ED Administration

How do we manage emergency department patients diagnosed with transient ischemic attack?

Jeffrey J. Perry, MD, MSc*†; Jonathan Kerr, MD†; Cheryl Symington, RN, ENCC*; Jane Sutherland, MEd†

ABSTRACT

Introduction: Multiple studies have demonstrated low rates of antithrombotic use, low neuroimaging rates, and high subsequent risk of stroke at 90 days following an emergency department (ED) diagnosis of transient ischemic attack (TIA). This study assessed the use of antithrombotic medications, neuroimaging, and subsequent 90-day stroke rate for patients in a more recent cohort of ED patients discharged home with TIA.

Methods: We conducted a 1-year historical cohort study of all patients discharged with a TIA at a tertiary care ED (census 60,000 visits/year), which was one of the four sites participating in one of the aforementioned studies. Data were extracted from paper and electronic records onto standardized data extraction forms. Clinical findings, medications, and tests were recorded.

Results: A total of 211 patients were enrolled in the study. The patients had the following characteristics: the mean age was 71.2 years (SD 13.8 years), 56.9% were female, 53.1% had a history of hypertension, 26.5% had a history of ischemic heart disease, and 17.1% had a previous stroke. The most frequent neurologic deficit was unilateral weakness (53.6%), and most deficits lasted for more than 60 minutes (71.6%). Antithrombotic medications were used for 96.7% of patients at ED discharge. Neuroimaging was conducted in 94.3% of patients while in the ED. Our cohort had a 90-day stroke rate of 1.9%.

Conclusion: This study established that most TIA patients receive neuroimaging in the ED and are started on or maintained on antithrombotic agents. Clinicians are encouraged to ensure that electrocardiography is done routinely and to involve Neurology in follow-up care.

Keywords: emergency department, stroke management, transient ischemic attack

Transient ischemic attack (TIA) is defined as a sudden, focal, neurologic deficit lasting for less than 24 hours, presumed to be of vascular origin, and confined to an area of the brain or eye perfused by a specific artery.[1,2] TIA is a clinical diagnosis that is made from the history and physical examination findings. Although often thought of as benign, the diagnosis of TIA carries with it a serious risk of stroke or death shortly after diagnosis. TIAs are common, with an annual incidence of 68 per 100,000 Canadians.[3] By definition, patients with TIAs do not have permanent neurologic deficits; however, TIAs do identify individuals who are at increased risk of a stroke, with its associated morbidity and mortality. Stroke is the leading cause of adult disability and the third leading cause of death in Canada. There are an estimated 50,000 new stroke cases per year in Canada, of which approximately 20,000 are preceded by a TIA, thus providing a unique opportunity for prevention.[4] Strokes cost the Canadian economy 2.7 billion dollars a year and account for 3 million hospital days annually.[4] Therefore, it is critical to ensure that all possible preventive measures are employed before patients experience a stroke.[5–10]

The risk of stroke following TIA is estimated at about 9% within 90 days of a TIA.[3,5,10,11] One of these studies was conducted in 2000 in Ontario.[5] In this study of 371 emergency department (ED) patients with TIA, physicians were only prescribing antithrombotic medications in 64% of cases and conducting neuroimaging in 31% of cases. Hence, there was a push in the literature to convince physicians to consider TIAs

From the *Department of Emergency Medicine, University of Ottawa, and the †Clinical Epidemiology Program, Ottawa Hospital Research Institute, Ottawa, ON.

Presented at the Canadian Association of Emergency Physicians (CAEP) Annual Scientific Meeting, Victoria, BC, 2007.

Correspondence to: Dr. Jeffrey J. Perry, Clinical Epidemiology Program, F647, The Ottawa Hospital, 1053 Carling Avenue, Ottawa, ON K1Y 4E9; jperry@ohri.ca.

This article has been peer reviewed.

