

Persistent Cognitive Impairment After Transient Ischemic Attack

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Background and Purpose—By definition, the symptoms of a transient ischemic attack (TIA) subside completely within 24 hours. Imaging studies show signs of persistent ischemic tissue damage in a substantial amount of patients with TIA. Cerebral infarction can cause permanent cognitive impairment. Whether permanent cognitive impairment occurs after TIA is unclear, as is its profile.

Methods—Patients with TIA aged 45 to 65 years without prior stroke or dementia underwent comprehensive neuropsychological testing within 3 months. Z scores per cognitive domain were obtained, based on the mean of a control group within the same age range. Cognitive impairment was defined as a domain z score < -1.65 . Patients underwent either computed tomography or MRI brain imaging.

Results—One hundred seven patients with TIA (63% women, mean age, 56.6 years) were included and compared with 81 controls (56% women, mean age, 52.9 years). Patients performed worse on all cognitive domains except episodic memory. Working memory (25%), attention (22%), and information processing speed (16%) were most frequently impaired and more often than in the control group (age- and sex-adjusted odds ratios, respectively, 22.5 [95% confidence interval, 2.9–174.3], 6.8 [1.9–24.3], 7.1 [1.5–32.5]). More than 35% of patients with TIA had impairment of ≥ 1 cognitive domain. Presence of silent brain infarcts was related to worse executive functioning but did not explain the whole relationship between TIA and cognitive impairment.

Conclusions—More than a third of patients with TIA have impairment of ≥ 1 cognitive domain within 3 months after their TIA. The affected domains fit in the vascular cognitive impairment profile. (*Stroke*. 2014;45:2270–2274.)

Key Words: cognition ■ ischemic attack, transient

By definition, symptoms of a transient ischemic attack (TIA) subside completely within 24 hours.¹ Studies using diffusion-weighted imaging found signs of cytotoxic edema beyond the point of symptom resolution in $>30\%$ of patients with TIA.²

Permanent cerebrovascular damage, both with and without clinical signs of brain infarction, may lead to cognitive decline.^{3–5} This cognitive impairment is referred to as vascular cognitive impairment and has a profile characterized by executive and attention deficits with a relatively intact memory function.⁶

A TIA could give rise to transient cognitive defects, although studies examining the persistency of this cognitive impairment beyond 1 month after the TIA are scarce and often include elderly patients.^{7–11} Any cognitive impairment potentially because of a TIA might be obscured by age- or Alzheimer pathology-related cognitive decline.

Furthermore, the profile of cognitive impairment after TIA is unknown.

We, therefore, determined the cognitive performance of patients within 3 months after a TIA in a single center cross-sectional study. We aimed to minimize the effects of aging and concomitant cognitive disorders by restricting our research population to an age range of 45 to 65 years. We hypothesized that cognitive function after TIA would be impaired and have a vascular profile.

Methods

Participants

Patients aged 45 to 65 years attending the stroke unit or TIA outpatient clinic of the Radboud University Medical Center, after a TIA between 2004 and 2010 were consecutively recorded. Our guidelines state that patients referred to the TIA clinic should be seen within 24 to 48 hours. As part of clinical care, a comprehensive

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neuropsychological assessment was performed within 3 months after the qualifying event. The number of available slots for these assessments was restricted. However, no selection for neuropsychological assessments was used.

TIA was defined as a sudden onset focal neurological deficit of vascular origin with complete resolution of focal symptoms within 24 hours.¹ Patients with prior stroke or dementia were excluded, whereas those with prior TIA were not. In addition, patients with incident stroke or carotid endarterectomy between TIA and cognitive testing were excluded as were those with a Mini-Mental State Examination score <24 because this was considered indicative for possible pre-existing dementia and would prevent a reliable cognitive assessment.¹² All data were acquired as part of clinical care, and both storage and processing were conducted anonymously in agreement with the local ethical committee rules.

Control Group

Patients' performances on all individual tests were compared with a control group, recruited among spouses, relatives, or social environment of patients attending our outpatient department. All participants were free from TIA, and the same exclusion criteria as for patients were applied. The stroke-free status was verified through a standardized, structured questionnaire. When a possible cerebrovascular event was reported, the general practitioner was contacted for additional information.

