Risk of fractures after stroke

Results from the Ontario Stroke Registry

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ABSTRACT

Objective: To determine the risk of fractures after stroke.

Methods: Using the Ontario Stroke Registry, we identified a population-based sample of consecutive patients seen in the emergency department or hospitalized with stroke ($n = 23,751$) or TIA ($n = 11,240$) at any of 11 stroke centers in Ontario, Canada, and discharged alive between July 1, 2003, and March 31, 2012. We compared the risk of low-trauma fractures in patients with stroke vs those with TIA using propensity score methods to adjust for differences in baseline factors. Secondary analyses compared fracture risk poststroke with that in age-/sex-matched controls without stroke or TIA ($n = 23,751$) identified from the Ontario Registered Persons Database.

Results: The 2-year rate of fracture was 5.7% in those with stroke compared to 4.8% in those with TIA (adjusted cause-specific hazard ratio [aHR] for those with stroke vs TIA 1.32; 95% confidence interval [CI] 1.19–1.46) and 4.1% in age-/sex-matched controls (aHR for those with stroke vs controls 1.47; 95% CI 1.35–1.60). In the cohort with stroke, factors associated with fractures were older age, female sex, moderate stroke severity, prior fractures or falls, and preexisting osteoporosis, rheumatoid arthritis, hyperparathyroidism, and atrial fibrillation.

Conclusions: Stroke is associated with an increased risk of low-trauma fractures. Individuals with stroke and additional risk factors for fractures may benefit from targeted screening for low bone mineral density and initiation of treatment for fracture prevention. *Neurology*® 2017;88:57–64

GLOSSARY

aHR = adjusted hazard ratio; CI = confidence interval; ICES = Institute for Clinical Evaluative Sciences.

Stroke is a leading cause of adult disability, and up to two-thirds of stroke survivors have limitations in mobility.¹ Stroke may also be associated with declines in bone mineral density and an increased risk of falls, both factors that can predispose to subsequent low-trauma (fragility) fractures.^{2–10}

Previous studies have suggested an up to 4-fold increase in the risk of fractures in those with stroke compared to healthy controls.^{7–13} However, it is not known how much of this fracture risk is due to the stroke itself rather than differences in other baseline factors such as age and comorbidity. A better understanding of both the fracture risk attributable to stroke and the specific factors associated with such fractures would allow for improved screening, prevention, and treatment of osteoporosis and fractures following stroke.

We used data from a clinical stroke registry in Ontario, Canada, linked with administrative data, to evaluate the risk of fractures in patients with acute stroke compared to those with TIA. In order to focus on the contribution of stroke (rather than baseline comorbid conditions) to fracture risk, we selected patients with TIA as our reference population, as they are anticipated

Supplemental data
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to be similar to those with stroke except for the presence of residual neurologic impairment, the major purported mechanism behind the increased risk of fractures following stroke. In secondary analyses, we compared the risk of fractures in patients with stroke to age- and sex-matched controls without stroke or TIA.

METHODS Setting. The province of Ontario, Canada, has a population of over 12 million people, more than 150 acute care hospitals, an organized system of regional stroke care,¹⁴ and universal health coverage for hospital care, physicians' services, diagnostic tests, and (for those aged over 65 years) medications covered by the provincial drug formulary.

Data sources and study sample. The Ontario Stroke Registry (formerly known as the Registry of the Canadian Stroke Network) collects clinical information on a population-based sample of patients with stroke or TIA seen at all acute care institutions in the province.¹⁵ Validation by duplicate chart abstraction has shown excellent agreement for key variables including stroke type, severity, and comorbid conditions.¹⁵ The diagnosis of stroke or TIA is verified by study personnel through review of physician and nursing notes and results of investigations. Stroke severity at the time of initial assessment is determined using the Canadian Neurological Scale score, where higher scores indicate a less severe stroke.¹⁶ The registry also includes information on specific neurologic deficits, functional status after stroke, and vascular comorbid conditions. We used the Ontario Stroke Registry to identify a cohort of patients with acute stroke or TIA seen in the emergency department or admitted to any of 11 stroke centers between July 1, 2003, and March 31, 2012, and discharged alive. For patients with more than one stroke/TIA event during the study time frame, only the first event was included. We excluded those with subarachnoid hemorrhage and, from the TIA cohort, those with preexisting hemiparesis.

