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Abstract

Background and Purpose: Knowledge of the burden and development of post-stroke cognitive impairments (CIs) in the long-term after the first event is limited. We aimed to assess the prevalence of mild CI (MCI) and dementia 7 years after first-ever stroke or transient ischemic attack (TIA), to subclassify the impairments, and to identify predictors for a favorable cognitive outcome. **Materials and Methods:** During 2007 and 2008, 208 patients with first-ever stroke or TIA without preexisting CI were included. After 1 and 7 years, survivors were invited to a follow-

up. Transitions of cognitive status from 1 to 7 years were recorded based on the 3 categories dementia, MCI, or none. Etiologic subclassification was based on clinical cognitive profile, magnetic resonance imaging (MRI) findings, and biomarkers at both time points. Favorable outcome was defined as normal cognitive function or MCI after 7 years with exclusion of those who had progression from normal to MCI. **Results:** Eighty patients died during follow-up, 12 patients refused further participation. After 7 years, 109 completed follow-up of whom 40 (37%) were diagnosed with MCI and 24 (22%) with dementia. Of the 64 patients diagnosed with CI, 9 were subclassified with degenerative cognitive disease, 13 with vascular disease, and 42 had mixed cognitive disease. In all, 65 patients (60%) had a favorable outcome. In multivariable logistic regression analysis, lower age and lower medial temporal lobe atrophy (MTLA) grade on MRI at 12 months were independently associated with a favorable outcome, adjusted OR (95% CI), 0.94 (0.86–0.92), and 0.55 (0.35–0.85), respectively. **Conclusions:** Sixty percent of stroke survivors have a favorable cognitive outcome. Lower age and lower MTLA grade on MRI were associated with favorable outcome.

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Introduction

Contrary to the linear increase seen in cognitive changes in an aging normal population, stroke survivors appear to follow different trajectories, with improved, accelerated, or persistent cognitive decline. Particularly in patients with minor stroke or transient ischemic attack (TIA), the frequency of progression is unclear. The Oxford Vascular Study showed that the 5-year risk of dementia was associated with factors such as age, previous stroke, event severity, baseline cognition, low education, and leucoaraiosis [1, 2]. Long-term cognitive transitions in the years after stroke and factors associated with these changes are not fully understood, as only a few studies have investigated the mechanisms and risk factors [3–8]. The lack of validated diagnostic criteria for post-stroke cognitive impairment (CI), heterogeneity of underlying cerebrovascular pathology, and a wide range of clinical manifestations and findings on neuroimaging contribute to a complex picture.

A recent review stated that delayed-onset post-stroke dementia is associated with small vessel disease and to a lesser extent degenerative pathology or recurrent stroke [9]. As many risk factors for vascular CI are modifiable, and several intervention trials with different approaches have shown promising results [10–15], identifying the combinations of pathologies may facilitate personalized treatment and optimal prevention [16]. In order to address a patients prognosis after stoke or TIA, more long-term follow-up data are needed, as outcomes may change over time [17].

During 2007–2008, all patients with first-ever stroke or TIA admitted to Bærum Hospital were invited to participate in a study on post-stroke cognitive functioning, and after 12 months, dementia and mild CI (MCI) were diagnosed and subclassified according to proposed underlying etiology. In total, 20% developed dementia and 37.5% MCI during the first-year post-stroke. Of these, 13% were subclassified as degenerative cognitive disease, 32% as vascular

cognitive disease, and 54% as mixed degenerative and vascular cognitive diseases [18]. We have conducted a 7-year follow-up of the same cohort, and to our knowledge, no study has subclassified MCI 7 years after stroke. In this follow-up study, the aim was to assess the prevalence and etiological subclassification of MCI and dementia 7 years after the index stroke or TIA. Further, we wished to identify predictors associated with a favorable cognitive outcome after 7 years.

Materials and Methods

Participants

All patients with a first-ever stroke or TIA admitted to the stroke unit, Bærum Hospital during 2007/2008, were invited to participate in the CAST (cognition after stroke) study. Bærum Hospital serves 2 counties with a total population of 160,000 inhabitants, and all stroke patients are admitted directly to the stroke unit. Patients with subarachnoid hemorrhage, dementia, or MCI diagnosed before the stroke, patients who did not speak Norwegian and patients with a remaining life expectancy of <1year estimated by the treating physician were excluded. The Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE) [19] was answered by the patient's spouse, a first-degree relative, or a close friend. A score of ≥3.44 on the IQCODE indicated prestroke cognitive decline, and the patient was excluded. From February 2014 to July 2016, surviving participants were invited to participate in the present follow-up study.