Only for the California Verbal Learning Test, performance was not compared with the control group but with a normative sample described in the test manual ($n=164$, age, 45–64 years). None of the subjects in the normative sample had a history of psychiatric or neurological disease.¹³

Clinical Characteristics

Hypertension was considered present when (1) systolic blood pressure was >140 mm Hg and diastolic blood pressure was >90 mm Hg at both time of presentation and at 90-day follow-up, (2) antihypertensive medication was used, or (3) a previous diagnosis by a physician was recorded. Hypercholesterolemia was considered present when (1) fasting total cholesterol level was >6.5 mmol/L, (2) lipid-lowering drugs were used, or (3) a physician had previously made the diagnosis. Diabetes mellitus was defined as (1) the use of antidiabetic medication or (2) a previous diagnosis by a physician. Smoking status was determined current when a patient smoked or had stopped within the past 6 months and former when smoking was stopped earlier. Any prior myocardial infarction was noted, and the body mass index was calculated.

Brain Imaging

Brain imaging, either computed tomography or MRI, performed within 3 weeks after the qualifying event was evaluated for the presence and severity of age-related white matter changes (ARWMC) using a semiquantitative scale, as well as the presence of silent brain infarct (SBI).¹⁴ SBI was defined as an infarct on computed tomography or MRI at a location that did not correspond with the symptoms of the qualifying or any previous TIA and was classified as lacunar or nonlacunar.¹⁵ Two experienced raters (F.G.v.R. and E.J.v.D.) separately performed assessment of brain imaging. In case of disagreement, a consensus meeting was held.

Neuropsychological Assessment

A trained examiner administered the neuropsychological tests in a quiet, well-lit room and under standard circumstances. Executive functioning was assessed with a verbal fluency task (naming as many animals as possible within 60 seconds; response generation) and the interference score of the Abbreviated Stroop Color Word Test (response inhibition).¹⁶ Information processing speed was tested with cards I and II of the Abbreviated Stroop Color Word Test and the Symbol-Digit Modalities Test.^{16,17} The Paper and Pencil Memory Scanning Test (4 subtasks) was used to measure working memory,

and attention was evaluated by the Verbal Series Attention Test.^{18,19} Finally, verbal episodic memory was tested with the Dutch version of the California Verbal Learning Test, using both the total correct answers of 5 immediate recall trials and the difference between trial 5 and long-term recall (consolidation).¹³ For tests requiring both speed and precision, a speed accuracy trade-off score was calculated by dividing the percentage of correct answers by the time taken to complete the test.²⁰ This applied to the Verbal Series Attention Test, cards I and II of the Stroop Color Word Test, and all subtasks of the Paper and Pencil Memory Scanning Test. The Stroop interference score was computed by dividing the speed accuracy trade-off score of card III by the mean of the speed accuracy trade-off scores of cards I and II.¹⁶

Subjective Cognitive Failures

In addition, a 15-item semistructured interview based on the Cognitive Failures Questionnaire was administered to identify subjective cognitive failures (SCF) experienced the month before.²¹ Responses were added to provide a sum score with a maximum of 25. SCF reported in remembering, word finding, planning, concentration, and slowness in thought were given a higher weight in the sum scores (range 0–3: none, mild, moderate, severe) than the other items (0–1). If ≥ 1 moderate problem (score ≥ 2) on an item with a score range of 0 to 3 or a score of 1 on a dichotomous item was reported, SCF was considered present.

Other Measurements

Age, sex, and level of education were recorded. The presence of depressive symptoms was defined as a Hospital Anxiety and Depression Scale depression subscale score ≥ 8 .^{22,23}

Statistical Analysis

All analyses were done with IBM SPSS Statistics version 20.0 (IBM Corp, Armonk, NY). Differences in characteristics between patients and controls were compared using Student *t* test, Pearson χ^2 , and age- and sex-adjusted ANCOVA when appropriate. Bonferroni correction for multiple testing was applied with α set at 0.01.

Individual *z* scores were computed for each neuropsychological test using the mean and SD of the control group, and domain *z* scores were calculated by averaging *z* scores of individual tests. Per cognitive domain, a *z* score < -1.65 of the control group was used as a cutoff to determine domain-specific impairment (ie, corresponding to a performance below the lower fifth percentile).²⁴ Domain-specific age- and sex-adjusted odds ratios (OR) of cognitive impairment after TIA were obtained by logistic regression. No OR of episodic memory impairment was calculated because California Verbal Learning Test results were not compared with the same control group.