The registry is housed at the Institute for Clinical Evaluative Sciences (ICES), where it is linked to population-based administrative databases using unique, encoded patient identifiers. We used the Ontario Registered Persons Database, which contains demographic information on all provincial residents, to identify an age-/sex-matched control for each patient in our stroke cohort, to whom we then assigned the same index date as the person with stroke. These controls were selected from the general population, and were not matched on any variables other than age and sex. We used the Canadian Institute for Health Information Discharge Abstract Database and National Ambulatory Care Reporting System Database to identify hospitalizations and emergency department visits for fractures, falls, and other medical conditions. By law in Ontario, all hospital and emergency department visits are recorded in these databases, ensuring complete capture of events. We used the Ontario Health Insurance Plan (physician claims) database to identify outpatient physician visits and procedures, the Registered Persons Database to identify deaths, the 2010 Canada Census to provide information on median neighborhood income, and the Ontario Drug Benefits database to identify medication claims for patients aged over 65 years. These databases are validated and used routinely for research.¹⁷

Outcomes. The primary outcome was a low-trauma fracture that occurred within 24 months of the index stroke/TIA,

defined as any fracture of the femur, forearm, humerus, pelvis, or vertebrae, excluding fractures that occurred as a result of trauma, motor vehicle accidents, falls from a height, or in people with active cancer. As a secondary analysis, we evaluated the occurrence of falls resulting in a hospital or physician visit within 24 months of the index stroke/TIA. We identified fractures and falls through the administrative databases listed above, using diagnostic and procedural codes and validated algorithms (table e-1 at [Neurology.org](#)).

Confounding variables. Factors that may affect fracture risk independent of stroke include age, sex, previous fractures and medical conditions, and medication use associated with either an increased or decreased risk of fractures.^{18,19} Stroke-specific risk factors for fractures include hemiplegia/hemiparesis, visual impairment, stroke type and severity, and functional status after stroke.^{20,21} We identified these variables through either the registry database or through linkages to administrative data; validated algorithms were available for some but not all diagnoses (table e-1).

Analysis. We compared baseline characteristics of patients with stroke and those with TIA, using *t* tests for continuous variables and χ^2 tests for categorical variables. We then compared the incidence of fractures as a function of time in those with stroke vs those with TIA, using cumulative incidence functions to account for the competing risk of death, and with censoring of TIA patients at the time of any subsequent stroke.²² Since there may be differences in baseline risk factors for fractures between patients with stroke and TIA, we used inverse probability of treatment weighting using the propensity score to account for confounding due to measured baseline covariates.²³ In the sample weighted by the stabilized weights, we computed standardized differences to assess the balance of measured baseline covariates between treatment groups.²⁴ We then used Cox proportional hazard models to estimate the effect of stroke on the cause-specific hazard of fractures, using the stabilized weights to adjust for confounding.²⁵ We did not adjust for stroke-related factors such as stroke severity or weakness, as these were presumed to be in the causal pathway for poststroke fractures. We also did not adjust for medications in the primary analysis, because of the potential for confounding by indication (for example, use of osteoporosis medications in those at increased risk of fractures). However, in secondary analyses limited to patients aged over 65 years, we adjusted for the use of corticosteroids and other medications potentially associated with fracture risk (table e-1).

We then compared the rate of fractures in those with stroke with age-/sex-matched controls without stroke or TIA. We used Cox proportional hazard models to estimate the effect of stroke on the cause-specific hazard of fractures with adjustment for comorbid conditions (dementia, prior falls, arthritis, kidney disease, osteoporosis, hyperparathyroidism, Parkinson disease, prior fractures), with robust standard errors to account for clustering in matched pairs. We also used cumulative incidence functions to estimate the incidence of fractures over time after accounting for death as a competing risk.