Examinations and Assessments

At baseline, vascular risk factors comprising treated hypertension prestroke, hyperlipidemia (total cholesterol >5.0 mmol/L, low-density lipoprotein-cholesterol >3.0 mmol/L), diabetes mellitus (an established diagnosis or hemoglobin A1C ≥7.0), atrial fibrillation (permanent or paroxysmal), current smoking, and daily alcohol intake were recorded. Neurological impairment was assessed using the National Institutes of Health Stroke Scale

(NIHSS) [20]. Patients underwent neuroimaging with computed tomography or magnetic resonance imaging (MRI). Stroke etiology was classified according to the Trial of ORG 10172 classification [21] and functional outcome according to the modified Rankin Scale [22] at baseline. Activities of daily living were assessed by the Barthel Activities of Daily Living Index [22].

Fasting blood samples, NIHSS, modified Rankin Scale, Barthel Index, electrocardiography, blood pressure, and heart rate were collected at baseline and after 12 months and 7 years. At the same time points, waist and hip circumferences, weight and height were measured, and body mass index calculated. Self-reported smoking habits, alcohol use, physical activity, and current medication were recorded.

Cognitive tests at baseline, 12 months post-stroke and at 7 years follow-up included the Mini Mental State Examination [23], the clock drawing test [24], the Trail making tests A and B [25], and the 10-word memory test [26]. Additional tests at the 7-year follow-up were the Montreal Cognitive Assessment [27] and Controlled Oral Word Association Test [28].

Supplementary investigations at 12 months and 7 years included MRI of the brain, color duplex of the precerebral arteries, and when possible, a lumbar puncture for examination of cerebrospinal fluid (CSF) biomarkers for neurodegenerative disease (tau-protein, phosphorylated tau-protein, and beta-amyloid).

Cerebral MRI

The MRI was evaluated for focal vascular lesions, medial temporal lobe atrophy (MTLA), and white matter lesions (WMLs), by 2 radiologists, blinded to the clinical information. Any discrepancies were resolved by consensus. MTLA was graded from 0 to 4; with MTLA grade 0 = no atrophy, MTLA 4 = highest degree of atrophy. MTLA 0–1 is considered normal [29]. WML was rated using the visual rating scale proposed by Fazekas, scores ranging from 0 to 3 [30].

Outcomes and Diagnosis of Cognitive Function

At 1 and 7 years post-stroke, the diagnoses of dementia and MCI were made in consensus meetings by 2 senior neurologists (B.F. and B.T.) and 1 senior geriatrician (A.R.Ø.), using the criteria outlined by Winblad et al. [31] for MCI and the International Classification of Diseases 10th revision criteria [32] for dementia. The evaluations were based on the results from the medical history, cognitive assessments, the IQCODE, and information regarding daily functioning. Subclassification for proposed underlying etiology was based on MRI findings of vascular or degenerative brain changes, biomarkers in the CSF, the patient's vascular risk factors, and clinical cognitive profile. Patients were classified with possible vascular disease when the radiological findings revealed WMLs without MTLA, while MTLA without WMLs was interpreted as degenerative origin. Patients with combined pathologies were classified with mixed vascular and degenerative disease. Details of the novel method for subclassification have previously been reported [18], with 6 potential subgroups; degenerative MCI or degenerative dementia, vascular MCI or vascular dementia or mixed degenerative and vascular MCI or dementia. After 7 years, a favorable cognitive outcome was defined as normal cognitive function (including going from dementia to normal or MCI) or stable MCI with exclusion of those who had progressed from normal to MCI. An unfavorable outcome was attributed to patients with stable dementia, progression from normal cognition to MCI or dementia, progression from MCI to dementia, and to those who died during the follow-up period.

Statistics

Baseline and 12 months characteristics are given as mean ± SD or as number and percentages as appropriate. Categorical variables were compared with Pearson's chi-square test and continuous variables with independent Student *t* test. The study population was dichotomized into the 2 groups, favorable or unfavorable cognitive outcome. In order to identify predictors of a favorable outcome, logistic regression models were used. Variables with a *p* value ≤0.1 in univariate analyses were considered for inclusion in the multivariable models. The

significance level was set at $p < 0.05$. Statistical analyses were performed using SPSS Statistic version 23.

Ethics

The study was approved by the Regional Committee for Ethics in Medical Research and by the Data Protection Authorities (2013/1829). Written informed consent was obtained from all patients; if the patient was cognitively impaired, relatives also gave their written assent.

