood sugar level), cardiac disease (coronary heart disease, myocardial infarction, valvular diseaes, cardiomyopathy, congestive heart failure, arrhythmia), hypercholesterolemia (> 250 mg/dl) and smoking.

The neurologic deficits were recorded with the Graded Neurologic Scale by Adams et al. [1] designed for use in hemispheric strokes. Besides giving a overall deficit score this scale quantifies the level of consciousness, language function, motor and sensory deficits as well as impairment of higher cortical functions.

Intellectual performance was evaluated utilizing Mattis Dementia Rating Scale [17]. This instrument allows bedside testing and provides an estimate of general cognitive functioning. It includes subtests of attention, initiation and perseveration, construction, conceptualization and verbal and non-verbal short-term memory. Adhering to Mattis [17] scores lower than 137 were considered abnormal. Conventional standards were used to grade cognitive impairment as mild (134–136), moderate (129–133) or severe (128 or less). CT images were obtained on a high resolution scanner with a slice thickness of 8 mm and a matrix size of 180 × 180 pixels. All images were oriented in the transverse plane at 10° from Reid's baseline. Contrast agents were not administered.

Each set of images was assessed by one experienced reader unaware of the patients' test results for presence, number, location, type (branch pattern, watershed, lacunar) and side (left, right) of infarcts. For measurements of infarct volume the contours of areas of abnormal low attenuation were outlined on subsequent slices using the cursor-controlled stylus of the CT console region of interest utility. Software utilities available with our system then calculated the volume of the respective regions and total infarct volume was obtained by summing the data from all slices containing the lesion. In the case of initially negative CT studies, the 3-week examination was used for analysis.

Additional CT features assessed were leukoariosis, which was defined and rated following a previously proposed classification of our own [15], and cerebral atrophy. Ventricular and cortical atrophy were graded as mild, moderate or severe. In addition, linear and volumetric atrophy measurements were conducted for each set of images on the CT console. Indices of ventricular atrophy were the frontal horn ratio (FHR) [16], the third ventricular ratio (TVR) [16], and the ventricle to intracranial cavity ratio (VICR) [21]. Measures of cortical atrophy were the Sylvian fissure width (SFW) [14], the frontal interhemispheric fissure ratio (FIFR) [23] and the cortical sulci ratio (CSR) [23].

All measurements on CT used the inner limit of the skull with bone windows for determination of intracranial width [29].

Statistical analysis was performed with the microcomputer version of the Statistical Package of Social Sciences (SPSS/PC) [18]. Differences between means were tested using one-way analysis of variance. The Duncan t-test was applied thereafter to ascertain significant differences. In the case of data with a non-normal distribution the Kruskal-Wallis test was administered. Proportions were compared by means of the Fisher's Exact Test. Logistic regression analysis was used to create a model of predictors of the cognitive outcome of study participants. Stepwise backward regression was performed to obtain the model with best fit.

Results

Fourty-five patients were eligible for the study. Four participants were lost to follow-up; 2 died from vascular causes, 1 had a recurrent stroke with severe aphasia and 1 declined to continue the study.

Table 1. Demographic data of groups

	Cognitive impairment		
	None ($n = 16$)	Transient ($n = 10$)	Persistent ($n = 15$)
Sex (M/F)	12/4	6/4	7/8
Age (mean ± SD)	65.1 ± 12.2	68.1 ± 6.7	69.3 ± 5.3
Age range (years)	33–80	55–78	58–82
Education/years (mean ± SD)	14.3 ± 2.0	11.4 ± 2.8	12.1 ± 3.1
Side of stroke (R/L)	10/6	6/4	9/6
Hypertension (%)	9 (56.3)	7 (70.0)	8 (53.3)
Diabetes (%)	3 (18.3)	2 (20.0)	6 (40.0)
Cardiac disease (%)	10 (62.5)	6 (60.0)	13 (86.7)
Hyperlipidemia (%)	5 (31.3)	4 (40.0)	6 (40.0)
Smoking (%)	4 (25.0)	3 (30.0)	3 (20.0)

Among those 41 subjects completing the study, cognitive impairment was noted in 25 (60.9%) at the first week, in 16 (39.0%) at the third week and in 15 (36.6%) at the 6-months evaluation. Among patients with persistent cognitive alterations the congitive domaine most commonly affected was attention (80%) followed by mnestic abilities (73.3%), conceptualization (66.7%), initiation (46.6%) and perseveration (6.7%). At each time of assessment intellectual impairment was always seen to involve at least two subcategories of cognitive functioning.