In the cohort of patients with stroke, we used Cox proportional hazards regression to estimate the effect of the following variables on the cause-specific hazard of fractures: age (categorized as <65, 65–74, and ≥ 75 years); sex; stroke type (ischemic or hemorrhagic); stroke severity based on the Canadian Neurological Scale score and categorized as mild (≥ 8), moderate (5–8), or severe (≤ 4); residence in a long-term care facility; prior fractures; falls in the 2 years prior to the index event; stroke unit admission;

Table 1 Baseline characteristics of patients with stroke or TIA seen in the emergency department or admitted to the hospital and discharged alive from July 1, 2003, to March 31, 2012, and age- and sex-matched controls

	Stroke (n = 23,751)	TIA (n = 11,240)	Controls (n = 23,751)
Female ^a	46.6	49.6	46.6
Age, y, median (IQR)	74 (62-82)	73 (62-82)	74 (62-82)
Rural residence ^{a,b}	10.8	8.6	13.4
Neighborhood income quintile ^{a,b}			
1 (lowest)	23.3	21.5	19.3
2	21.2	19.1	20.4
3	18.5	18.0	19.7
4	17.9	18.7	20.0
5 (highest)	19.0	22.7	20.6
Stroke type			
Ischemic	86.0	—	—
Intracerebral hemorrhage	14.0	—	—
Stroke severity			
Severe (CNS 0-4)	11.7	—	—
Moderate (CNS 5-7)	18.8	—	—
Mild (CNS ≥ 8)	69.5	—	—
Admitted to a stroke unit ^a	70.7	55.4	—
Thrombolysis given if ischemic stroke	14.7	—	—
Diabetes ^a	24.4	21.7	—
Hypertension ^a	67.0	63.6	—
Hyperlipidemia ^a	36.7	40.7	—
Atrial fibrillation ^a	15.8	11.7	—
Prior stroke ^a	17.4	15.7	—
Dementia ^{a,b}	10.2	9.0	6.6
Renal disease ^b	7.9	8.1	5.0
Rheumatoid arthritis	1.4	1.7	1.0
Hyperparathyroidism	0.3	0.5	0.3
Malabsorption	0.3	0.3	0.2
Parkinson disease ^a	1.1	1.8	1.0
Prior fracture ^b	7.6	7.6	6.5
Falls prior to event ^b	20.5	21.5	9.7
Medications in year prior to event ^c			
Corticosteroids ^b	8.7	9.6	7.0
Thyroid medications ^a	14.4	16.9	13.8
Proton pump inhibitors ^{a,b}	27.0	29.7	24.0
Benzodiazepines ^{a,b}	20.5	23.6	18.8
Opioids ^b	25.7	26.3	21.5
Antidepressants ^{a,b}	10.5	12.3	8.4
Osteoporosis therapy ^a	19.3	22.1	19.6

Abbreviations: CNS = Canadian Neurological Scale; IQR = interquartile range.

Values are percentages unless noted otherwise.

^a $p < 0.001$ For comparison between stroke and TIA groups.

^b $p < 0.001$ For comparison between stroke and control groups.

^cMedications prescribed within the 12 months prior to the index event in patients aged over 65 years (no. with stroke = 16,581; no. with TIA = 7,893; no. controls = 16,581). Osteoporosis therapy includes bisphosphonates, denosumab, raloxifene, estrogen, and calcitonin.

Table 2 Two-year risk of fractures and falls after stroke

	Stroke (n = 23,751)	TIA (n = 11,240)	Age-/sex-matched controls (n = 23,751)	HR ^a for stroke vs TIA (95% CI)	HR ^a for stroke vs age-/sex-matched controls (95% CI)
Any low-trauma fracture	1,346 (5.7)	537 (4.8)	968 (4.1)	1.32 (1.19–1.46)	1.47 (1.35–1.60)
Femur fracture	671 (2.8)	225 (2.0)	404 (1.7)	1.57 (1.35–1.83)	1.76 (1.55–2.00)
Fall	3,374 (14.2)	1,628 (14.5)	2,293 (9.7)	1.08 (1.02–1.14)	1.60 (1.51–1.69)

Abbreviations: CI = confidence interval; HR = hazard ratio.

Values are n (%). Fall = fall resulting in physician or emergency department visit or hospitalization; low-trauma fracture = emergency department visit, hospitalization, or physician visit for fracture of femur, humerus, vertebrae, forearm, or pelvis, excluding fractures occurring as a result of trauma, motor vehicle accidents, falls from height, or in people with active cancer.

^aFor the stroke-TIA comparison, cause-specific hazard ratios were estimated using Cox models in the sample weighted using the stabilized inverse probability of treatment weights. For the stroke age-/sex-matched control comparison, cause-specific hazard ratios were estimated using Cox models with robust standard errors to account for clustering in matched pairs, and with adjustment for comorbid conditions.

and comorbid conditions including preexisting osteoporosis, stroke, hypertension, hyperlipidemia, diabetes, congestive heart failure, peripheral vascular disease, chronic kidney disease, atrial fibrillation, depression, cancer, dementia, rheumatoid arthritis, hyperparathyroidism, malabsorption syndromes, and Parkinson's disease. We used SAS version 9.4 (SAS Institute, Cary, NC) for all analyses.

