Prevalence of Early Dementia After First-Ever Stroke
A 24-Year Population-Based Study

Yannick Béjot, MD; Corine Aboa-Eboulé, MD; Jérôme Durier, MD; Olivier Rouaud, MD; Agnès Jacquin, MD; Eddy Ponavoy, MD; Dominique Richard, MD; Thibault Moreau, MD; Maurice Giroud, MD

Backgound and Purpose—No data about temporal change in the prevalence of poststroke dementia are available. We aimed to evaluate trends in the prevalence of early poststroke dementia.

Methods—From 1985 to 2008, overall first-ever strokes occurring within the population of the city of Dijon, France (150 000 inhabitants) were recorded. The presence of dementia was diagnosed during the first month after stroke, according to Diagnostic and Statistical Manual of Mental Disorders, Third and Fourth Editions criteria. Time trends were analyzed according to 4 periods: 1985 to 1990, 1991 to 1996, 1997 to 2002, and 2003 to 2008. Logistic regression was used for nonmultivariate analyses.

Results—Over the 24 years, 3948 first-ever strokes were recorded. Among patients with stroke, 3201 (81%) were testable of whom 653 (20.4%) had poststroke dementia (337 women and 316 men). The prevalence of nontestable (mostly due to death) patients declined from 28.0% to 10.2% ($P<0.0001$). Multivariate analysis revealed significant temporal changes in the prevalence of poststroke dementia; prevalence in the second and fourth periods was, respectively, almost half and twice that in the first period. The prevalence of poststroke dementia associated with lacunar stroke was 7 times higher than that in intracerebral hemorrhage but declined over time as did prestroke antihypertensive medication. Age, several vascular risk factors, hemiplegia, and prestroke antiplatelet agents were associated with an increased prevalence of poststroke dementia.

Conclusions—This study covering a period of 24 years highlights temporal changes in the prevalence of early dementia after first-ever stroke. These changes may be explained by concomitant determinants of survival and incidence such as stroke care management or prestroke medication. **(*Stroke*. 2011;42:607-612.)**

Key Words: dementia ■ epidemiology ■ risk factors ■ stroke

Beyond its harmful effect on patients' functional capabilities, stroke is also associated with a higher risk of cognitive impairment and dementia; a history of stroke almost doubles the risk of dementia in the population aged >65 years.[1] The prevalence of poststroke dementia was recently reviewed by Pendlebury and Rothwell.[2] Despite a heterogeneous study design and case mix, the authors concluded that 7% to 23% of patients developed new dementia in the first year after their first-ever stroke. Data were obtained from both hospital- and population-based studies, but all of these studies were of short duration and included small numbers of patients. Hence, none provided reliable data on temporal changes in the prevalence of poststroke dementia. However, such information is essential to determine the impact of acute stroke care management on cognitive function and to provide projections for the burden of poststroke cognitive impairment to design appropriate health policy.

Hence, the aim of this study was to evaluate trends in the prevalence of early poststroke dementia, from 1985 to 2008, from the Dijon Stroke Registry.

Methods

The prevalence of early dementia after stroke was studied in patients included from January 1, 1985, to December 31, 2008, in the Dijon Stroke Registry, Dijon, France. This registry complies with the core criteria defined by Sudlow and Warlow to approach the "ideal" stroke incidence study and provide reliable and comparable results.[3,4] The study population comprised all residents of the city of Dijon (150 000 inhabitants).

Data Collection

Definition of Stroke

Stroke was defined according to World Health Organization recommendations.[5] The clinical diagnoses were validated on the basis of either CT or MRI.[3] Only first-ever symptomatic stroke was considered for this study. The classification of the stroke subtype used since

Received July 2, 2010; final revision received October 5, 2010; accepted October 26, 2010.

From the Dijon Stroke Registry, EA4184 (Inserm and InVS; Y.B., C.A.-E., J.D., A.J., M.G.), IFR 100 (STIC-Santé), Faculty of Medicine, University of Burgundy and University Hospital of Dijon, France; and Centre Mémoire (Y.B., O.R., E.P., D.R., T.M., M.G.), Ressources et Recherche, Memory Clinic, University Hospital of Dijon, Dijon, France.