By the course of cognitive performance three subsets of patients could be distinguished. Sixteen (39.0%) subjects were free of cognitive impairment throughout the study. There were 10 patients (24.4%) with a transient intellectual decline and another 15 (36.6%) patients in whom cognitive deficits persisted over 6 months. The minimal improvement of any study participant with transient cognitive alterations was five points on the Mattis scale with a mean of 6.4, SD 2.2 points. The investigational subsets were comparable for sex, age and level of education. They differed neither in the prevalence of cerebrovascular risk factors nor in the side of clinical strokes (Table 1).

Twelve of the 15 patients (80%) with persistent intellectual dysfunction had moderately severe cognitive impairment at the initial examination as opposed to 3 of the 10 (30%) with transient cognitive decline ($P < 0.02$).

Throughout the study, patients with persistent cognitive deficit were always found to be more severely impaired neurologically than their cognitively never-affected counterparts. The overall neurologic score, however, did not discriminate patients with reversible from those with persistent cognitive dysfunction (Table 2). The scores evaluating language functions and hemineglect did not differ among groups.

Initially decreased alertness was noted in 6 (40%) individuals with persistent intellectual impairment, but in none of the other two subgroups ($P < 0.01$ vs cognitively-never affected patients and $P < 0.05$ vs patients with transient cognitive impairment).

Table 2. Severity of the overall neurologic deficit (Adams Stroke Scale)

	Cognitive impairment		
	None ($n = 16$) Mean (SD)	Transient ($n = 10$) Mean (SD)	Persistent ($n = 15$) Mean (SD)
1st week	10.6 (10.5)	24.2 (21.3)	29.0 (17.8)*
3rd week	6.5 (8.6)	15.3 (18.0)	22.2 (17.5)*
6th month	4.1 (6.1)	14.7 (17.7)	17.7 (14.7)*

* Kruskal-Wallis test $P < 0.05$ vs patients with cognitive impairment

Table 3. Location and side of infarcts[a]

	Cognitive impairment		
	None ($n = 16$) n (%)	Transient ($n = 10$) n (%)	Persistent ($n = 15$) n (%)
Frontal lobe	1 (6.3)	2 (20.0)	5 (33.3)
Right	0	2 (20.0)	4 (26.7)
Left	1 (6.3)	0	1 (6.7)
Parietal lobe	9 (56.3)	3 (30.0)	9 (60.0)
Right	3 (18.8)	1 (10.0)	6 (40.0)
Left	6 (37.5)	2 (20.0)	3 (20.0)
Temporal lobe	1 (6.3)	2 (20.0)	10 (66.7)*, **
Right	0	1 (10.0)	6 (40.0)
Left	1 (6.3)	1 (10.0)	4 (26.7)
Occipital lobe	1 (6.3)	1 (10.0)	0
Right	1 (6.3)	1 (10.0)	0
Left	0	0	0
Basal ganglia	9 (56.3)	4 (40.0)	6 (40.0)
Right	6 (37.5)	2 (20.0)	2 (13.3)
Left	3 (18.8)	2 (20.0)	4 (26.7)
Thalamus	0	0	3 (20.0)
Right	0	0	2 (13.3)
Left	0	0	1 (6.6)
Cerebellum	0	2 (20.0)	1 (6.6)
Right	0	2 (20.0)	1 (6.6)
Left	0	0	0

[a] Several patients had several lesions in different location. Several lesions involved more than one territory
Fisher's Exact test * $P < 0.001$ vs controls; ** $P < 0.05$ vs transient cognitive impairment

Overall, the first CT was negative in 13 (28.9%) subjects. At least one area of infarction was seen in all patients after 3 weeks. Table 3 gives a breakdown of lesion location and side among the investigational groups. As can be seen from this table long-term cognitive deficits were commonly associated with infarcts involving the temporal lobes. Right hemispheric infarcts occurred in 10 (66.7%) individuals with persistent intellectual decline, in 7 (70.0%) with transient cognitive deficits, and in 10 (62.5%) never cognitively impaired subjects. The respective figures for left hemispheric involvement were 8 (53.3%), 5 (50.0%) and 8 (50.0%), respectively. These differences were not statistically significant. As can be seen from Table 4, the group with persistent cognitive dysfunction had significantly more infarcts. While multiple lesions were noted in 8 (53.3%) patients of the latter subset this was the case in only 2 (20% individuals with transient cognitive decline ($P < 0.05$) and 2 (12.5%) never cognitively impaired subjects ($P < 0.01$). By contrast, infarct volume (range 0.2–103 cm^3) was not clearly related to unfavourable cognitive outcome. The same was true for the type of infarction.