Standard protocol approvals, registrations, and patient consents. Data collection for the registry is done without patient consent, since ICES is named as a prescribed entity under provincial privacy legislation. The study was approved by the Sunnybrook Health Sciences Centre Research Ethics Board.

RESULTS The study sample consisted of 34,991 patients, 23,751 of whom had stroke and 11,240 had a TIA. Baseline characteristics of participants are

shown in table 1. After using the inverse probability of treatment weights derived from the propensity score to balance baseline characteristics of patients with stroke and those with TIA, the weighted standardized differences for baseline covariates were all <0.10, suggesting good balance between the groups (table e-2, a and b).

The crude incidence of low-trauma fracture within 2 years of the index event was higher in those with stroke than in those with TIA (5.7% vs 4.8%, $p < 0.001$). This difference persisted even after using propensity score methods to account for differences in baseline factors (adjusted hazard ratio [aHR] for stroke vs TIA, 1.32; 95% confidence interval [CI] 1.19–1.46) (table 2, figure). Results were similar when medications were included in the model (aHR for fractures after stroke vs TIA 1.25; 95% CI 1.13–1.39). The incidence of fractures was also greater in those with stroke than in age-/sex-matched controls (5.7% vs 4.1%; aHR 1.47; 95% CI 1.35–1.60) (table 2). Almost half of all low-trauma fractures were femur fractures (table 2). Within 2 years of the index event, the risk of falls was similar in those with stroke and TIA (14.2% vs 14.5%; aHR 1.08; 95% CI 1.02–1.14) but higher in those with stroke compared to age-/sex-matched controls (14.2% vs 9.7%; aHR 1.60; 95% CI 1.51–1.69) (table 2). The risk of falls varied with stroke severity, with falls occurring in 14.8%, 14.5%, and 11.0% of those with mild, moderate, and severe strokes, respectively.

Factors associated with an increased rate of fractures after stroke were older age (aHR for age 65–74 vs under 65 years, 1.66; 95% CI 1.38–2.00 and aHR for age over 75 vs under 65 years, 2.42; 95% CI 2.05–2.85), female sex (aHR 1.72; 95% CI 1.53–1.94), stroke severity (aHR for moderate vs mild stroke, 1.16; 95% CI 1.01–1.32), prior stroke (aHR 1.16; 95% CI 1.02–1.33), atrial fibrillation (aHR 1.45; 95% CI 1.04–2.01), hyperparathyroidism (aHR 1.95; 95% CI 1.07–3.54), prior diagnosis

Figure Cumulative incidence of fracture in those with stroke vs TIA

Figure constructed using cumulative incidence functions to account for the competing risk of death. $p < 0.001$ for difference between patients with stroke and TIA, using Gray test for equality of cumulative incidence functions.

Table 3 Predictors of low-trauma fracture in the cohort of patients with stroke (n = 23,751)

Variable	% With fracture	Adjusted HR	95% CI
Male sex	3.6	Reference	—
Female sex	8.0	1.72	1.53-1.94
Age, y			
18-64	2.8	Reference	—
65-74	4.9	1.66	1.38-2.00
≥75	7.8	2.42	2.05-2.85
Stroke type			
Intracerebral hemorrhage	5.7	Reference	—
Ischemic	5.2	0.96	0.81-1.14
Stroke severity			
Mild (CNS ≥ 8)	5.6	Reference	—
Moderate (CNS 5-7)	6.4	1.16	1.01-1.32
Severe (CNS < 5)	5.2	1.00	0.85-1.18
Pre-event long-term care residence	7.7	1.03	0.80-1.31
Previous stroke	6.9	1.16	1.02-1.33
Hyperlipidemia	5.2	0.86	0.77-0.97
Atrial fibrillation	7.5	1.16	1.01-1.34
Hypertension	5.9	0.98	0.86-1.11
Diabetes mellitus	5.4	1.03	0.90-1.18
Congestive heart failure	6.1	0.89	0.71-1.10
Chronic kidney disease	6.0	0.97	0.62-1.51
Peripheral vascular disease	5.9	1.14	0.89-1.46
Dementia	7.8	1.05	0.88-1.25
Depression	6.9	1.11	0.91-1.36
Cancer	5.2	0.93	0.76-1.13
Rheumatoid arthritis	11.2	1.45	1.04-2.01
Hyperparathyroidism	13.4	1.95	1.07-3.54
Malabsorption	9.1	1.13	0.50-2.52
Parkinson disease	7.0	0.85	0.53-1.35
Prior diagnosis of osteoporosis	11.3	1.35	1.18-1.55
Pre-event falls	9.5	1.38	1.21-1.57
Pre-event fractures	15.9	2.31	1.98-2.70
Falls after stroke ^a			
Present	28.8	—	—
Absent	1.8	—	—
Weakness ^b			
Present	5.9	—	—
Absent	5.2	—	—
Modified Rankin Scale score at discharge ^b			
3-5 (greater disability)	6.5	—	—
0-2	4.7	—	—
Discharge destination ^b			
Home	4.4	—	—