Correspondence to Yannick Bejot, MD, Dijon Stroke Registry, Service de Neurologie, Hôpital Général, 3 Rue du Faubourg Raines, 21033 Dijon, France. E-mail ybejot@yahoo.fr

Stroke **is available at http://stroke.ahajournals.org** **DOI: 10.1161/STROKEAHA.110.595553**

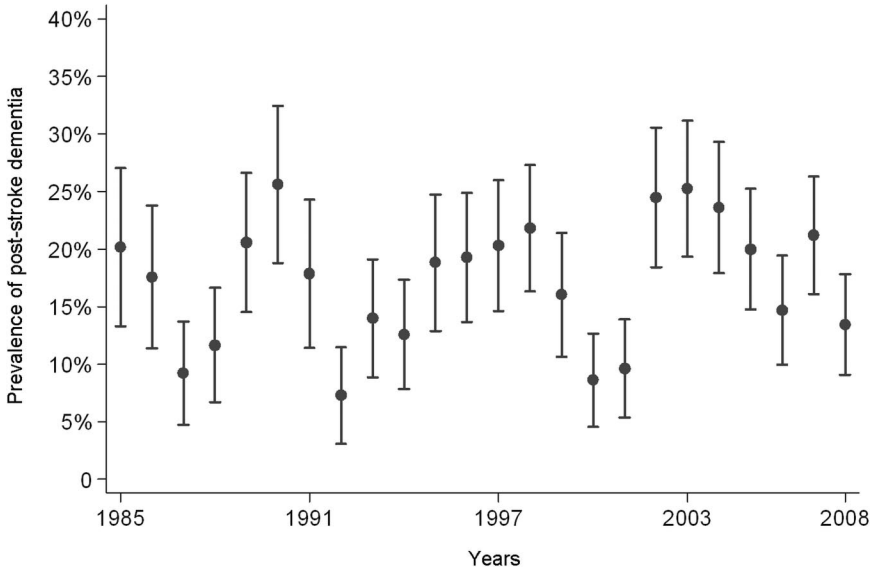

Figure. Annual prevalence of poststroke dementia. Vertical bars represent 95% CI.

1985 was as follows: (1) lacunar infarct; (2) ischemic stroke from cardiac embolism; (3) nonlacunar noncardioembolic ischemic strokes; (4) spontaneous intracerebral hemorrhage; and (5) subarachnoid hemorrhage.[3]

Case Ascertainment

Information was provided by multiple overlapping sources[3]: (1) from the emergency rooms and all of the clinical and radiological departments of Dijon University Hospital with the diagnosis of stroke made by a neurologist; (2) from the 3 private hospitals of the city with diagnosis made by private neurologists; (3) from the patient's home or from the nursing homes of the city with diagnosis assessed by the general practitioners with the help of an outpatient clinic with either a public or private neurologist; (4) from the 3 private radiological centers to identify missed cases; (5) from the Doppler ultrasound centers of the University Hospital and private centers; and (6) from the death certificates obtained from the local Social Security Bureau that is responsible for registering all deaths in the community to identify fatal strokes occurring in nonhospitalized patients. All of the collected death certificates were checked by a member of our team.

Collection of Vascular Risk Factors

Vascular risk factors were collected with the same methodology over the whole study period.[3] We recorded hypertension if high blood pressure was noted in a patient's medical history (either self-reported or from medical notes) or if a patient was under antihypertensive treatment. Diabetes mellitus was recorded if a glucose level of $\geq$7.8 mmol/L had been reported in the medical record or if the patient was under insulin or oral hypoglycemic agents. Hypercholesterolemia was considered if a total cholesterol level $\geq$5.7 mmol/L was reported in the medical history of the patient or if patients were treated with lipid-lowering therapy. We also recorded a history of atrial fibrillation, previous myocardial infarction, and a history of transient ischemic attack. Smoking was not included in the final analysis because of 15% of missing data.

Assessment of Poststroke Dementia

Given the design of our registry and its inherent constraints, the evaluation of cognitive function was performed by a neurologist in the early stages of the stroke, that is, within the first month after stroke onset to collect as much information as possible. Patients and their relatives attended an interview and the diagnosis of poststroke dementia was based on a simple standardized clinical approach using Diagnostic and Statistical Manual of Mental Disorders, Third and Fourth Editions.[6,7] Therefore, the diagnosis of dementia was made if, according to the informant and the examination of the patient, multiple cognitive deficits manifested by memory impairment associated with $\geq$1 cognitive disturbances, including language disturbance, apraxia, agnosia, or disturbance in executive function, with significant impairment in social or occupational function representing a significant decline from a previous level of function were noted.