Table 4. Number and volume of infarcts

	Cognitive impairment		
	None ($n = 16$) Mean (SD)	Transient ($n = 10$) Mean (SD)	Persistent ($n = 15$) Mean (SD)
Infarct number[a]	1.1 (0.3)	1.2 (0.4)	1.8 (1.1)*, **
Infarct volume[b]			
1st week	3.9 (6.8)	12.2 (16.3)	25.5 (37.1)
3rd week	3.3 (4.3)	15.4 (25.3)	15.8 (25.2)
6th month	2.0 (3.9)	11.0 (22.8)	17.4 (29.4)

[a] Duncan t-test and [b] Kruskal-Wallis test * $P < 0.05$ vs controls; ** $P < 0.05$ vs transient cognitive impairment

Table 5. Comparison of atrophy measurements (1st-week results)

	Cognitive impairment		
	None ($n = 16$) Mean (SD)	Transient ($n = 10$) Mean (SD)	Persistent ($n = 15$) Mean (SD)
FHR (%)	32.6 (5.6)	33.9 (4.9)	35.9 (5.6)
TVR (%)	3.1 (1.5)	3.2 (1.4)	4.6 (1.6)*, **
VICR (%)	5.4 (2.2)	5.9 (1.4)	7.6 (3.1)*
SFW (mm)	3.2 (1.7)	3.9 (2.4)	5.1 (2.5)*
FIFR (%)	3.9 (1.3)	3.0 (1.6)	4.5 (1.7)
CSR (%)	10.4 (4.6)	12.4 (4.6)	13.2 (3.3)*

FHR = frontal horn ratio, TVR = third ventricular ratio, VICR = ventricle to intracranial cavity ratio, SFW = Sylvian fissure width, FIFR = frontal interhemispheric fissure ratio, CSR = cortical sulci ratio

A total of 9 (20%) individuals had evidence of leukoaraiosis. No interval changes were appreciated. Leukoaraiosis was seen in 3 (20%) subjects with persistent and in 2 (20%) with transient intellectual deficits. However, there were also 4 (25%) cognitively-never impaired patients with such abnormalities. The severity of leukoaraiosis was comparable between groups.

The prevalence of both ventricular and cortical atrophy was higher in patients with persistent mental dysfunction than in the other subsets. Moderately severe ventricular enlargement was noted in 7 (46.6%) individuals with poor cognitive outcome, in 1 (10%) with transient cognitive decline and in two patients (12.5%) without intellectual impairment ($P < 0.05$ persistent intellectual dysfunction vs both other groups). Cortical atrophy ratings followed the same trend, the differences between the comparative groups were not statistically significant, however. Linear and volumetric measurements confirmed the results of subjective atrophy assessments (Table 5).

An increase of ventricular size over time was noted in 6 (14.6%) patients. Those were equally distributed among the three groups of cognitive outcome. Ventricular enlargement during follow-up was focal and related to parenchymal loss from large infarcts. No definite interval change was seen in terms of cortical atrophy.

Logistic regression analysis was used to create a model of putative predictors of persistent cognitive dysfunction. Factors included in this analysis were moderately severe cognitive impairment and diminished alertness at the acute stroke stage, temporal infarct location, side and size of infarction, evidence of multiple infarcts as well as moderately severe ventricular atrophy. The cognitive outcome could be correctly predicted by these variables in 87.8% of study participants. When using stepwise backward regression temporal infarcts ($\beta = 1.4$; SE (β) = 0.47; $P < 0.01$) and multiple infarcts ($\beta = 1.6$; SE (β) = 0.7; $P < 0.05$) were determined as the only significant and independent predictors of the patients' cognitive outcome. 85.4% of subjects were correctly classified based on these two variables alone.

Discussion

To date there have been only two studies [by Tatemichi and coworkers (25, 26)] that described the prevalence and risk factors of global cognitive impairment after acute ischemic strokes. This investigation has focused on a smaller gorup of subjects, but extends previous work by examining first-ever stroke patients without overt pre-existing intellectual problems. This is probably the best possible way to establish a causal relationship between the cerebrovascular accident and cognitive dysfunction. Moreover, our study is significantly different from Tatemichi's work [25, 26] by seeking for clinical and CT factors that may enable prediction of long-term cognitive impairment already in the acute stroke stage.

