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ORIGINAL RESEARCH

Hospital-Level Variability in Reporting of Ischemic Stroke Subtypes and Supporting Diagnostic Evaluation in GWTG-Stroke Registry

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BACKGROUND: Secondary prevention of ischemic stroke (IS) requires adequate diagnostic evaluation to identify the likely etiologic subtype. We describe hospital-level variability in diagnostic testing and IS subtyping in a large nationwide registry.

METHODS AND RESULTS: We used the GWTG-Stroke (Get With The Guidelines–Stroke) registry to identify patients hospitalized with a diagnosis of acute IS at 1906 hospitals between January 1, 2016, and September 30, 2017. We compared the documentation rates and presence of risk factors, diagnostic testing, achievement/quality measures, and outcomes between patients with and without reported IS subtype. Recording of diagnostic evaluation was optional in all IS subtypes except cryptogenic, where it was required. Of 607 563 patients with IS, etiologic IS subtype was documented in 57.4% and missing in 42.6%. Both the rate of missing stroke pathogenesis and the proportion of cryptogenic strokes were highly variable across hospitals. Patients missing stroke pathogenesis less frequently had documentation of risk factors, evidence-based interventions, or discharge to home. The reported rates of major diagnostic testing, including echocardiography, carotid and intracranial vascular imaging, and short-term cardiac monitoring were <50% in patients with documented IS pathogenesis, although these variables were missing in >40% of patients. Long-term cardiac rhythm monitoring was rarely reported, even in cryptogenic stroke.

CONCLUSIONS: Reporting of IS etiologic subtype and supporting diagnostic testing was low overall, with high rates of missing optional data. Improvement in the capture of these data elements is needed to identify opportunities for quality improvement in the diagnostic evaluation and secondary prevention of stroke.

Key Words: diagnostic testing ■ ischemic ■ quality and outcomes ■ stroke

Ischemic stroke (IS) is a leading cause of death and disability in adults, and patients who sustain a first IS are at high risk for recurrent stroke.[1] Although the clinical presentation and characteristic imaging findings make IS a relatively straightforward diagnosis, the etiologic mechanisms that cause IS are heterogeneous. This may include large-artery atherosclerosis, small-vessel disease, and cardioembolism, but there are a number of other conditions that lead to IS. Appropriate diagnostic testing is required to identify a etiologic subtype of IS so that secondary prevention strategies can be individualized for patients. For example, carotid endarterectomy and stenting are recommended in stroke due to cervical carotid stenosis; oral anticoagulation should be used in atrial fibrillation; and antiplatelet monotherapy is recommended for

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RESEARCH PERSPECTIVE

What Is New?

- In the GWTG-Stroke (Get With The Guidelines–Stroke) registry of hospitalized patients with a diagnosis of acute ischemic stroke, ischemic stroke subtype was not documented in 42.6% of patients, and there was high variability in documentation across hospitals. Additionally, among patients with documented ischemic stroke subtype, reported rates of major diagnostic testing were <50%; in particular, long-term cardiac rhythm monitoring was rarely reported, even in cryptogenic stroke.
- Although GWTG-Stroke data are generally representative, with high levels of accuracy, the validity of the stroke subtype and diagnostic testing data elements has not been established, and it is not known whether these results represent broad uncertainty around stroke etiologic subtype and significant deficiencies in the diagnostic evaluation of patients with stroke in clinical practice, or whether they are driven primarily by deficiencies in documentation and data collection/reporting.

What Question Should Be Addressed Next?

- Given the importance of diagnostic testing and etiologic subtype determination to optimize secondary stroke prevention, an effort to validate etiologic stroke subtype and diagnostic testing within GWTG-Stroke should be undertaken.
- With validated data, the GWTG-Stroke registry would become a powerful tool to identify potential disparities in evaluation and secondary stroke prevention and could be used to create targeted quality improvement initiatives to reduce those disparities.

Nonstandard Abbreviations and Acronyms

CS	cryptogenic stroke
GWTG-Stroke	Get With The Guidelines–Stroke
IS	ischemic stroke
TOAST	Trial of ORG 10172 in Acute Stroke Treatment

small-vessel disease.[2–4] There have been recent advances in the detection of major etiologic factors, most notably the development of insertable cardiac monitors, which identify paroxysmal atrial fibrillation in a large proportion of patients with IS.[5,6]

Relatively little is known about the variability in diagnostic evaluation and etiologic subtype classification of IS across hospitals and across types of patients. In this study, we aimed to identify the hospital-level variability in reported diagnostic testing and ischemic stroke subtype documentation using a large national cohort of hospitalized patients with acute IS enrolled in the GWTG-Stroke (Get With The Guidelines–Stroke) registry.