In case of missing neuropsychological test results (maximum 12.2%), the domain score was based on the remaining tests, or if no tests were performed within a cognitive domain, the domain *z* score was not used in further analyses.

Results

Between September 2004 and December 2010, 246 patients with TIA aged 45 to 65 years were registered, of whom 114 underwent neuropsychological testing within 3 months after the qualifying event (mean [SD], 56 [14.7] days; range, 26–91 days). Patients with and without neuropsychological assessment did not differ on age and sex (Student *t* test and Pearson χ^2 , $P=0.92$ and 0.43, respectively). Subsequently, 5 patients were excluded because of a history of stroke and 2 because of a Mini-Mental State Examination score <24. Characteristics of included patients ($n=107$) are summarized in Table 1. The control group included 81 individuals. No differences were

Table 1. Characteristics of Patients With TIA and Controls

	Patients With TIA (n=107)	Controls (n=81)	P Value*
Women	67 (62.6)	45 (55.6)	0.41
Age, mean (SD)	56.6 (6.3)	52.9 (6.2)	<0.001
Low level of education	28 (26.2)	17 (21.0)	0.52
HADS-D, mean (SD)	3.2 (3.9)	2.6 (2.7)	0.22
HADS-D score \geq 8	13 (12.1)	6 (7.4)	0.35
Body mass index, mean (SD)	26.8 (4.0)	27.4 (4.9)	0.36
Hypertension	52 (48.6)	27 (33.3)	0.05
Dyslipidemia	31 (29.0)	15 (18.5)	0.14
Diabetes mellitus	5 (4.7)	1 (1.2)	0.36
Prior myocardial infarction	8 (7.5)	1 (1.2)	0.10
Smoking status			0.32
Current	19 (17.8)	22 (27.2)	
Former	53 (49.5)	35 (43.2)	
ARWMC score, median (IQR)	1 (3)	N/A	
Silent brain infarct	18 (18.2)	N/A	
Single	17 (15.9)	N/A	
Lacunar	14 (13.1)	N/A	
DWI lesion (only MRI)	16 (27.1)	N/A	

Numbers are n (%) unless stated otherwise. ARWMC indicates age-related white matter changes; DWI, diffusion-weighted imaging; HADS-D, Hospital Anxiety and Depression Scale, depression subscale; IQR, interquartile range; N/A, not applicable; and TIA, transient ischemic attack.

*Difference using Student *t* test and Pearson χ^2 (continuity correction) when appropriate.

present between patients and controls for sex, low level of education, Hospital Anxiety and Depression Scale depression subscale mean score, Hospital Anxiety and Depression Scale depression subscale score \geq 8, or the presence of vascular risk factors. Patients were on average older than controls (56.6 and 52.9 years, respectively; $P<0.001$).

Patients with TIA performed worse than controls on each individual cognitive test and all cognitive domains, except episodic memory (Table 2). The highest impairment rates were present in the domains of working memory and attention, whereas episodic memory was relatively preserved. Age- and sex-adjusted ORs for domain-specific cognitive impairment after TIA ranged from 3.5 (executive function, 95% confidence interval, 0.7–16.7) to 22.5 (working memory, 95% confidence interval, 2.9–174.3). Impairment of \geq 1 cognitive domain (excluding episodic memory) was present in 38.3% of patients with TIA, with an associated age- and sex-adjusted OR of 5.9 (95% confidence interval, 2.4–14.5; Table 3).

Brain imaging was performed within 3 weeks after the qualifying event in 99 patients with TIA (59% MRI). Within the patient group, SBI, but not ARWMC, was associated with worse executive functioning (Table 1 in the online-only Data Supplement). SBI were almost exclusively single lacunar infarcts (4 nonlacunar infarcts, 3 of which were subcortical). Patients with TIA with signs of cytotoxic edema on diffusion-weighted imaging were not more frequently impaired (age- and sex-adjusted OR for any

Table 2. Mean Difference in Z Scores per Cognitive Domain and Cognitive Test Between Patients With TIA (n=107) and Controls (n=81)