CJEM 2012;14(1):20-24 DOI 10.2310/8000.2011.110533

as true emergencies. Relatively little work has been conducted since this study to determine if practice has changed in Ontario EDs.

The goal of this study was to determine the frequency of antithrombotic use for TIA patients in the ED. Secondary outcomes include assessing the use of neuroimaging in the ED, assessing the use of electrocardiograms (ECG), and determining the incidence of subsequent stroke for patients at 90 days following their TIA.

METHODS

Design and setting

We conducted a 1-year historical cohort study (January 1 to December 31, 2005) at a Canadian university-affiliated tertiary care ED (census 60,000 visits/year).

Study population

All patients with weakness, TIA, or stroke were screened from the hospital's electronic database of emergency diagnoses. All patients 18 years of age or older with a final ED diagnosis of TIA or stroke (if neurologic deficits were present for less than 24 hours) who were assessed by an emergency physician and not seen directly by a specialty service were eligible for inclusion. We excluded patients in the following categories: 1) a prior TIA within the previous 90 days; 2) symptoms longer than 24 hours at the time of the ED visit; 3) decreased level of consciousness (i.e., Glasgow Coma Scale score $<$ 15); 4) admitted to hospital; and 5) documented other cause for their deficit that is not a TIA (e.g., hypoglycemia, seizure, electrolyte imbalance).

Data abstraction

The data extraction form was generated by consensus of three experienced physicians (two emergency physicians and one neurologist). A trained medical student then piloted this form on 20 patient charts. These charts were reviewed with the supervising emergency physician researcher, revised, and finalized before further use. The remaining charts were subsequently abstracted by the medical student. A random subset of 5% of the charts was reviewed by a second reviewer for accuracy. Data, including clinical findings, medications, and tests, were extracted from each record of treatment, which included physician, nursing, consultant, triage, and ambulance reports, follow-up neurologic consultations, and radiology reports. Data for clinical variables not recorded in any of the clinical information were coded as negative (i.e., history of diabetes, hypertension). Additional information to assess outcomes and follow-up was sought from the hospital's computer archiving system to assess subsequent ED visits or subsequent consultation in the stroke or general neurology clinic at either campus of The Ottawa Hospital. Data were recorded on a standardized paper data extraction form and then input into a computerized database using *Statistical Analysis System* (*SAS*, SAS Institute, Cary, NC) software by a single experienced data entry specialist.

Analysis

Descriptive analyses were conducted for the primary outcome of secondary stroke prevention with antithrombotic use and patient demographics, clinical findings, and testing conducted, as well as for subsequent stroke at 90 days following the index ED visit.

Sample size

The sample size was determined primarily by feasibility, with the funding sufficient to obtain a 1-year cohort of ED patients with TIA.

RESULTS

Our electronic health records search identified 2,235 potential patient encounters. There were 2,014 patients excluded for not meeting eligibility criteria. There were 10 patients whose medical records could not be found. Ultimately, we enrolled 211 patients in the study. There were no discrepancies in the information recorded in the random 5% of charts audited by the senior study emergency physician/researcher.

Table 1 displays the clinical characteristics of the study population. Our cohort included older patients with a mean age of 71.2 years and was mostly female, and over half had comorbid medical conditions. Of these conditions, 17.1% had suffered a previous stroke and 16.6% reported having one or more previous

https://doi.org/10.2310/8000.2011.110533 Published online by Cambridge University Press