METHODS

Standard Protocol Approvals, Registrations, and Patient Consent

Each participating hospital received either human research approval to enroll cases without individual patient consent under the common rule, or a waiver of authorization and exemption from subsequent review by their institutional review board. The Duke Clinical Research Institute serves as the data analysis center and has an agreement to analyze the aggregate deidentified data for research purposes. The institutional review board at Duke University Health approved this study.

Data Availability

GWTG-Stroke data were collected by the American Heart Association (the steward of the data according to contracts between the American Heart Association and participating hospitals) and are stored securely at the Duke Clinical Research Institute. Given that data were collected for clinical care and quality improvement, rather than primarily for research, data-sharing agreements require an application process for other researchers to access the data. Interested researchers can submit proposals to use GWTG for research purposes, including for validation purposes. Proposals can be submitted at www.heart.org/qualityresearch. Additional information regarding the statistical analysis plan and analytic code may be available from Duke Clinical Research Institute upon request.

Data Source

The GWTG-Stroke registry is based on an in-hospital program for improving stroke care by promoting consistent adherence to the latest scientific management guidelines in the United States. Participating hospitals record data related to the clinical characteristics, management, and outcomes of patients admitted with an acute IS via a web-based Patient Management Tool (IQVIA, Cambridge, MA).[7]

Data Collection and Study Population

We identified all patients with a final diagnosis of IS at participating hospitals between January 1, 2016, and

September 30, 2017, using the GWTG-Stroke registry. GWTG-Stroke data elements related to the documentation of IS etiology and accompanying coding instructions were developed and finalized in 2015 and implemented in January 2016.

Variables of Interest

The main variable of interest was the reporting versus nonreporting of etiologic IS subtype based on the TOAST (Trial of ORG 10172 in Acute Stroke Treatment) criteria.[8] Stroke subtypes categorized according to the TOAST criteria included cardioembolism, large-artery atherosclerosis, small-vessel disease, other determined pathogenesis, and cryptogenic stroke (CS).[8] CS was defined as occurring when physician documentation stated that a potential cause of stroke was not identified or multiple possible causes were identified, following a thorough diagnostic evaluation. Coding instructions noted that low- and moderate-risk potential cardioembolism sources such as patent foramen ovale, heart failure with preserved ejection fraction, mitral annulus calcification, or atrial or ventricular arrhythmias other than atrial fibrillation or flutter were of uncertain significance and not adequate to ascribe stroke mechanism. The other primary variables of interest were common etiologic tests, including extracranial carotid imaging, intracranial vascular imaging, echocardiography, hypercoagulability testing, short-term cardiac rhythm monitoring, extended surface cardiac rhythm monitoring, and extended implantable cardiac rhythm monitoring. Etiologic evaluation was required to be completed only for patients with a TOAST subtype of CS, and entry was optional for all other subtypes including when this was missing.

Other Data

Demographic data included age, sex, race and ethnicity, health insurance status, and arrival and admission data including on/off hours, mode of arrival to the hospital, National Institutes of Health Stroke Scale score, time of arrival, and thrombolytic drug administration. Risk factors that were recorded included hypertension, diabetes, dyslipidemia, atrial fibrillation, previous stroke or transient ischemic attack, prosthetic heart valve, coronary artery disease, carotid artery stenosis, peripheral vascular disease, heart failure, smoking, and sickle cell disease. Hospital characteristics were analyzed including geographic region, the number of beds, hospital location (rural versus urban), teaching and primary stroke center status, and annual volume of stroke. In-hospital treatment included antithrombotic therapy, and other risk factor–modifying medications at discharge were recorded. Quality and outcome measures included thrombolytic therapy with intravenous tissue plasminogen activator within 3 hours or 4.5 hours of last known well in eligible patients, administration of tissue plasminogen activator within 1 hour of hospital arrival, early antithrombotic therapy, anticoagulation for atrial fibrillation or flutter, deep venous thrombosis prophylaxis, statin initiation and low-density lipoprotein measurement, smoking cessation, dysphagia screening, stroke education, consideration for rehabilitation, and defect-free care. In-hospital death, ambulatory status at discharge, modified Rankin Scale score at discharge, discharge destination, and length of stay were analyzed as outcomes, although we did not plan to look for cause-and-effect relationships between our main/primary variables of interest and outcomes because of residual confounding in this exploratory, nonrandomized study.