Cognitive Test and Domain	Difference in Z Score, Mean	P Value*	% Impaired†
Executive function	-0.59	<0.001	10.3
Verbal fluency	-0.69	<0.001	
Stroop interference task	-0.48	0.02	
Information processing speed	-0.66	<0.001	16.1
Stroop task 1	-0.64	0.001	
Stroop task 2	-0.74	<0.001	
SDMT	-0.55	0.01	
Working memory	-0.87	<0.001	24.5
PPMST %	-1.47	<0.001	
PPMST 1 letter	-0.80	<0.001	
PPMST 2 letters	-0.68	0.002	
PPMST 3 letters	-0.55	0.03	
Attention	-0.51	0.01	21.7
VSAT	-0.51	0.01	
Episodic memory	0.01‡	0.96	8.4
CVLT, immediate recall	-0.02‡	0.91	
CVLT, consolidation	0.03‡	0.87	

Percentage of patients with domain-specific cognitive impairment. CVLT indicates California Verbal Learning Test; PPMST, Paper and Pencil Memory Scanning Test; SDMT, Symbol-Digit Modalities Test; TIA, transient ischemic attack; and VSAT, Verbal Series Attention Test.

*Difference using age- and sex-adjusted ANCOVA.

†Z score <-1.65 .

‡Compared with normative data based on 164 individuals aged 45 to 64 y.¹³

domain-specific cognitive impairment 0.7 [95% confidence interval, 0.3–2.0]). After excluding patients with SBI, TIA was still associated with impairment of cognition within 3 months (Table 3).

We found no difference between patients and controls with respect to mean Cognitive Failures Questionnaire sum score. SCF were reported by 59% of patients with TIA, which did not differ from the control group. SCF were associated with lower z scores in all cognitive domains for patients with TIA (age- and sex-adjusted ANCOVA; $P<0.01$).

Table 3. Odds Ratios for Cognitive Impairment Within 3 Months After TIA Compared With Controls Without TIA (n=81)

Cognitive Domain	Cognitive Impairment, OR (95% CI)*	
	All Patients With TIA (n=107)	Patients With TIA Without SBI Only (n=89)
Executive function	3.5 (0.7–16.7)	1.5 (0.3–8.7)
Information processing speed	7.1 (1.5–32.5)	4.8 (1.0–23.4)
Working memory	22.5 (2.9–174.3)†	16.2 (2.0–128.7)†
Attention	6.8 (1.9–24.3)†	5.7 (1.5–20.9)†
\geq 1 cognitive domain	5.9 (2.4–14.5)‡	5.4 (2.1–13.3)‡

Cognitive impairment defined as domain z score <-1.65 . CI indicates confidence interval; OR, odds ratio; SBI, silent brain infarct; and TIA, transient ischemic attack.

*Age- and sex-adjusted logistic regression; † $P<0.01$; ‡ $P<0.001$.

Discussion

The main findings of our study are that (1) more than a third of patients aged 45 to 65 years who had a TIA in the previous 3 months has impairment of function in ≥ 1 cognitive domain, (2) working memory, attention, and information processing speed are the most affected cognitive domains, whereas global memory functions remain relatively intact, (3) this cognitive impairment is only partly related to cerebrovascular damage on conventional neuroimaging, and (4) subjective cognitive complaints are not more frequently reported by patients with TIA than by healthy individuals.

This study was not without limitations. Given the cross-sectional design of the study without information on cognitive performance before the qualifying event, neither definite causal relations between cognitive function and TIA nor its time course could be established. Not all patients with TIA seen during the study period underwent cognitive assessment because of restricted availability of slots. To prevent selection bias, patients were assigned to cognitive assessment irrespective of clinical data. Furthermore, California Verbal Learning Test was not performed in the control group, and patients' results for this test were compared with normative data derived from a different age-adjusted reference group published in the test manual. Although this prevented calculation of ORs for overall cognitive impairment, the composition of the cognitive profile of patients with TIA separately could be described. In addition, not all patients completed the neuropsychological test battery. By computing a domain-specific compound z score based on less than all associated tests when necessary and by not using missing values in further analyses, we might have reduced statistical power of our results. Despite swift analysis after referral, patient delay caused initial brain imaging to take place ≤ 3 weeks after the qualifying event, which could have influenced diffusion-weighted imaging lesion prevalence.²⁵ Also, controls were on average slightly younger than the patients. However, they were all from the same relatively narrow age range, and although statically significant, the mean difference was small and analyses were adjusted for differences in age. Therefore, we think that this potential factor has been adequately accounted for and cannot entirely explain our findings. Finally, to reduce the influence of concomitant cognitive disorders, we excluded older patients and those with low Mini-Mental State Examination performance. This limits the generalizability of our results to the whole TIA population and might have excluded representatives of the more severe spectrum of cognitive impairment after TIA.