Table 1. Characteristics of the 211 enrolled TIA study patients

Characteristic	Number of patients (N = 211)
Demographics	
Mean age (SD)	71.2 (13.8)
Female (%)	120 (56.9)
Past medical history (%)	
Hypertension	112 (53.1)
History of ischemic heart disease	56 (26.5)
Previous stroke	36 (17.1)
Previous TIA	35 (16.6)
Diabetes mellitus	32 (15.2)
Atrial fibrillation	15 (7.1)
Current smoker	13 (6.2)
Peripheral vascular disease	3 (1.4)
Neurologic deficits (%)	
Unilateral weakness	113 (53.6)
Unilateral sensory loss	76 (36.0)
Aphasia	61 (28.9)
Dysarthria	58 (27.5)
Visual disturbance	34 (16.1)
Duration of symptoms (%)	
< 10 min	25 (11.8)
10–59 min	34 (16.1)
≥ 60 min	151 (71.6)
Multiple TIAs in last 24 hours	33 (15.6)
Diagnostic procedures in ED (%)	
Computed tomography	199 (94.3)
Infarcts present (old or acute)	32 (15.2)
Electrocardiography	179 (84.8)
Disposition (%)	
Family physician	21 (10.0)
Neurology clinic	173 (82.0)
No follow-up plan recorded	17 (8.0)
TIA confirmed by Neurology (%) (n = 130)	100 (76.9)

ED = emergency department; TIA = transient ischemic attack.

TIAs. The most common neurologic deficit present was unilateral weakness (53.6%). Most patients (94.3%) underwent head computed tomography (CT), of which 16.1% demonstrated an acute or previous infarct.

Table 2 lists the changes made to patient medications. In our cohort, 96.7% were treated with an antithrombotic agent (any antiplatelet agent and/or warfarin). Of these patients, 64.5% had their antithrombotic agent altered in the ED. In addition, 6.2% of patients were started on a statin (total patients on statin medication was 34.1% after the ED visit), and 4.7% were started on antihypertensive medications (total patients on antihypertensive medications was 59.2%).

The TIA diagnosis was confirmed by Neurology in 76.9% of the 130 patients seen at one of the general neurology or stroke clinics affiliated with our hospital. The subsequent stroke rate at 90 days was 1.9% for our cohort of TIA patients.

DISCUSSION

Almost all patients diagnosed with TIA in our cohort were treated with antithrombotic agents. The vast majority of patients were investigated with head CT and ECG while in the ED. The number of patients with a subsequent stroke was low, with only 1.9% experiencing a subsequent stroke at 90 days.

Gladstone and colleagues conducted a study using the Ontario Stroke Registry, which is a cohort of patients with stroke or TIA assessed at one of four Ontario stroke centre EDs over a 7-month period in 2000.[5] Data were extracted from patients' charts and from linked databases with the Ontario Health Insurance Plan and to the Canadian Institute for Health Information to assess subsequent outcomes for subsequent stroke. One of the regional stroke centres included in this study was our site. Our cohort characteristics are similar to Gladstone and colleagues' results with regard to age, sex, past medical history of hypertension, previous stroke, and diabetes. Symptoms were also similar; however, they had slightly more patients with unilateral weakness (65% versus 54% in our cohort). Their study included patients admitted to hospital; however, 76% were discharged home from the ED. Over one-third of the patients were not treated with antithrombotic agents at the time of ED discharge in 2000 versus almost all patients maintaining or starting antithrombotic agents in our cohort. Nearly two-thirds of the patients had an alteration in the antithrombotic agent in the ED (i.e., starting a patient who was on no antithrombotic or any agent or changing a patient who was on acetylsalicylic acid [ASA] to clopidogrel or a dipyridamole-ASA combination).[12–14] The alteration of other medications, such as starting statins or antihypertensive medications, was more tepid. There was also a marked improvement in the neuroimaging rates in the ED. In 2000, only 31% of patients had either magnetic resonance imaging (MRI) or CT while in the ED versus 94.3% who had neuroimaging (all CT) while in the ED. The ECG rate was little changed from the 81% found in 2000 to 85% in our study.

https://doi.org/10.2310/8000.2011.110533 Published online by Cambridge University Press

Table 2. Stroke prevention medication changes for 211 TIA patients

Medication	Previously on medication, *n* (%)	Started on in ED, *n* (%)	Discontinued in ED, *n* (%)
ASA	54 (25.6)	69 (32.7)	33 (15.6)
Dipyridamole-ASA	8 (3.8)	50 (23.7)	1 (0.5)
Clopidogrel	19 (9.0)	17 (8.1)	1 (0.5)
Ticlopidine	2 (0.9)	0 (0)	0 (0)
Warfarin	13 (6.2)	1 (0.5)	0 (0)
Statin	72 (34.1)	13 (6.2)	0 (0)
Antihypertensive	125 (59.2)	10 (4.7)	0 (0)

ASA = acetysalicylic acid; ED = emergency department; TIA = transient ischemic attack.