Statistical Analysis

We described the baseline patient and hospital characteristics, including all data related to management and outcomes, and we compared them between patients who had IS pathogenesis missing versus reported. Percent standardized difference was calculated as a measure of effect size between groups.[9] A percent standardized difference >10 indicates a statistically large between-group difference. We plotted the hospital-level stroke pathogenesis reporting rate using a histogram to better explore variability in coding. We described the reported rates of major IS etiologic testing according to the reported IS etiologic subtype. These diagnostic tests included extracranial carotid imaging, intracranial vascular imaging, echocardiography, hypercoagulability testing, short-term cardiac rhythm monitoring, extended surface cardiac rhythm monitoring, and extended implantable cardiac rhythm monitoring. We plotted diagnostic tests completed by IS subtype and the hospital-level percentage of CS out of total documented stroke pathogenesis to better visualize the distribution of these data. Categorical variables were presented as counts and percentages, and the difference between groups was tested using Pearson χ^2 tests. Continuous variables were presented as mean and SDs or medians with 25th and 75th percentiles, and the difference between groups was tested using the Kruskal–Wallis test. All statistical analyses were performed using SAS version 9.3 software (SAS Institute, Cary, NC).

RESULTS

Reporting of Pathogenesis After IS

Among 607 563 patients with IS from 1906 sites, etiologic IS subtype was reported in 348 715 (57.4%) and missing in 258 848 (42.6%). Figure 1 provides the distribution of hospital stroke pathogenesis documentation rates, which ranged from 0% to >90%. Of the 1906

hospitals, there were 181 (9.5%) that did not document stroke pathogenesis in any patients and 1271 (67%) that documented stroke pathogenesis in <70% of IS admissions.

Of the 348 715 patients with reported IS subtype, 81 394 (23%) were categorized as large-artery atherosclerosis, 96 592 (28%) as cardioembolism, 82 542 (24%) as small-vessel disease, 18 330 (5%) as other determined pathogenesis, and 69 857 (20%) as CS. There was wide variation across hospitals in the proportion of patients documented as CS, ranging from <10% (43.5% of hospitals) to >90% (1.0% of hospitals) with a right skewed distribution (Figure 2). Less than 20% of ISs were categorized as CS in 65% of hospitals.

Patient characteristics are summarized in Table 1. There were minor differences between patients with reported IS pathogenesis and those missing IS pathogenesis with respect to age, sex, and race and ethnicity (Table 1). Insurance status was undocumented in 28% of the patients missing stroke subtype and 15.5% with a reported IS subtype (P<0.0001). This imbalance precluded analysis of the association between insurance status and IS subtype determination. History of atrial fibrillation was less common in the stroke pathogenesis missing group (15.91% versus 20.57%; P<0.0001). There were small differences in other risk factors, including hypertension, dyslipidemia, diabetes, carotid stenosis, heart failure, and prosthetic heart valve, which were generally more common among those missing stroke pathogenesis (Table 1). Past medical history variables were completely missing in 0.07% of patients with documented stroke pathogenesis versus 5.03% of patients without (P<0.0001). Patients missing pathogenesis had slightly less severe strokes with lower mean National Institutes of Health Stroke Scale (6.03±7.24 versus 6.55±7.51) and shorter duration of hospital stay (5.06±3.06 versus 5.80±3.24). They were more frequently discharged home (52.2% versus 49.76%) and more frequently independent in activities of daily living (42.35% versus 40.06%). Achievement and quality measures are summarized in Table 1. Patients missing stroke pathogenesis had these measures less frequently across the board, including early tissue plasminogen activator administration, antithrombotic use, anticoagulation for atrial fibrillation, deep vein thrombosis prophylaxis, statin use based on low-density lipoprotein levels, smoking cessation, defect-free measure, dysphagia screen, stroke education, consideration of rehabilitation, door-to-intravenous tissue plasminogen activator time ≤1 hour, documentation of low-density lipoprotein, intensive statin therapy, and reporting of National Institute of Health Stroke Scale. Patients admitted to hospitals with a lower annual volume of ISs and smaller number of beds and patients admitted to nonacademic and rural hospitals more frequently had stroke pathogenesis missing (Table 1).

Variability in Reporting of Etiologic Testing Among Patients With Reported Pathogenesis

We analyzed the variability in reported etiologic testing in 348 715 patients with IS who had reported stroke pathogenesis. Table 2 provides a detailed review of etiologic workup in patients with a documented stroke pathogenesis. In the overall group, carotid imaging