Statistical Analysis

The prevalence of poststroke dementia was calculated as the ratio of the number of patients with poststroke dementia to the total number of first-ever strokes in testable patients. A 95% CI was computed for all estimates. We plotted data per year to graphically assess trends in the prevalence of poststroke dementia over 24 years. Due to a multimodal variation of prevalence over the years (Figure), the time period was split into 4 intervals of 6 years: 1985 to 1990, 1991 to 1996, 1997 to 2002, and 2003 to 2008, with 1985 to 1990 as the reference. Baseline characteristics for analyses were demographics, vascular risk factors, prestroke treatments, and clinical features. Data on statins were analyzed by univariate analysis only because they had only been recorded in the registry since 2006. Proportions, ORs, and means of baseline characteristics were compared between patients with and without poststroke dementia using the χ^2 test, logistic regression, and analysis of variance, respectively. Univariate associations among age, sex, time periods, vascular risk factors, prestroke treatments, stroke subtypes, clinical features at onset, and poststroke dementia were analyzed using logistic regression to estimate unadjusted ORs. In multivariate analyses, we introduced into the models age, sex as well as all potential confounders with a probability value $<$0.20 in univariate analysis. We investigated whether there were nonlinear trends over the study periods (unequal prevalence over study periods) for age, prestroke treatment, stroke subtypes, vascular risk factors, and clinical features as observed for the prevalence of poststroke dementia. A likelihood ratio test was performed to examine whether the fits of multivariate nested models were improved by including statistical interaction terms of study periods with baseline characteristics. None of the statistical interaction terms were significant except for lacunar subtype with study periods ($P<0.0001$) and the prior antihypertensive treatment with study periods ($P=0.0061$), which were taken into account along with the main effects in the final multivariate models. Probability values $<$0.05 were considered statistically significant. Statistical analysis was performed with STATA 10.0 software (StataCorp LP, College Station, TX).

Ethics

Our registry was approved by the National Ethics Committee and the French Institute for Public Health Surveillance.

Results

Over the whole study period, 3948 patients with first-ever stroke were recorded (1842 in men, 2106 in women). Among patients with stroke, 3201 (81%) were testable and included in the analyses. The inability to test the cognitive status of patients (n=747 [19%]) was due to either severe aphasia (n=66 [8.7%]) or death (n=681 [91.3%]) in the acute phase of stroke. The prevalence of nontestable patients decreased with study periods from 28.0% in 1985 to 1990 to 24.7% in 1991 to 1996, 15.5% in 1997 to 2002, and 10.2% in 2003 to 2008 ($P<0.0001$). Nontestable patients were older (79.1±12.5 years) than testable patients (72.9±14.7; $P<0.0001$) with an increasing age over the 4 study periods (respectively, 77.4±11.7, 78.6±12.5, 81.1±12.5, and 81.3±13.0 years; $P=0.004$) contrary to testable patients (respectively, 72.0±14.6, 73.6±13.9, 72.8±14.9, and 73.0±15.0 years; $P=0.261$). They had a more severe initial presentation, more often prior transient ischemic attack, myocardial infarction, or anticoagulant therapy, but they had a lower prevalence of hypercholesterolemia and prior antiplatelet medication than did testable patients ($P<0.05$).

Among testable patients, 653 (20.4%) had poststroke dementia (337 women and 316 men). The annual prevalence distribution of poststroke dementia roughly showed a trimodal curve with 3 peaks, in 1990 (25.6%), 1998 (21.8%), and 2003 (25.2%), with varying falls in prevalence in between (Figure). There was no significant temporal change ($P=0.123$) in the prevalence of poststroke dementia over the 4 study periods with, respectively, 23.7% (95% CI, 20.4% to 27%), 19.3% (16.4% to 22.3%), 19% (16.4% to 21.6%), and 20.2% (17.8% to 22.7%). Patients with poststroke dementia differed from those without poststroke dementia. They were older (77.3±10.8 years versus 71.7±15.4 years, $P=0.0001$); had a higher prevalence of several vascular risk factors, including hypertension, diabetes, atrial fibrillation, previous myocardial infarction, and history of transient ischemic attack; and were more likely to have received stroke-preventive medications, including antiplatelet agents and antihypertensive treatment (Table 1). In addition, they were more likely to have hemiplegia at admission, and there was a higher prevalence of lacunar stroke but a lower prevalence of intracerebral hemorrhage, subarachnoid hemorrhage, and nonlacunar noncardioembolic ischemic strokes in these patients (Table 1). The prestroke use of anticoagulants and antiplatelet agents in the whole study population showed linear trends from, respectively, 2.8% and 5.8% in the first period to 9.2% and 25.7% in the last period ($P<0.0001$). However, the use of antihypertensive treatment showed a nonlinear trend with 48.1%, 53.4%, 51.0%, and 45.6% over the 4 periods ($P=0.003$).