Continued

of osteoporosis (aHR 1.35; 95% CI 1.18–1.55), prior falls (aHR 1.38; 95% CI 1.21–1.57), and fractures prior to the index stroke (aHR 2.31; 95% CI 1.98–2.70) (table 3). Stroke type, thrombolysis administration, and stroke unit care were not associated with fracture risk. Motor weakness, disability at discharge, and discharge destination were all associated with fractures in the univariable analyses, but were not included in the final models due to collinearity with stroke severity. More than one quarter of those with a fall following stroke also experienced a fracture (table 3).

DISCUSSION We found that the rate of a low-trauma fracture was 32% higher in patients with stroke than in those with TIA, and 47% higher than in age-/sex-matched controls. Fractures are important because they are associated with mortality and morbidity, including hospitalizations, reduced mobility, admission to long-term care facilities, and chronic pain.^{26–28} Fractures following stroke may have a particularly poor prognosis; individuals with prior stroke appear more likely to require rehabilitation and to have poorer mobility following fracture compared to those without stroke.²⁹ Although the absolute risk of poststroke fractures in our study was relatively modest at 5.7% over 2 years, and with only a 0.9% increase over the fracture risk seen in those with TIA, the number of excess fractures attributable to stroke becomes substantial when applied to the more than 10 million people who experience stroke worldwide each year.³⁰ Our findings are consistent with previous studies that have estimated the fracture risk in the year following stroke at between 3% and 10%, with the majority of fractures occurring at the hip, and with rates of hip fracture after stroke 1.4–4 times those seen in healthy controls.^{7,9,11–13,31} Our study adds to this literature by quantifying the risk associated with stroke compared to a well-matched population with TIA, permitting ascertainment of the fracture risk most directly attributable to the stroke itself rather than to differences in other baseline factors. One prior study found no difference in the risk of fractures after stroke vs after TIA; however, its sample size of 642 may have been underpowered to detect small differences in risk.¹¹

The increased risk of fractures following stroke is hypothesized to be related to both poststroke declines in bone mineral density and to an increased risk of falls. Bone loss after stroke preferentially affects the paretic extremity and is presumed to be primarily due to reduced mobility leading to decreased skeletal loading and increased osteoclast-mediated bone resorption.^{5,10,32,33} Longitudinal studies suggest an approximate 12%–17% decline in bone mineral density at the paretic extremities at 1 year poststroke,

Table 3 Continued

Variable	% With fracture	Adjusted HR	95% CI
Inpatient rehabilitation	7.1	—	—
Long-term care facility	7.0	—	—
Retirement home	14.9	—	—

Abbreviations: CI = confidence interval; CNS = Canadian Neurological Scale; HR = hazard ratio.

^a Post-event falls were not included in the multivariable models due to colinearity with fractures.