Adhering to conventional standards [17], we found 61% of patients to be cognitively impaired initially. The deficit resolved in 24% and persisted in 37% of the study participants. Tatemichi et al. [25] in their first investigation reported a 16% dementia rate among stroke survivors and, comparable to our study, a 26.3% rate of dementia in their second investigation [26], which also utilized neuropsychological testing for the assessment of the patients' intellectual abilities. For two reasons caution is advised when considering any of these figures as representative of the entire stroke population. First, even though all storke patients admitted to our institute during the enrollment phase have been screened for possible study inclusion only 27.4% of them fullfilled the selection criteria. Second, all stroke samples studied thus far have been derived from hospital series which may be subject of selection bias. In several populations up to 40% of the stroke patients may never become hospitalized for their stroke and subjects who are hospitalized may be younger, may have more severe deficits or may be better educated. Nevertheless, within a selected cohort it is still possible to identify patients developing or not developing cognitive impairment and make meaningful subgroup comparisons.

Other confounding factors in the evaluation of cognitive function of stroke patients may be speech disturbances or hemineglect resulting from focal brain lesions. Such an influence on our results is unlikely, since both language function and hemineglect grading on Adam's stroke scale were similar for cognitively impaired and non-impaired groups throughout the observation period.

Tatemichi et al's first study [25] described age and previous cerebrovascular attacks as risk factors for cognitive impairment in patients with stroke. In the authors' second investigation [26] a significant correlation between dementia and length of education was reported. We also noted a trend towards higher age in patients with persistent cognitive alterations. The extent of formal education was not related to intellectual decline in our study. This, however, might have been due to the smaller size of present cohort which certainly reduced the power of statistical analysis. The role of previous cerebrovascular accidents in the evolution of post-stroke cognitive dysfunction remained unaddressed by our approach, since we examined first-stroke-ever patients only. We corraborate previous results in that sex and major cerebrovascular risk factors were unrelated to the occurrence of long-term cognitive impairment as were the type and severity of the neurologic deficit.

Our study demonstrates two clinical findings, namely a moderately severe cognitive impairment and altered alertness at the initial examination to be significantly more common in patients with persistent intellectual dysfunction than in individuals with favorable cognitive outcome. In agreement with previous research the radiographical correlates of persistent cognitive deficits were infarctions of the temporal lobes [12, 28] and multiple ischemic lesions [3]. Regression analysis demonstrated that these two findings were the two most powerful predictors of persistent cognitive impairment in our study. Based on their evidence 85.4% of study participants would have been correctly classified in terms of their cognitive outcome. Patients with persistent intellectual decline also more frequently showed moderately severe ventricular atrophy [25]. A similar relationship between cerebral atrophy and intellectual dysfunction is well known for patients with Alzheimer's disease [8, 9, 11, 14, 16] which may lead to the postulation that in a portion of patients with clinically stroke-related cognitive impairment, strokes simply unmasked an already present yet subclinical degenerative process. Alternatively, their brain atrophy might have resulted from chronic ischemia and superimposition of an acute focal lesion to the previously damaged brain may have ultimately exceeded the "cognitive reverse". The size of infarcts tended to be larger in patients with persistent intellectual dysfunction as well. There was, however, considerable overlap of infarct volumes between the three comparative study subsets, which made this parameter a poor predictor of the patients' cognitive outcome. Neither the presence nor the extent of leukoaraiosis was seen to be associated with intellectual impairment. This opposes previous reports [2, 13, 15] only at first glance, but can be readily attributed to our selection of patients with strokes but without preceeding cognitive deficits. As a

consequence we most likely excluded patients with marked leukoaraiosis which is the hallmark of Binswanger's disease and typically presents with insidiously progressing mental alterations, but frequently without concomitant neurologic signs or symptoms [7].

In summary, cognitive deficits are a common sequela of supratentorial strokes. They may resolve with time or persist beyond 6 months. Risk factors for long-term intellectual dysfunction include CT evidence of temporal lobe infarction and multiple infarcts, profound cognitive deficits and impaired alertness in the acute stroke stage as well as brain atrophy.

Acknowledgements. This study was supported by the "Fond zur Förderung wissenschaftlicher Forschung", Vienna, Austria (project J 407MED).

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