Previous data on cognitive function after TIA are scarce and heterogeneous.^{7–11,26} Patient characteristics, definition of cognitive impairment, and delay from TIA to assessment of cognition differ widely between studies. Furthermore, cognitive assessment is mostly limited to screening tools such as Mini-Mental State Examination and Montreal Cognitive Assessment, which are not sensitive to mild cognitive deficits after stroke and do not assess specific cognitive domains.^{27,28} Previously reported prevalence of cognitive impairment after TIA varies from 30% to 57%.^{7,9,10} Compared with our study, these studies included substantially older patients, performed cognitive assessment much later after TIA, and did not exclude

patients with previous stroke. In contrast, we aimed to minimize the effect of possible concomitant causes of cognitive impairment by performing cognitive testing within 3 months after the qualifying event in patients aged < 65 years and excluding those with a history of stroke. Despite our rigorous measures to minimize the effects of neurodegenerative or previous vascular cognitive disorders, we found a prevalence of 38% of cognitive impairment in a relative young cohort of patients with a recent TIA.

The cognitive profile after TIA showed prominent impairment in the domains of working memory, attention, and information processing speed, whereas global memory functions remained within normal ranges. This nonamnestic cognitive impairment is compatible with the vascular cognitive impairment profile and mainly driven by subcortical brain damage disrupting subcortical–frontal connections.²⁹ Only a few studies have previously described the cognitive profile of patients with TIA and found prominent deficits in executive functioning, visuoconstruction, and attention.^{7,9,10} However, one of these studies included only patients with internal carotid artery occlusion and did not exclude patients with prior stroke, whereas the others performed Montreal Cognitive Assessment instead of a more comprehensive neuropsychological evaluation.

Presence of SBI was related only to worse executive functioning, whereas ARWMC were not related to any cognitive impairment after TIA. Because of limited numbers, we were unable to assess the relationship of different types of SBI with cognitive function. The prevalence of SBI and the severity of ARWMC were low compared with population based studies, probably related to our relatively young study population.^{15,30} Both SBI and ARWMC are markers for cerebral small vessel disease, indicating that in our study population the role of small vessel disease in cognitive impairment after TIA seems limited. This is further strengthened by the robust association of TIA with cognitive impairment after excluding patients with SBI. The influence of concomitant neurodegeneration on cognitive function was minimized through a rigorous age restriction. This suggests a role for TIA itself in cognitive impairment afterward, the mechanism of which remains to be elucidated. Transient ischemia might lead to microstructural damage and loss of white matter structural integrity, giving rise to subcortical–frontal disconnection in a similar fashion as the vascular cognitive impairment construct. Verification of this potential pathway was not possible in our study because imaging modalities assessing the structural integrity of white matter were not performed.

In contrast to the high prevalence of objective cognitive dysfunction, patients with TIA did not report more SCF than controls. The prevalence of SCF among patients with TIA is remarkably lower than in elderly persons with white matter lesions and is comparable to the one study that previously reported cognitive complaints in patients with TIA.^{31,32} However, those results were not separately reported for TIA and minor stroke patients, and assessment of SCF was limited. The discrepancy between prevalence of objective and subjective cognitive dysfunction in TIA might mean that only relatively minor difficulties in everyday life are perceived. Still,

because patients with TIA in our study were relatively young, a large proportion would still be working and be socially active. Even minor cognitive decline might, therefore, have impact.

The causes of cognitive impairment after TIA remain unknown. Future studies should include advanced brain imaging techniques to identify microstructural and functional cerebrovascular damage and perform longitudinal assessment of cognitive function after TIA to observe whether cognitive impairment is transient, stationary, or progresses over time. Nevertheless, our results show the extent of cognitive impairment after TIA in relatively young adults and warrant the need for more clinical awareness of this problem.

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Disclosures

None.

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