In Gladstone and colleagues' study, patients had a 30-day stroke risk of 6.0%.[5] The number of subsequent strokes was lower in our study than the numbers reported by Gladstone and colleagues. This may have occurred for several reasons. The first is that they used a provincial database to determine subsequent events. Hence, due to the retrospective nature of our study, we may have missed some patients who went to one of the smaller regional hospitals or to their family physician and received a diagnosis of stroke. We did not include admitted patients; therefore, some of the sickest patients may not have been included in our cohort, although relatively few patients are admitted locally for TIA. Alternatively, the event rate may have dropped due to better care. Two studies have demonstrated that rapid optimal care decreases subsequent early strokes from a baseline of 6.0 to 10.3% at 90 days to 1.2 to 2.1% with improvements in care, including early use of antithrombotic agents.[15,16]

LIMITATIONS

We identified several potential weaknesses in our study. First, we used the traditional time-based (i.e., deficit lasting less than 24 hours) clinical diagnosis of TIA.[2] A newer tissue-based diagnosis has been suggested by the American Heart Association; however, this requires MRI to exclude brain infarct and that the symptoms last for less than 1 hour.[17] Given that these events are both ischemic, the secondary prevention is identical, and MRI is not readily available in most Canadian centers, the clinical diagnosis of TIA is still more appropriate.

Additional limitations of this study are inherent in its design. The retrospective nature of the study results in some missing data. Although the charting was very complete for variables of interest in this study, it is impossible to discern if all patient risk factors, neurologic deficits, or medications were fully recorded in the patient medical records. Another limitation was that we primarily used one data abstractor to record the results. We did have a second reviewer audit about 5% of the charts, which did not reveal any problems; however, not all charts were double-checked for accuracy by a second reviewer.

Finally, we excluded admitted patients with TIA. In the previous study by Gladstone and colleagues, only 24% of TIA patients were admitted, and the median duration of these admissions was 1 day.[5] We excluded admitted patients because they are much more likely to be treated aggressively and likely constitute a sicker patient population. If a bias exists due to this exclusion, it would lead us to have a lower rate of investigations and lower medication use, including antithrombotics; however, if patients at highest risk for a subsequent event were admitted, this may also have contributed to our low subsequent stroke rate. Our low admission rate could also be due to not actively following patients to assess stroke-free status, which has been demonstrated to identify about two times the number of positive events than passive follow-up.[18] Given that our study hospital is the largest in the region and hosts the stroke prevention clinics, it is likely that most patients with a subsequent event would have been captured with our study methods.

CONCLUSION

This study established that a relatively large number of TIA patients are managed very well with investigation and antithrombotic agents in the ED. Most patients also received neuroimaging during their ED assessment. Care could be further improved by ensuring that

https://doi.org/10.2310/8000.2011.110533 Published online by Cambridge University Press

ECG is routinely done and that neurology specialists are involved.

Acknowledgements: The authors would like to acknowledge peer-reviewed funding from the CAEP 2006 Funding Competition. As well, Dr. Perry is supported by a Canadian Institutes of Health Research New Investigator Award and was previously supported as a Career Scientist by the Ontario Ministry of Health. We thank our colleagues at the Ottawa Hospital Research Institute (My-Linh Tran, Irene Harris, Catherine Clement, and Angela Marcantonio) for their assistance with this project.

Competing interests: None declared.