Results

Study Population

Of the 208 patients included, 184 patients completed the 12-month follow-up, and 109 patients were followed for 7 years post-stroke. In all, 80 patients died during the study period, and 19 refused further participation. A flow chart is presented in Figure 1. Baseline characteristics and 7 year assessments are presented in Table 1.

Prevalence and Subclassification of CIs at Seven Years

At the 7-year follow-up, 40 patients (37%) were diagnosed with MCI and 24 (22%) with dementia, while 45 (41%) had normal cognition. Of the 64 patients diagnosed with CI, 9 (14%) were subclassified with degenerative cognitive disease, 13 (20%) with vascular disease, and 42 (66%) had mixed cognitive disease.

Cognitive changes in the study population between normal, MCI, or dementia from 1 to 7 years are illustrated in Figure 2. In total, 11 of the 109 patients improved their cognitive function, which 2 went from dementia to normal cognition, 64 were unchanged, and 33 deteriorated. Six out of 11 patients with better cognitive function from 1 to 7 years had pure vascular cognitive disease diagnosed at 1 year. Three out of 6 patients diagnosed with degenerative MCI 12 months post-stroke had stable cognition 6 years later.

Predictors for a Favorable Outcome at Seven Years

Baseline characteristics, clinical diagnosis, and MRI finding at 12 months for patients in the predefined groups of favorable versus unfavorable outcome are presented in Table 2. In all, 60% (65) of 109 patients had a favorable outcome after 7 years. Patients with a favorable outcome at 7 years were more often considered to have normal cognition after 1 year, were more often subclassified with pure vascular disease, and had less neuropathological changes on MRI. In total, 7 of the included patients had recurrent stroke between 1 and 7 years, 4 with favorable outcome, and 3 with unfavorable outcome at 7 years. Eight patients had recurrent TIA, 3 with favorable outcome, and 5 with unfavorable outcome.

A selection of baseline and 12 months variables were analyzed by in a logistic regression model (Table 3). In the univariate models, lower age, hyperlipidemia, higher body mass index, lower NIHSS score, lower Fazekas score, lower MTA grade, higher Mini Mental State Examination score, and normal cognition at 12 months were associated with a favorable outcome after 7 years, whereas living alone and cardio embolic stroke etiology were associated with an unfavorable outcome. In the multivariable regression model, only younger age and lower MTLA grade on MRI at 12 months were independently associated with a favorable outcome at 7 years, OR 0.94 (95% CI 0.90–0.98), and 0.55 (95% CI 0.35–0.85), respectively.

Discussion

In this cohort of 109 stroke or TIA survivors, 37% were diagnosed with MCI, 22% with dementia, and 41% had normal cognition after 7 years. Of the 64 patients diagnosed with CI, 9 were subclassified with degenerative cognitive disease, 13 with vascular disease, and 42 had mixed cognitive disease. Eleven patients improved their cognition during follow-up; 6 of these had pure vascular disease at 1 year. About 60% had a favorable cognitive outcome at 7 years, associated with lower age and lower MTLA grade on MRI at 12 months.

The prevalence of delayed post-stroke dementia observed by others varies from 4.4 to 23.9% (mean study duration 2–4 years) [9]. The prevalence of MCI beyond the first years is not known, but a systematic review and meta-analysis, including 16 studies with use of 6 different criteria for neurocognitive disorders and with up to 18 months follow-up, found that 36.4% of patients surviving stroke experienced post-stroke mild neurocognitive disorders (mild NCD) and 16.5% major NCD [33]. The prevalence of MCI and dementia among survivors in our study is in line with findings form a recently published study that followed 151 patients for 3 years after minor stroke (defined as NIHSS <8) [34]. After 3 years, 21% had dementia, 23% had MCI, and 56% had normal cognition. The study differs from our study with respect to the use of Addenbrooke's Cognitive Examination-Revised [35], predefined cutoff values for diagnoses, and shorter follow-up time. With the introduction of the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) in 2013, the term "neurocognitive disorders," with a distinction between "mild" and "major" was established. In DSM-5, impairment in any cognitive domain, including executive function, is sufficient for the diagnosis of a neurocognitive disorder, and the memory domain is no longer hierarchically prominent [36]. We used the criteria outlined by Winblad et al. [31], emphazising memory deficits in particular. In future studies, it is possible that more stroke survivors will meet the criteria for mild NCD as other domains than memory might be affected first. The proposed VASCOG criteria [37], categorizing cognitive disorders of vascular etiology, are in line with DSM-5, with the use of at least one domain criterion. Barbay et al. [38] showed that the use of VASCOG criteria resulted in a high false-positive rate in the GRECOG-VASC (Groupe de Réflexion pour l'Évaluation Cognitive Vasculaire) cohort. The reliability of the VASCOG criteria is being examined in the ongoing Norwegian CI after stroke study [39].