Multivariate analyses revealed a significant temporal change in the prevalence of poststroke dementia in the second (1991 to 1996) and fourth periods (2003 to 2008) as compared with the first period (1985 to 1990). The prevalence in the second period was almost half that in the first, whereas prevalence in the fourth period was twice that in the first. Prevalence in the third period was the same as that in the first ($P<0.0001$). Several variables remained significant in the multivariate model (Table 2). Age correlated strongly with poststroke dementia with a relative 3% increase for each additional year of age. The prevalence of poststroke dementia in patients with lacunar stroke was 7 times that in patients with intracerebral hemorrhage. Stratum-specific analyses by periods revealed a significant temporal decrease in the prevalence of poststroke dementia among patients who had lacunar stroke compared with those who did not. Hypertension, previous myocardial infarction, hemiplegia at onset, and prestroke treatment by antiplatelet agents were all associated with an increased prevalence of poststroke dementia. In contrast, the prevalence of poststroke dementia in patients with hypercholesterolemia was 24% lower than that in patients without hypercholesterolemia. Overall, antihypertensive treatment was not associated with poststroke dementia. However, stratum-specific analyses by periods revealed a significant temporal decrease in the risk of poststroke dementia over the last 2 periods (1997 to 2002 and 2003 to 2008) among patients who had been treated with prior antihypertensive medication compared with those who had not.

Discussion

This is the first study to evaluate temporal trends of early dementia after stroke over 24 years with constant methodologies for stroke ascertainment and the same definition of dementia throughout the study period.

The prevalence of poststroke dementia observed in this study is similar to that reported in hospital-based studies, including first-ever stroke and prestroke dementia. In their meta-analysis, Pendlebury and Rothwell estimated the general prevalence from such studies at 26.5% (95% CI, 24.3% to 28.7%) irrespective of the delay after stroke onset.[2] In contrast, the only population-based study that included first-ever stroke and did not exclude prestroke dementia found a lower prevalence of 12.5% (95% CI, 5.6% to 19.4%).[8] However, some differences in the methodology applied may account for these discrepancies between the results of this study and ours. In the latter study, cognitive assessment was performed at 1 year, and the patients with stroke were younger than ours. Several other studies have evaluated the prevalence of dementia in the first 3 months after stroke onset. They reported a frequency ranging from 18.7% to 21.2%, which is in line with our results despite their hospital-based setting.[9–13]

Although it seemed to be stable over time, multivariate analysis demonstrated that the prevalence of poststroke dementia changed in the second (1991 to 1996) and fourth (2003 to 2008) periods. Indeed, it was, respectively, half and twice the prevalence of the first period. We investigated whether these trends could also be observed for age, prestroke treatment, stroke subtypes, vascular risk factors, and clinical features. There was no temporal change for age in testable patients, but there was an increasing age over the study periods for nontestable patients. In addition, lacunar stroke and antihypertensive treatment were both associated with a significant temporal decline in the prevalence of poststroke dementia. Theoretical explanations for these results may be changes in the prevalence of prestroke dementia, in the incidence of poststroke dementia, and/or in survival after stroke. The decline in the prevalence of poststroke dementia