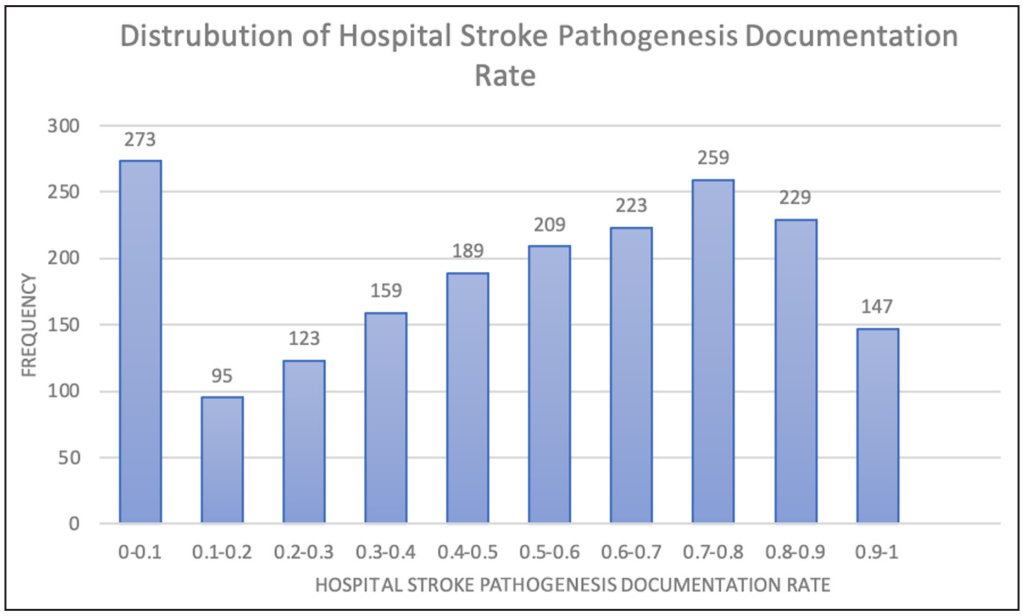

Figure 1. Histogram of hospital-level stroke pathogenesis documentation rate.

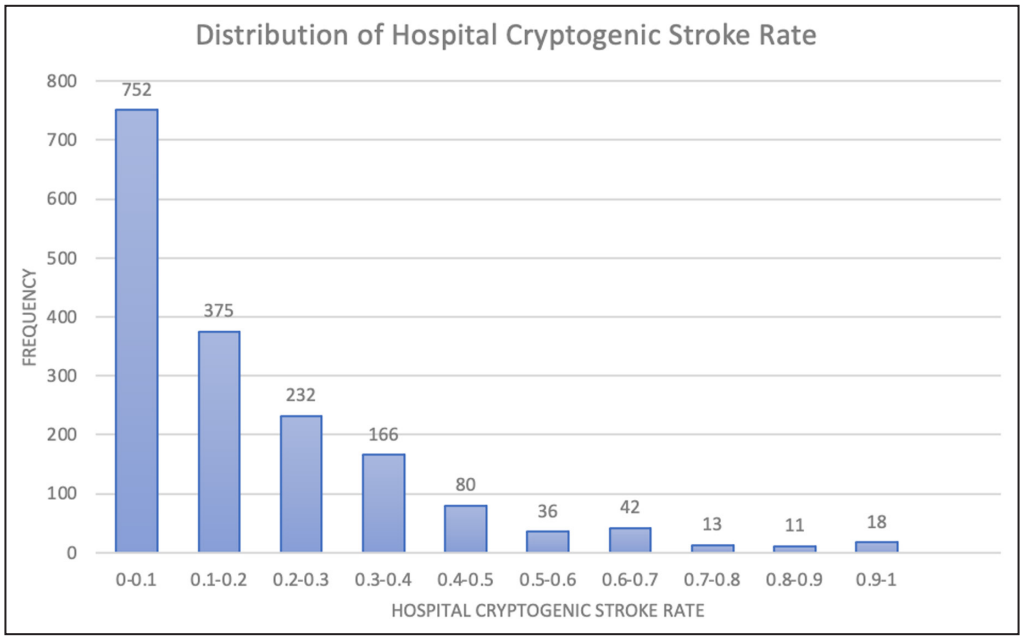

Figure 2. Histogram of the hospital-level rate of cryptogenic stroke out of total documented stroke pathogenesis.

was reported in 48.3%, intracranial vascular imaging in 40.9%, echocardiography in 50.7%, hypercoagulability testing in 8.2%, short-term cardiac rhythm monitoring in 44.7%, extended surface cardiac rhythm monitoring in 4.5%, and extended implantable cardiac rhythm monitoring in 1.6% of the patients. These data elements were missing in 40% to 45% of subjects.

Figure 3 shows, for each class of diagnostic testing, the proportion of patients with the test reported as performed during the hospitalization, not done during the hospitalization, planned following discharge, and missing by etiologic subtype. Carotid and intracranial vascular imaging (69.1% and 58.7%, respectively), echocardiography (74.3%), cardiac rhythm monitoring (76.2%), and hypercoagulability testing (14.2%) were reported more frequently with fewer missing data in patients with CS compared with other subtypes (Table 2 and Figure 3; $P<0.001$ for each comparison). Among patients with CS, short-term cardiac rhythm monitoring was reported in 65.7%, extended surface cardiac rhythm monitoring in 6.1%, and extended implantable cardiac rhythm monitoring in 4.4%.