To our knowledge, no other study has subclassified MCI several years after stroke according to proposed underlying etiology. As expected, we found an increasing fraction of mixed cognitive disease with increasing time since the index event, as both neurodegenerative

and vascular pathologies increase with age [40, 41]. In a 4-year follow-up study of 150 patients published in 2004, 41 survivors were diagnosed with dementia during follow-up, and of these, 63.4% met the criteria for probable VaD and 36.6% for possible Alzheimer Disease [7]. As previously pointed out, different diagnostic criteria were applied also in this study [42, 43].

Our results need to be interpreted with caution, as the diagnostic accuracy in most cognitive tests is low, and the total and clinical evaluation might to some extent be person-dependent [44]. Of the 51 patients subclassified at both 1 and 7 years, 4 patients had to be reclassified with respect to pathology. This substantiates the diagnostic work from our consensus group, as the subclassification was based on MRI findings of vascular and degenerative changes in the brain, biomarkers in CSF when available, patients' clinical profile and vascular risk factors without clear diagnostic criteria or cognitive score thresholds. Eleven patients were found to have better cognitive function after 7 years compared to after 12 months. When looking into medical records, the 2 patients moving from vascular dementia to normal cognition were under 70 years of age when included, lived together with spouse, had optimal secondary prevention, and seemed to have rich and stimulating lives. Due to small numbers and the heterogeneous cohort, no statistics were made on this group, but we observed that 6 out of 11 had pure vascular pathology at 12 months. As many risk factors for vascular CI are modifiable, and most stroke patients have several potential modifiable risk factors, our results are promising. As stated in the review paper based on the proceeding of the International Congress on Vascular Dementia, Ljubljana, 2015 [45], stroke patients follow different cognitive trajectories that may change over time. This is in line with our findings.

In our study, patients with a favorable cognitive outcome seem to have less severe strokes, expressed by NIHSS, lower age, and better scores on cognitive test at baseline. Further, those with favorable outcome had less white matter changes and less MTLA on their MRI scans at 12 months. Our findings are in line with the longitudinal Stroke Registry Investigating Cognitive Decline Study [5], but in that study patients who died during follow-up

were excluded from the final analysis and also with findings from the Oxford Vascular Study (Lancet Neurology, in press).

Only age and lower MTLA grade on MRI at 12 months remained independently associated with a favorable outcome in our study. MTLA is a marker of neurodegeneration, strongly associated with Alzheimer disease [46], and after a cerebrovascular event, preclinical stages might become clinically apparent [47]. Autopsy studies have shown that stroke survivors with dementia have more neurodegenerative pathology compared to survivors without dementia [4]. The absence of underlying neurodegeneration could be seen as a part of brain resilience, a factor that may help to preserve cognition after a cerebrovascular event. Two recently published population-based cohort studies looking at predictors of reversion from MCI to normal or stable MCI found that the progressors had a profile broadly typical of Alzheimer's disease, while the stable had other profiles, including vascular morbidity [48, 49].

Strengths and Limitation

Trajectories as well as predictors must be interpreted with caution as we do not know the cognitive function after 12 months in patients who died before follow-up, and we have a limited sample size. However, the fact that our cohort has been followed by the same team of nurses and physicians, and all diagnoses are based on consensus, strengthening our findings. Studies of long-term prognosis after stroke are scarce, and in particular those including a cognitive subclassification, so our study adds important clinical knowledge.

Conclusion

Sixty percent of long-term stroke survivors have a favorable cognitive outcome. Lower age and lower MTLA grade on MRI at 12 months were associated with a favorable outcome. Our findings should be confirmed in larger studies using updated and validated diagnostic criteria.

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Disclosure Statement

The authors declare that there is no conflict of interest.

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Author Contributions

B.F., B.T., A.-R.Ø, He.I.-H., T.B.W., and G.H.: researched literature and conceived the study. He.I.-H., B.F., B.T., and G.H.: were involved in the protocol development and gaining ethical approval. Hå.I.-H., He.I.-H., and G.H.: were involved in patient recruitment. G.H. wrote the first draft of the manuscript. All authors contributed to data analysis, drafting and revising the article, gave final approval of the version to be published, and agree to be accountable for all aspects of the work.