Table 1. Characteristics of Patients With and Without Poststroke Dementia

	No Poststroke Cognitive Disorder (N=2548)				Poststroke Cognitive Disorder (N=653)				Poststroke Cognitive Disorder Vs No Poststroke Cognitive Disorder			
	No.	%	95% CI		No.	%	95% CI		OR	95% CI		*P*
Age, years												
<40	119	4.7%	3.9%	5.5%	3	0.5%	0.0%	1.0%	0.09	0.03	0.29	<0.001
40–50	147	5.8%	4.9%	6.7%	9	1.4%	0.5%	2.3%	0.22	0.11	0.44	<0.001
50–60	234	9.2%	8.1%	10.3%	29	4.4%	2.9%	6.0%	0.45	0.30	0.67	<0.001
60–70	424	16.6%	15.2%	18.1%	91	13.9%	11.3%	16.6%	0.79	0.62	1.01	0.064
70–80	751	29.5%	27.7%	31.2%	218	33.4%	29.8%	37.0%	1.21	1.01	1.45	0.038
>80	873	34.3%	32.4%	36.1%	303	46.4%	42.6%	50.2%	1.66	1.40	1.98	<0.001
Male sex	1188	46.6%	44.7%	48.6%	316	48.4%	44.5%	52.2%	1.08	0.91	1.28	0.37
Stroke subtype												
Intracerebral hemorrhage	238	9.3%	8.2%	10.5%	28	4.3%	2.7%	5.8%	0.47	0.32	0.70	<0.001
Subarachnoid hemorrhage	85	3.3%	2.6%	4.0%	3	0.5%	0.0%	1.0%	0.13	0.04	0.42	0.001
Ischemic stroke												
Lacunar	554	21.7%	20.1%	23.3%	333	51.0%	47.2%	54.8%	3.57	2.99	4.27	<0.001
Cardioembolic	394	15.5%	14.1%	16.9%	96	14.7%	12.0%	17.4%	1.01	0.80	1.28	0.94
Nonlacunar noncardioembolic	1210	47.5%	45.5%	49.4%	184	28.2%	24.7%	31.6%	0.43	0.36	0.52	<0.001
Undetermined	67	2.6%	2.0%	3.3%	9	1.4%	0.5%	2.3%	0.57	0.29	1.11	0.10
Vascular risk factors												
Hypertension	1624	63.7%	61.9%	65.6%	483	74.0%	70.6%	77.3%	1.58	1.31	1.91	<0.001
Diabetes	358	14.1%	12.7%	15.4%	114	17.5%	14.5%	20.4%	1.30	1.04	1.63	0.024
hypercholesterolemia	631	24.8%	23.1%	26.4%	139	21.3%	18.1%	24.4%	0.82	0.67	1.01	0.064
Atrial fibrillation	445	17.5%	16.0%	18.9%	147	22.5%	19.3%	25.7%	1.42	1.15	1.75	0.001
Myocardial infarction	403	15.8%	14.4%	17.2%	153	23.4%	20.2%	26.7%	1.61	1.30	1.98	<0.001
TIA	280	11.0%	9.8%	12.2%	104	15.9%	13.1%	18.7%	1.50	1.17	1.91	0.001
Prestroke treatments												
Anticoagulants	158	6.2%	5.3%	7.1%	33	5.1%	3.4%	6.7%	0.79	0.54	1.16	0.24
Antiplatelet agents	501	19.7%	18.1%	21.2%	175	26.8%	23.4%	30.2%	1.52	1.25	1.85	<0.001
Antihypertensive treatment	1225	48.1%	46.1%	50.0%	356	54.5%	50.7%	58.3%	1.26	1.07	1.50	0.007
Statins (2006–2008)	46	10.6%	7.7%	13.6%	5	5.4%	0.7%	10.2%	0.48	0.19	1.25	0.13
Clinical features at onset												
Coma	101	4.0%	3.2%	4.7%	26	4.0%	2.5%	5.5%	1.07	0.70	1.64	0.77
Hemiplegia	1806	70.9%	69.1%	72.6%	522	79.9%	76.9%	83.0%	1.63	1.32	2.01	<0.001
Confusion	259	10.2%	9.0%	11.3%	79	12.1%	9.6%	14.6%	1.19	0.91	1.55	0.21
Left hemisphere lesion	1103	43.3%	41.4%	45.2%	290	44.4%	40.6%	48.2%	1.03	0.87	1.22	0.74

TIA indicates transient ischemic attack.

in the second period (1991 to 1996), as compared with the first period (1985 to 1990), may be explained by the fact that this period could have been a transition period. As observed in the first period, the prevalence of nontestable patients, mainly because of early death, was still high (25%), which consequently decreased the number of prevalent cases that would potentially have had poststroke dementia if they had survived, all the more so because these patients were older, had a more severe clinical presentation, and more vascular risk factors, so were at a high risk of dementia. In parallel, the frequency of prestroke preventive treatments, which are known to decrease the risk of dementia,[14,15] showed a marked increase during this second period and may have contributed to the decrease in both the prevalence of prestroke dementia and the incidence of poststroke dementia. As a consequence, both the low number of prevalent cases and the lower incidence of poststroke dementia may have resulted in a significantly lower prevalence of poststroke dementia.