DISCUSSION

In this study evaluating the reporting of IS etiologic subtype and diagnostic testing in US hospitals, we found that only 57% of all patients with IS had a etiologic stroke subtype reported. There was considerable variability in coding across participating hospitals, and 181 hospitals (9.5%) did not document stroke pathogenesis on any patients. Evidence-based treatments that form the basis of GWTG-Stroke quality and achievement measures were less common in patients with missing stroke pathogenesis, suggesting overall lower quality of care in these patients. Among patients with documented stroke pathogenesis, there was significant variability in reporting of CSs, with hospital level rates ranging from <10% to >90%. In our data, major diagnostic tests were reported in only 40% to 50% of patients, including echocardiography, extracranial and intracranial vessel imaging, and cardiac rhythm monitoring. These data elements were missing in 40% to 45% of subjects overall. Reporting of diagnostic evaluation was required in CS, and so, not surprisingly, there was less missingness in this group. Still, in CS, an echocardiogram was not performed in 10%, carotid imaging in 15%, and intracranial imaging in 25%. These rates are too high, given that a diagnosis of CS should occur only after testing to exclude other known causes of stroke. Rates of extended cardiac rhythm monitoring were particularly low, which may be problematic given the high incidence of atrial fibrillation in patients with CS.[5] Inadequate workup and failure to identify other stroke mechanisms may lead to suboptimal treatment and a failure to prevent future strokes.

While some aspects of secondary stroke prevention are broadly generalizable, such as blood pressure control and lipid management, optimizing secondary stroke prevention requires an appropriate and complete diagnostic evaluation. This should allow for more accurate etiologic subtyping and a lower risk of

Table 1. Comparison of Patient and Hospital Characteristics Between Patients Who Had Documented Stroke Pathogenesis and Patients Who Had Stroke Pathogenesis Missing

Characteristic	No. (%)			*P* value	% Std. Diff
	Overall (N=607 563)	Stroke pathogenesis missing (N=258 848)	Stroke pathogenesis documented (N=348 715)		
Demographics					
Age, y, median (IQR)	71.0 (60.0–81.0)	71.0 (60.0–82.0)	71.0 (60.0–81.0)	0.006	0.7
Female sex, n (%)	303 332 (49.9)	130 994 (50.6)	172 338 (49.4)	<0.001	2.4
Race and ethnicity,*n (%)				<0.001	14.9
Asian	18 106 (3.0)	7330 (2.8)	10 776 (3.1)		
Non-Hispanic Black	106 474 (17.5)	46 500 (18.0)	59 974 (17.2)		
Non-Hispanic White	412 130 (67.8)	174 146 (67.3)	237 984 (68.3)		
Hispanic	43 863 (7.2)	19 468 (7.5)	24 395 (7.0)		
Other*	26 243 (4.3)	10 741 (4.2)	15 502 (4.5)		
Missing	747 (0.1)	663 (0.3)	84 (0.0)		
Insurance status, n (%)				<0.001	31.8
Not documented	2447 (0.4)	1031 (0.4)	1416 (0.4)		
Self-pay/no insurance	21 710 (3.6)	8531 (3.3)	13 179 (3.8)		
Medicare	182 952 (30.1)	71 956 (27.8)	110 996 (31.8)		
Medicaid	59 657 (9.8)	23 648 (9.1)	36 009 (10.3)		
Private/other	214 196 (35.3)	81 142 (31.4)	133 054 (38.2)		
Missing	126 601 (20.8)	72 540 (28.0)	54 061 (15.5)		
Patient arrival, n (%)				<0.001	14.9
Not documented or unknown	4740 (0.8)	2527 (1.0)	2213 (0.6)		
Transfer from other hospital	100 713 (16.6)	35 797 (13.8)	64 916 (18.6)		
Private transport	206 131 (33.9)	91 965 (35.5)	114 166 (32.7)		
EMS	276 032 (45.4)	118 320 (45.7)	157 712 (45.2)		
Missing	19 947 (3.3)	10 239 (4.0)	9708 (2.8)		
Arrival during off-hours, n (%)	282 789 (46.5)	118 487 (45.8)	164 302 (47.1)	<0.001	2.7
Medical history, n (%)					
Atrial fibrillation/flutter	110 795 (18.6)	39 108 (15.9)	71 687 (20.6)	<0.001	12.1
Prosthetic heart valve	7205 (1.2)	2400 (1.0)	4805 (1.4)	<0.001	3.7
Prior stroke/TIA	182 904 (30.8)	76 380 (31.1)	106 524 (30.6)	<0.001	1.1
CAD/prior MI	137 085 (23.1)	55 174 (22.4)	81 911 (23.5)	<0.001	2.5
Carotid stenosis	20 261 (3.4)	7027 (2.9)	13 234 (3.8)	<0.001	5.2
Diabetes	209 901 (35.3)	87 805 (35.7)	122 096 (35.0)	<0.001	1.4
Peripheral vascular disease	25 766 (4.3)	9847 (4.0)	15 919 (4.6)	<0.001	2.8
Hypertension	449 800 (75.7)	183 953 (74.8)	265 847 (76.3)	<0.001	3.4
Heart failure	56 254 (9.5)	21 609 (8.8)	34 645 (9.9)	<0.001	4.0
Smoker	112 332 (18.9)	46 398 (18.9)	65 934 (18.9)	0.650	0.1
Medical history panel missing, n (%)	13 279 (2.2)	13 026 (5.0)	253 (0.1)	<0.001	31.8
NIHSS, median (IQR)	3 (1–9)	3 (1–8)	4 (1–9)	<0.001	7.0
NIHSS missing, %	11.3	13.9	9.3	<0.001	14.4
Hospital characteristics					
Volume of ischemic stroke admissions, median (IQR)	238.7 (160.4–373.8)	228.4 (152.9–346.7)	248.5 (165.7–391.1)	<0.001	15.9
Number of beds, median (IQR)	371.0 (236.0–583.0)	347.0 (220.0–524.0)	393.0 (253.0–625.0)	<0.001	19.9
Region, n (%)				<0.001	5.7