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Appendix after References (Editorial Comments)

Legend(s)

Fig. 1. Patient flow chart.

Fig. 2. Cognitive changes in the study population between normal, MCI, or dementia from 1 to 7 years. MCI, mild cognitive impairment.

Table(s)

Footnote(s)

Table 1. Baseline characteristics (*n* = 208) and assessments at 7 years (*n* = 109)

Male, *n* (%)	105 (51)	44 (40)
Age, years mean (SD)	*72.0 (12.2)*	74.5 (13.1)
Less than 9 years of education (%)	50 (24)	
Stroke subtype (%)		
Cerebral infarction	164 (79)	
TIA	28 (14)	
Cerebral hemorrhage	16 (7)	
Risk factors, *n* (%)		
Hypertension*	123 (59)	
Hyperlipidemia	117 (56)	
Diabetes	23 (11)	
Cigarette smoking (present)	18 (18)	
Coronary heart disease	45 (22)	
Atrial fibrillation	65 (31)	
Daily alcohol use	20 (24)	
BMI >25	*119 (57)*	
TOAST classification, *n* (%)		
Large-vessel disease	21 (10)	
Cardio-embolic disease	60 (29)	
Small-vessel disease	64 (31)	
Stroke of undetermined etiology	63 (30)	
Assessments, mean (SD), *n*		
NIHSS	*2.44 (4.6)*	1.02 (2.3)
BI	*17.5 (5.1)*	18.6 (3.7)
mRS	*1.5 (1.4)*	1.4 (1.2)
IQCODE	*3.1 (0.2) (208)*	
MMSE	*26.0 (4.5) (195)*	25.8 (5.9) (208)
TMT-A	73.7 (66.7) (177)	40.1 (39.6) (101)
TMT-B	152.8 (89.8) (152)	126.4 (67.7) (82)
10-Word test immediate recall	17.9 (2.9) (170)	22.5 (8) (106)
10-Word test, delayed recall	4.3 (2.5) (184)	4.7 (3) (105)

* Hypertension, use of BP-lowering drugs at baseline.

TIA, transient ischemic attack; Hyperlipidemia, total cholesterol >5 mmol/L or LDL >3 mmol/L; LDL, low-density lipoprotein; Coronary Heart Disease, previous myocardial infarction or present angina pectoris; BMI, body mass index; TOAST, the trial of org 10,172 in acute stroke treatment classification; NIHSS, National Institute of Health Stroke Scale; BI, Barthel activities of daily living index; mRS, modified Rankin scale; MMSE, mini mental state examination; IQCODE, the informant questionnaire on cognitive decline in the elderly

Table 2. Characteristics at baseline, clinical cognitive profile, and MRI findings at 12 months by 7 years cognitive outcome favorable or unfavorable

	Favorable	Unfavorable	*p* value
Number	65	119	
Male, *n* (%)	36 (55)	60 (50)	0.620
Age, years, mean (SD)	64.8 (10.3)	*77.24 (10)*	<0.000
Less than 9 years of education, *n* (%)	10 (15)	32 (27)	0.083
Living alone, *n* (%)	15 (23)	46 (37)	0.025
Stroke subtype, *n* (%)			0.322
Cerebral infarction	49 (75)	98 (82)	
TIA	11 (17)	11 (9)	
Cerebral hemorrhage	5 (8)	10 (8)	
Risk factors, *n* (%)			
Hypertension*	34 (52)	73 (61)	0.302
Hyperlipidemia	43 (66)	60 (50)	0.057
Diabetes	5 (8)	17 (14)	0.280
Cigarette smoking (present)	12 (18)	29 (24)	0.462
Coronary heart disease	8 (12)	17 (15)	0.881
Atrial fibrillation	15 (23)	43 (25)	0.131
Daily alcohol use	11 (17)	23 (19)	0.304
BMI, kg/m^2, mean (SD)	26.3 (3.5)	*25.0 (4.4)*	0.048
TOAST classification, *n* (%)			0.198
Large-vessel disease	8 (12)	12 (10)	
Cardio-embolic disease	12 (18)	39 (33)	
Small-vessel disease	23 (30)	32 (27)	
Stroke of undetermined etiology	22 (29)	36 (30)	
Topography, *n* (%)			0.785
Right hemisphere	23 (35)	*48 (40)*	
Left hemisphere	33 (51)	*57 (87)*	
Cerebellum/brainstem	9 (14)	*14 (12)*	
Assessments, mean (SD), *n*			
NIHSS	1.18 (2.33) (65)	*3.73 (5.5) (118)*	0.003
BI	19.26 (2.5) (65)	*16.2 (6.1) (116)*	<0.000
mRS	1.0 (1.1) (65)	*1.8 (1.5) (119)*	<0.000
IQCODE	3.1 (0.1) (65)	*3.1 (0.2) (119)*	0.306
MMSE	27.4 (3.2) (65)	*25.0 (5.6) (107)*	0.001
Clinical profile 12 months			
Dementia	4 (6)	30 (25)	0.001
MCI	25 (38)	40 (34)	1.0
Normal cognition	36 (55)	34 (28)	0.006
MRI 12 months, mean (SD), *n*			
MTLA grade	0.75 (0.9) (65)	1.95 (1.3) (95)	<0.000
Fazekas score	1.57 (0.8) (60)	2.29 (0.8) (95)	<0.000
Global cortical atrophy	1.32 (0.6) (60)	1.94 (0.7) (93)	<0.000