Conversely, the third (1997 to 2002) and the fourth (2003 to 2008) periods were associated with a lower prevalence of nontestable patients due to a decrease in early case-fatality possible because of better acute stroke management. The consequence may have been a higher number of prevalent cases with poststroke dementia. Moreover, unlike the use of anticoagulants and antiplatelet agents, the use of prestroke hypertensive treatment did not show a linear trend but a decrease during the last 2 periods, which may have contributed to the increase in the prevalence of poststroke dementia.

Table 2. Multivariate Analysis of Predictors of Poststroke Dementia

	OR	95% CI		*P*
Time period				
1985–1990	1.00			
1991–1996	0.52	0.30	0.90	0.018
1997–2002	1.14	0.72	1.81	0.576
2003–2008	1.91	1.25	2.91	0.003
Time period (continuous)	1.03	1.01	1.06	0.003
Age (continuous)	1.03	1.02	1.04	<0.001
Stroke subtype				
Intracerebral hemorrhage	1.00			
Subarachnoid hemorrhage	0.47	0.14	1.60	0.225
Lacunar stroke	7.24	4.13	12.69	0.000
Lacunar stroke (period 1991–1996)	10.01	3.16	31.75	0.000
Lacunar stroke (period 1997–2002)	5.86	1.92	17.86	0.005
Lacunar stroke (period 2003–2008)	1.51	0.50	4.52	0.150
Nonlacunar	1.20	0.80	1.80	0.388
Undetermined	1.17	0.53	2.60	0.701
Male sex	1.17	0.97	1.42	0.098
Vascular risk factors				
Hypertension	1.38	1.05	1.82	0.021
Diabetes	1.26	0.98	1.64	0.074
Hypercholesterolemia	0.76	0.60	0.96	0.023
Atrial fibrillation	1.29	1.02	1.63	0.036
Myocardial infarction	1.35	1.06	1.72	0.016
TIA	1.28	0.97	1.68	0.077
Prestroke treatments				
Antiplatelet agents	1.39	1.11	1.75	0.005
Antihypertensive treatment	1.02	0.66	1.59	0.925
Period 1991–1996	1.20	0.42	3.43	0.078
Period 1997–2002	0.49	0.18	1.31	0.204
Period 2003–2008	0.66	0.25	1.08	0.150
Hemiplegia	1.27	1.01	1.59	0.038

TIA indicates transient ischemic attack.

Several factors were independently associated with the risk of poststroke dementia, including age, hypertension, atrial fibrillation, history of myocardial infarction, or hemiplegia at onset. Such associations have been previously described in the literature but with conflicting results.[9,12,16–18] In their meta-analysis, Pendlebury and Rothwell found that both diabetes and atrial fibrillation but neither hypertension nor previous ischemic heart disease were associated with poststroke dementia.[2] In addition, they identified hemorrhagic stroke as a predictor of poststroke dementia, whereas lacunar stroke was associated with a nonsignificantly higher risk (OR, 0.8; 95% CI, 0.7 to 1.0; $P=0.09$). This contrasts with our findings, which show that lacunar stroke is a strong predictor of poststroke dementia with an OR of approximately 7. This association could be explained by the fact that patients with lacunar stroke frequently have many asymptomatic cerebral ischemic lesions, and it has been demonstrated that silent strokes increase the risk of poststroke dementia. Another explanation could be that these patients are already demented at the time of first clinical manifestation of stroke, but we cannot confirm this assumption because prestroke dementia was not recorded.