(*Continued*)

Table 1. Continued

Characteristic	No. (%)			*P* value	% Std. Diff
	Overall	Stroke pathogenesis missing	Stroke pathogenesis documented		
	(N=607 563)	(N=258 848)	(N=348 715)		
West	112 562 (18.5)	45 814 (17.7)	66 748 (19.1)		
South	248 296 (40.9)	107 679 (41.6)	140 617 (40.3)		
Midwest	120 650 (19.9)	50 950 (19.7)	69 700 (20.0)		
Northeast	126 055 (20.8)	54 405 (21.0)	71 650 (20.6)		
Teaching hospital, n (%)	458 538 (75.5)	185 651 (71.7)	272 887 (78.3)	<0.001	15.1
Rural location, n (%)	23 039 (3.8)	11 275 (4.4)	11 764 (3.4)	<0.001	5.1
Achievement measure, n (%)					
Onset to intravenous tPA by 3 h (if onset <2 h)	44 140 (85.5)	18 104 (83.8)	26 036 (86.8)	<0.001	8.4
Early antithrombotics	356 584 (97.0)	149 705 (96.1)	206 879 (97.7)	<0.001	9.6
Antithrombotics	498 588 (98.6)	208 603 (97.7)	289 985 (99.4)	<0.001	14.1
Anticoagulation for atrial fibrillation	77 415 (96.3)	25 663 (94.6)	51 752 (97.1)	<0.001	12.9
DVT prophylaxis	459 221 (99.2)	189 750 (99.2)	269 471 (99.3)	<0.001	1.8
Statin therapy	361 195 (98.4)	149 486 (98.0)	211 709 (98.7)	<0.001	6.0
Smoking cessation	95 778 (97.5)	39 212 (96.9)	56 566 (97.9)	<0.001	6.2
Defect-free care	532 517 (93.9)	221 278 (92.4)	311 239 (95.0)	<0.001	10.9
Quality measure, n (%)					
Dysphagia screen	457 829 (85.5)	191 292 (83.7)	266 537 (86.7)	<0.001	8.5
Stroke education	275 069 (95.8)	118 566 (94.5)	156 503 (96.9)	<0.001	11.8
Rehabilitation considered	509 224 (98.6)	213 541 (97.7)	295 683 (99.2)	<0.001	12.3
Door-to-intravenous tPA time ≤1 h	37 930 (80.7)	14 625 (76.5)	23 305 (83.6)	<0.001	17.6
LDL documented	475 734 (92.7)	196 942 (90.7)	278 792 (94.2)	<0.001	12.9
Intensive statin therapy	134 178 (70.7)	51 903 (65.4)	82 275 (74.6)	<0.001	20.1
Intravenous tPA by 4.5 h if onset to door <3.5 h	56 780 (77.6)	23 317 (74.7)	33 463 (79.8)	<0.001	12.1
NIHSS reported	492 039 (89.0)	203 228 (86.1)	288 811 (91.1)	<0.001	15.5
Other outcomes					
In-hospital death, n (%)	25 998 (4.3)	11 133 (4.3)	14 865 (4.3)	0.298	0.3
Length of stay, median (IQR), d	4.0 (2.0–6.0)	3.0 (2.0–5.0)	4.0 (2.0–6.0)	<0.001	11.9
Ambulatory status at discharge, n (%)				<0.001	48.9
Not documented	22 466 (3.7)	11 695 (4.5)	10 771 (3.1)		
Unable to ambulate	70 465 (11.6)	25 743 (10.0)	44 722 (12.8)		
With assistance from another person	166 631 (27.4)	62 277 (24.1)	104 354 (29.9)		
Able to ambulate independently	276 551 (45.5)	106 854 (41.3)	169 697 (48.7)		
Missing	71 450 (11.8)	52 279 (20.2)	19 171 (5.5)		
Discharge to home, n (%)	292 119 (50.8)	127 139 (52.2)	164 980 (49.8)	<0.001	4.9
mRS* at discharge, n (%)				<0.001	43.0
Not done	258 331 (42.5)	114 072 (44.1)	144 259 (41.4)		
Documented	321 942 (53.0)	118 605 (45.8)	203 337 (58.3)		
Missing	27 290 (4.5)	26 171 (10.1)	1119 (0.3)		
≤2	130 364 (40.9)	48 969 (42.4)	81 395 (40.1)	<0.001	4.7