* Hypertension, use of BP-lowering drugs at baseline.

TIA, transistent ischemic attack; Hyperlipidemia, total cholesterol >5 mmol/L or LDL >3 mmol/L; LDL, low-density lipoprotein; Coronary Heart Disease, previous myocardial infarction or present angina pectoris; BMI, body mass index; TOAST, the trial of org 10,172 in acute stroke treatment classification; NIHSS, National Institute of Health Stroke Scale; BI, Barthel activities of daily living index; mRS, modified Rankin scale; IQCODE, the informant questionnaire on cognitive decline in the elderly; MMSE, mini-mental state examination; MTLA, medial temporal lobe atrophy.

Table 3. Logistic regression; predictors for a favorable outcome at 7 years

	Univariate			Multivariate		
	OR	95% CI	*p* value	OR	95% CI	*p* value
Baseline						
Age, years	0.89	0.86–0.92	<0.001	0.94	0.90–0.98	0.015
Sex, male	0.82	0.45–1.50	0.819			
Living alone	2.10	1.06–4.17	0.034	1.26	0.45–3.53	0.656
Hyperlipidemia	1.92	1.03–3.60	0.041	1.27	0.51–3.19	0.611
BMI, kg/m^2	1.08	1.01–1.18	0.051	0.99	0.88–1.12	0.914
NIHSS	0.85	0.75–0.96	0.009	0.89	0.72–1.11	0.289
TOAST, cardioembolic	0.45	0.22–0.94	0.033	1.04	0.35–2.94	0.989
MMSE	1.19	1.07–1.33	0.002	0.99	0.87–1.14	0.938
12 months						
Fazekas	0.37	0.25–0.56	<0.001	0.64	0.38–1.08	0.096
MTLA grade	0.40	0.29–0.57	<0.001	0.55	0.35–0.85	0.008
Normal cognition	2.56	1.15–4.84	0.004	0.88	0.35–2.20	0.782

BMI, body mass index; NIHSS, National Institute of Health Stroke Scale; TOAST, the trial of org 10,172 in acute stroke treatment classification; MMSE, mini-mental state examination; MTLA, medial temporal lobe atrophy.

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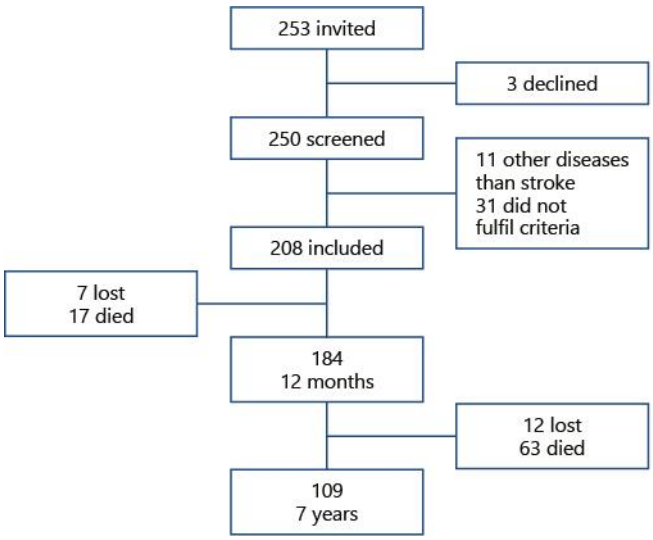

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