Finally, the negative association between hypercholesterolemia and poststroke dementia must be interpreted with caution. Actually, we were not able to include statin treatment in the multivariable analysis because these data were not recorded in our files before 2006, and this treatment may influence the risk of dementia, although a recent meta-analysis suggested that statins are not effective in the prevention of dementia.[19]

The major strength of our study is the use of multiple overlapping sources of information to identify both hospitalized and nonhospitalized fatal and nonfatal strokes. Nevertheless, because no population screening was performed, undiagnosed strokes were not recorded. However, such cases are probably rare and did not affect our results. In addition, despite constant methodology for case ascertainment, it is possible that differences in case-finding effectiveness influenced our findings. The evaluation of poststroke dementia was based on the same criteria throughout the study period. Although Diagnostic and Statistical Manual of Mental Disorders, Third and Fourth Edition criteria are less specific for the diagnosis of vascular dementia than other criteria such as National Institute of Neurological Disorders and Stroke–Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS-AIREN) criteria,[20] the fact that we maintained the same definition of poststroke dementia over the 24 years made it possible to analyze temporal changes in its prevalence.

Because of the design of the study, the cognitive evaluation of patients was performed during the first month after the stroke, which is certainly early. Time from stroke was not entered into our model because these data were not recorded in our files. Data about long-term dementia were not available because active follow-up of patients was not systematically performed over the whole study period. However, several studies have suggested that cognitive disorders after the acute phase of stroke are associated with long-term poststroke dementia, which probably reflects poor cognitive reserve in patients with such impairments.[21,22] Moreover, the primary aim of this study was to evaluate temporal trends in poststroke dementia, and because the methodology of case ascertainment was maintained over time, we can conclude that the changes observed were not biased. In addition, we did not consider the cognitive status of patients before stroke, which may explain why the prevalence of poststroke dementia observed in our study was higher than the 7% to 12% prevalence reported in population-based studies that excluded prestroke demented patients.[23,24]

Another limitation inherent to the use of prevalence as a measurement concerns the generalization of our results. This limitation, called length-biased sampling, stems from the fact that cases of poststroke dementia would overrepresent those who did not die during the first month (testable patients) but underrepresent those who died sooner (nontestable pa-

tients).[25] Lastly, although our study covered most causal assumptions to investigate an etiologic hypothesis with a cross-sectional design (population in a steady state, no selective survival, unchanged mean duration of outcome, no reverse causality, and temporal directionality), we preferred to interpret our measurements of association with caution by using prevalence ORs rather than incidence density ratios.[26]

To conclude, this study highlights temporal changes in the prevalence of early dementia after first-ever stroke over 24 years. These changes may be explained by concomitant determinants of survival and incidence, like stroke care management or prestroke medication.

Acknowledgments

We thank Mr Philip Bastable for reviewing the English.

Sources of Funding

The Dijon Stroke Registry is supported by the French Institute for Public Health Surveillance (InVS) and Inserm.

Disclosures

None.