CAD indicates coronary artery disease; DVT, deep vein thrombosis; EMS, emergency medical services; IQR, interquartile range; LDL, low-density lipoprotein; MI, myocardial infarction; mRS, Modified Rankin Scale; NIHSS, National Institute of Health Stroke Scale; Std. Diff, standardized difference; TIA, transient ischemic attack; and tPA, tissue plasminogen activator.

*Race and ethnicity are reported as the percentage of the total population in each group.

†Other: Native Hawaiian or Pacific Islander, American Indian or Alaska Native, and unable to determine.

Table 2. Tests Obtained for Stroke Pathogenesis Workup

	Overall, n (%)		Cryptogenic, n (%)		LAA, n (%)		Cardioembolism, n (%)		SVD, n (%)		OTH, n (%)		*P*-value
	(N=348 715)		(N=69 857)		(N=81 394)		(N=96 592)		(N=82 542)		(N=18 330)		
Cardiac ultrasound													<0.001
Not performed	27 078	(7.8)	7187	(10.3)	6300	(7.7)	6531	(6.8)	5621	(6.8)	1439	(7.9)	
Planned after discharge	696	(0.2)	265	(0.4)	114	(0.1)	144	(0.2)	149	(0.2)	24	(0.1)	
Performed during admission	176 704	(50.7)	51 903	(74.3)	35 977	(44.2)	44 985	(46.6)	36 573	(44.3)	7266	(39.6)	
Missing	144 237	(41.4)	10 502	(15.0)	39 003	(47.9)	44 932	(46.5)	40 199	(48.7)	9601	(52.4)	
Carotid imaging													<0.001
Not performed	35 076	(10.1)	10 723	(15.4)	6399	(7.9)	9318	(9.7)	7034	(8.5)	1602	(8.7)	
Planned after discharge	267	(0.1)	107	(0.2)	51	(0.1)	47	(0.1)	50	(0.1)	12	(0.1)	
Performed during admission	168 415	(48.3)	48 249	(69.1)	36 214	(44.5)	41 496	(43.0)	35 090	(42.5)	7366	(40.2)	
Missing	144 957	(41.6)	10 778	(15.4)	38 730	(47.6)	45 731	(47.3)	40 368	(48.9)	9350	(51.0)	
Intracranial vascular imaging													<0.001
Not performed	57 457	(16.5)	17 668	(25.3)	10 537	(13.0)	14 669	(15.2)	12 503	(15.2)	2080	(11.4)	
Planned after discharge	250	(0.1)	99	(0.1)	52	(0.1)	31	(0.0)	48	(0.1)	20	(0.1)	
Performed during admission	142 653	(40.9)	40 982	(58.7)	31 085	(38.2)	35 267	(36.5)	28 502	(34.5)	6817	(37.2)	
Missing	148 355	(42.5)	11 108	(15.9)	39 720	(48.8)	46 625	(48.3)	41 489	(50.3)	9413	(51.4)	
Short-term cardiac rhythm monitoring													<0.001
Not performed	42 472	(12.2)	12 256	(17.5)	9229	(11.3)	10 936	(11.3)	8254	(10.0)	1797	(9.8)	
Planned after discharge	961	(0.3)	502	(0.7)	94	(0.1)	227	(0.2)	115	(0.1)	23	(0.1)	
Performed during admission	155 889	(44.7)	45 886	(65.7)	31 954	(39.3)	38 777	(40.2)	32 288	(39.1)	6984	(38.1)	
Missing	149 393	(42.8)	11 213	(16.1)	40 117	(49.3)	46 652	(48.3)	41 885	(50.7)	9526	(52.0)	
Extended surface cardiac rhythm monitoring													<0.001
Not performed	160 532	(46.0)	48 063	(68.8)	33 472	(41.1)	39 184	(40.6)	32 990	(40.0)	6823	(37.2)	
Planned after discharge	9680	(2.8)	5279	(7.6)	905	(1.1)	2240	(2.3)	1056	(1.3)	200	(1.1)	
Performed during admission	15 810	(4.5)	4277	(6.1)	3687	(4.5)	4674	(4.8)	2299	(2.8)	873	(4.8)	
Missing	162 693	(46.7)	12 238	(17.5)	43 330	(53.2)	50 494	(52.3)	46 197	(56.0)	10 434	(56.9)	
Extended implantable cardiac rhythm monitoring													<0.001
Not performed	176 057	(50.5)	52 570	(75.3)	36 989	(45.4)	43 575	(45.1)	35 325	(42.8)	7598	(41.5)	
Planned after discharge	3125	(0.9)	1752	(2.5)	288	(0.4)	806	(0.8)	222	(0.3)	57	(0.3)	
Performed during admission	5706	(1.6)	3058	(4.4)	594	(0.7)	1439	(1.5)	494	(0.6)	121	(0.7)	
Missing	163 827	(47.0)	12 477	(17.9)	43 523	(53.5)	50 772	(52.6)	46 501	(56.3)	10 554	(57.6)	