REFERENCES

1. WHO MONICA Project Principal Investigators. The World Health Organization MONICA Project (monitoring trends and determinants in cardiovascular disease): a major international collaboration. *J Clin Epidemiol* 1988;41:105-14, doi:10.1016/0895-4356(88)90084-4.
2. Albers GW, Caplan LR, Easton JD, et al. Transient ischemic attack—proposal for a new definition. *N Engl J Med* 2002;347:1713-6, doi:10.1056/NEJMsb020987.
3. Hill MD, Yiannakoulias N, Jeerakathil T, et al. The high risk of stroke immediately after transient ischemic attack. *Neurology* 2004;62:2015-20.
4. Heart and Stroke Foundation of Canada. *Stroke statistics*. Available at: http://www.heartandstroke.ca/Page.asp?PageID=33&ArticleID=1078&scc=news&From=SubCategory (accessed Mar 1, 2007).
5. Gladstone DJ, Kapral MK, Fang J, et al. Management and outcomes of transient ischemic attacks in Ontario. *Can Med Assoc J* 2004;170:1099-104, doi:10.1503/cmaj.1031349.
6. Rothwell PM, Giles MF, Flossmann E, et al. A simple score (ABCD) to identify individuals at high early risk of stroke after transient ischaemic attack. *Lancet* 2005;366:29-36, doi:10.1016/S0140-6736(05)66702-5.
7. Lovett JK, Dennis MS, Sandercock PAG, et al. Very early risk of stroke after a first transient ischemic attack. *Stroke* 2003;34:e138-42, doi:10.1161/01.STR.0000080935.01264.91.
8. Kleindorfer D, Panagos P, Pancioli A, et al. Incidence and short-term prognosis of transient ischemic attack in a population-based study. *Stroke* 2005;36:720-4, doi:10.1161/01.STR.0000158917.59233.b7.
9. Johnston SC. Transient ischemic attacks are emergencies. *Stroke* 2005;36:724.
10. Johnston SC, Gress DR, Browner WS, et al. Short-term prognosis after emergency department diagnosis of TIA. *JAMA* 2000;284:2901-6, doi:10.1001/jama.284.22.2901.
11. Giles MF, Rothwell PM. Risk of stroke early after transient ischaemic attack: a systematic review and meta-analysis. *Lancet* 2007;6:1063-72, doi:10.1016/S1474-4422(07)70274-0.
12. Halkes PH, van Gijn J, Kappelle LJ, et al. Aspirin plus dipyridamole versus aspirin alone after cerebral ischaemia of arterial origin (ESPRIT): randomised controlled trial. *Lancet* 2006;367:1665-73, doi:10.1016/S0140-6736(06)68734-5.
13. A randomised, blinded trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). CAPRIE Steering Committee. *Lancet* 1996;348:1329-39, doi:10.1016/S0140-6736(96)09457-3.
14. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ* 2002;324:71-86, doi:10.1136/bmj.324.7329.71.
15. Rothwell PM, Giles MF, Chandratheva A, et al. Effect of urgent treatment of transient ischaemic attack and minor stroke on early recurrent stroke (EXPRESS study): a prospective population-based sequential comparison. *Lancet* 2007;370:1432-42, doi:10.1016/S0140-6736(07)61448-2.
16. Lavallee PC, Meseguer E, Abboud H, et al. A transient ischaemic attack clinic with round-the-clock access (SOS-TIA): feasibility and effects. *Lancet Neurol* 2007;6:953-60, doi:10.1016/S1474-4422(07)70248-X.
17. Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. *Stroke* 2009;40:2276-93, doi:10.1161/STROKEAHA.108.192218.
18. Wu CM, McLaughlin K, Lorenzetti DL, et al. Early risk of stroke after transient ischemic attack: a systematic review and meta-analysis. *Arch Intern Med* 2007;167:2417-22, doi:10.1001/archinte.167.22.2417.