References

1. Savva GM, Stephan BC; Alzheimer's Society Vascular Dementia Systematic Review Group. Epidemiological studies of the effect of stroke on incident dementia: a systematic review. *Stroke*. 2010;41:e41–e46.
2. Pendlebury ST, Rothwell PM. Prevalence, incidence, and factors associated with pre-stroke and post-stroke dementia: a systematic review and meta-analysis. *Lancet Neurol*. 2009;8:1006–1018.
3. Béjot Y, Rouaud O, Jacquin A, Osseby GV, Durier J, Manckoundia P, Pfitzenmeyer P, Moreau T, Giroud M. Stroke in the very old: incidence, risk factors, clinical features, outcomes and access to resources—a 22-year population-based study. *Cerebrovasc Dis*. 2010;29:111–121.
4. Sudlow CL, Warlow CP. Comparing stroke incidence worldwide: what makes studies comparable? *Stroke*. 1996;27:550–558.
5. World Health Organization. *The World Health Report 2000: Health Systems Improving Performance*. Geneva: WHO; 2000.
6. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (III ed, revised) (DSM-III-R)*. Washington, DC: APA; 1987.
7. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (IV ed) (DSM-IV)*. Washington, DC: APA; 1994.
8. Srikanth VK, Anderson JF, Donnan GA, Saling MM, Didus E, Alpitsis R, Dewey HM, Macdonell RA, Thrift AG. Progressive dementia after first-ever stroke: a community-based follow-up study. *Neurology*. 2004; 63:785–792.
9. Tang WK, Chan SS, Chiu HF, Ungvari GS, Wong KS, Kwok TC, Mok V, Wong KT, Richards PS, Ahuja AT. Frequency and determinants of poststroke dementia in Chinese. *Stroke*. 2004;35:930–935.
10. del Ser T, Barba R, Morin MM, Domingo J, Cemillan C, Pondal M, Vivancos J. Evolution of cognitive impairment after stroke and risk factors for delayed progression. *Stroke*. 2005;36:2670–2675.
11. Klimkowicz A, Dziedzic T, Slowik A, Szczudlik A. Incidence of pre- and poststroke dementia: Cracow stroke registry. *Dement Geriatr Cogn Disord*. 2002;14:137–140.
12. Pohjasvaara T, Erkinjuntti T, Vataja R, Kaste M. Dementia three months after stroke. Baseline frequency and effect of different definitions of dementia in the Helsinki Stroke Aging Memory Study (SAM) cohort. *Stroke*. 1997;28:785–792.
13. Zhou DH, Wang JY, Li J, Deng J, Gao C, Chen M. Study on frequency and predictors of dementia after ischemic stroke: the Chongqing stroke study. *J Neurol*. 2004;251:421–427.
14. Peters R, Beckett N, Forette F, Tuomilehto J, Clarke R, Ritchie C, Waldman A, Walton I, Poulter R, Ma S, Comsa M, Burch L, Fletcher A, Bulpitt C; HYVET investigators. Incident dementia and blood pressure lowering in the Hypertension in the Very Elderly Trial cognitive function assessment (HYVET-COG): a double-blind, placebo controlled trial. *Lancet Neurol*. 2008;7:683–689.
15. Deschaintre Y, Richard F, Leys D, Pasquier F. Treatment of vascular risk factors is associated with slower decline in Alzheimer disease. *Neurology*. 2009;73:674–680.
16. Inzitari D, Di Carlo A, Pracucci G, Lamassa M, Vanni P, Romanelli M, Spolveri S, Adriani P, Meucci I, Landini G, Ghetti A. Incidence and determinants of poststroke dementia as defined by an informant interview method in a hospital-based stroke registry. *Stroke*. 1998;29:2087–2093.
17. Barba R, Martínez-Espinosa S, Rodríguez-García E, Pondal M, Vivancos J, Del Ser T. Poststroke dementia: clinical features and risk factors. *Stroke*. 2000;31:1494–1501.
18. Censori B, Manara O, Agostinis C, Camerlingo M, Casto L, Galavotti B, Partziguian T, Servalli MC, Cesana B, Belloni G, Mamoli A. Dementia after first stroke. *Stroke*. 1996;27:1205–1210.
19. McGuinness B, Craig D, Bullock R, Passmore P. Statins for the prevention of dementia. *Cochrane Database Syst Rev*. 2009;2:CD003160.
20. Wetterling T, Kanitz RD, Borgis KJ. Comparison of different diagnostic criteria for vascular dementia (ADDTC, DSM-IV, ICD-10, NINDS-AIREN). *Stroke*. 1996;27:30–36.
21. Rasquin SM, Lodder J, Visser PJ, Lousberg R, Verhey FR. Predictive accuracy of MCI subtypes for Alzheimer's disease and vascular dementia in subjects with mild cognitive impairment: a 2-year follow-up study. *Dement Geriatr Cogn Disord*. 2005;19:113–119.
22. Nys GM, van Zandvoort MJ, de Kort PL, Jansen BP, de Haan EH, Kappelle LJ. Cognitive disorders in acute stroke: prevalence and clinical determinants. *Cerebrovasc Dis*. 2007;23:408–416.
23. Kokmen E, Whisnant JP, O'Fallon WM, Chu CP, Beard CM. Dementia after ischemic stroke: a population-based study in Rochester, Minnesota (1960–1984). *Neurology*. 1996;46:154–159.
24. Kase CS, Wolf PA, Kelly-Hayes M, Kannel WB, Beiser A, D'Agostino RB. Intellectual decline after stroke: the Framingham Study. *Stroke*. 1998;29:805–812.
25. Rothman KJ, Greenland S, Lash TL. *Modern Epidemiology*. Philadelphia: Lippincott Williams & Wilkins; 2008.
26. Reichenheim ME, Coutinho ES. Measures and models for causal inference in cross-sectional studies: arguments for the appropriateness of the prevalence odds ratio and related logistic regression. *BMC Med Res Methodol*. 2010;10:66.