^b Weakness, modified Rankin Scale score, and discharge destination were not included in the multivariable model due to colinearity with stroke severity.

with either no change or an increase in bone mineral density in the nonparetic extremities.^{3,12} There appears to be a correlation between functional status and changes in bone mineral density after stroke, with lower mobility associated with greater declines in bone density.^{4,5,34–36} Poststroke falls, which typically occur at times of transfer or during mobilization, may be particularly likely to result in injury or fractures as they tend to occur toward the hemiparetic side, where the bone mineral density may be lower and where the ipsilateral arm may be unable to break the fall.⁶ Consistent with this, we found that fractures occurred in more than one quarter of those with falls in the 2 years after stroke. We also found that people with weakness and with residual disability at discharge were at increased risk of fractures, as were those with moderate strokes compared to those with mild strokes (who may have less bone loss and lower falls risk) and to those with severe strokes (who may have fewer falls due to greatly reduced mobility and fewer transfers). Interestingly, we found similar overall prestroke and poststroke fall rates in those with stroke and TIA, suggesting that factors other than stroke-related neurologic deficits account for some of the increased falls risk in those with cerebrovascular disease compared to the general population. Falls risk is not routinely included in osteoporosis treatment algorithms,³⁷ and this is something that could be addressed in future iterations of such tools. Other factors associated with poststroke fractures in our study cohort were older age, female sex, prior fractures, rheumatoid arthritis, and hyperparathyroidism, factors that are associated with osteoporosis and fractures in the general population and that are included in current fracture risk prediction scores.^{18,37}

Current osteoporosis clinical practice guidelines recommend screening with bone mineral density testing in those aged over 65 years and in younger people with specific risk factors for bone loss or fractures, followed by individualized fracture risk assessment and treatment.^{18,19} Our findings suggest that bone mineral density testing should be considered in people with recent

stroke, particularly those with additional risk factors for fractures, with the aim of initiating treatment in those at moderate to high risk of future fractures. Additional research is needed to determine if current fracture risk stratification systems are adequate for identifying treatment thresholds in people with stroke, or whether a recent stroke warrants osteoporosis therapy even in the absence of low bone mineral density or other fracture risk factors. Identification and management of falls risk is likely to be of particular importance in the prevention of poststroke fractures,⁶ and weight-bearing exercise protocols may help maintain bone mineral density.^{35,38} Bisphosphonates appear to reduce the risk of fractures after stroke^{39,40} and it is likely that other anti-resorptive and anabolic agents currently approved for osteoporosis therapy in the general population would also be effective in those with stroke.

Our study has some limitations. We were only able to identify fractures resulting in hospital or physician visits, and therefore will have underestimated fracture incidence, particular for vertebral fractures, which are frequently asymptomatic. Similarly, we only identified falls resulting in physician assessment; this selective identification of particularly injurious falls could have resulted in overestimates of the association between falls and fractures. Comorbid conditions such as dementia are likely to have been undercoded in our administrative databases, potentially leading to either overestimates or underestimates of the association between such variables and the risk of fractures. We did not have information on bone mineral density, and therefore could not assess rates of bone loss after stroke or the association between bone mineral density and fracture risk in our study cohort; we also could not determine whether fractures occurred preferentially in paretic extremities. We did not have information on some risk factors for osteoporosis, such as low body mass index and vitamin D deficiency, or on prognostic variables such as results of formal fall risk assessments. Finally, despite our choice of a well-matched TIA control population and additional matching using propensity score methods, there is the risk of residual confounding due to imbalances in baseline variables, and the inability to balance unmeasured confounders such as frailty. Despite these limitations, our results from a large, population-based sample of patients with detailed information on stroke characteristics and with complete follow-up through administrative data are likely to provide valid data on the risks of fractures after stroke.

The results of this study suggest that low-trauma fractures are a frequent and clinically important complication of stroke. Future work should focus on increasing awareness of the risk of fractures after stroke and on identifying individuals who might benefit from targeted screening and treatment for fracture prevention.

AUTHOR CONTRIBUTIONS

M.K.K. drafted the manuscript. J.F. conducted the statistical analyses. P.C.A. provided additional statistical expertise related to the study design, analyses, and interpretation. All authors contributed to the conception and design of the study, interpretation of the data, and revisions of the work for important intellectual content. All authors gave final approval of the version of the manuscript submitted for publication, and agree to be accountable for the work. M.K.K. had full access to all the data in the study, takes responsibility for the integrity of the data and the accuracy of the data analysis, and had final responsibility for the decision to submit for publication.

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DISCLOSURE

The authors report no disclosures relevant to the manuscript. Go to <http://www.ics.on.ca/~media/Files/Atlases-Reports/2011/RCSN-2008-09-Ontario-stroke-audit/Full%20report.ashx> for full disclosures.

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