Table 2. Continued

	Overall, n (%)		Cryptogenic, n (%)		LAA, n (%)		Cardioembolism, n (%)		SVD, n (%)		OTH, n (%)		*P*-value
	(N=348 715)		(N=69 857)		(N=81 394)		(N=96 592)		(N=82 542)		(N=18 330)		
Hypercoaguability testing													<0.001
Not performed	157 005	(45.0)	47 349	(67.8)	32 467	(39.9)	39 823	(41.2)	31 489	(38.2)	5877	(32.1)	
Planned after discharge	678	(0.2)	383	(0.6)	91	(0.1)	93	(0.1)	64	(0.1)	47	(0.3)	
Performed during admission	28 698	(8.2)	9908	(14.2)	5534	(6.8)	6120	(6.3)	5120	(6.2)	2016	(11.0)	
Missing	162 334	(46.6)	12 217	(17.5)	43 302	(53.2)	50 556	(52.3)	45 869	(55.6)	10 390	(56.7)	

LAA indicates large-artery atherosclerosis; OTH, stroke of other determined pathogenesis; and SVD, small-vessel disease.

stroke recurrence through the use of strategies that are subtype specific. For example, revascularization for cervical carotid stenosis, anticoagulation for atrial fibrillation, and patent foramen ovale closure all have high-quality evidence supporting their use in appropriately selected patients.[4] American Heart Association guidelines recommend cervical and intracranial vascular imaging, a transthoracic echocardiogram, ECG, and basic laboratory tests in most patients with stroke and extended cardiac rhythm monitoring in patients with unexplained IS.[4]

Importantly, our results represent the reporting of etiologic stroke subtype and diagnostic evaluation within GWTG. Although prior audits of GWTG data show that the data are generally representative with high levels of accuracy, the validity of the stroke subtype and diagnostic testing data elements has not been established.[10,11] It is possible that the reported data do not accurately reflect the diagnostic evaluation that occurred in the hospital. These data elements were relatively recently added to the GWTG registry, and they are not used for established GWTG quality achievement awards. Both of these factors could contribute to suboptimal data abstraction. Additionally, assigning a etiologic stroke subtype can be challenging. The criteria used in GWTG are based on the TOAST classification scheme.[8] The interobserver reliability of this classification scheme is moderate overall but may be low with inexperienced observers.[12–14] Additionally, the TOAST classification scheme has low interobserver reliability for CS, assigns more strokes as CSs than other classification schemes, and does not optimally describe the overlap between stroke mechanisms in individual patients.[12,15–17] Furthermore, some clinicians may be hesitant to assign a etiologic subtype, particularly if the stroke evaluation is incomplete at the time of hospital discharge. All of these issues may be barriers to the accurate documentation and reporting of etiologic stroke subtype in GWTG, contributing to the low overall rate of etiologic subtype reporting and the observed variability across hospitals. It is uncertain whether alternative stroke classification systems, such as the Causative Classification of Stroke or ASCOD (atherosclerosis, small-vessel disease, cardiac pathology, other causes, dissection) systems would fare better. Another alternative would be to simply collect more discrete data elements relevant to stroke mechanism without asking the local site to speculate about mechanism (ie, was there a greater than 50% stenosis in a vessel supplying the stroke). Future research is needed to evaluate whether refinement of the coding instructions or the use of an alternative stroke classification scheme can improve etiologic subtyping in GWTG-Stroke.

Our study found that patients with missing etiologic diagnoses less frequently received evidence-based

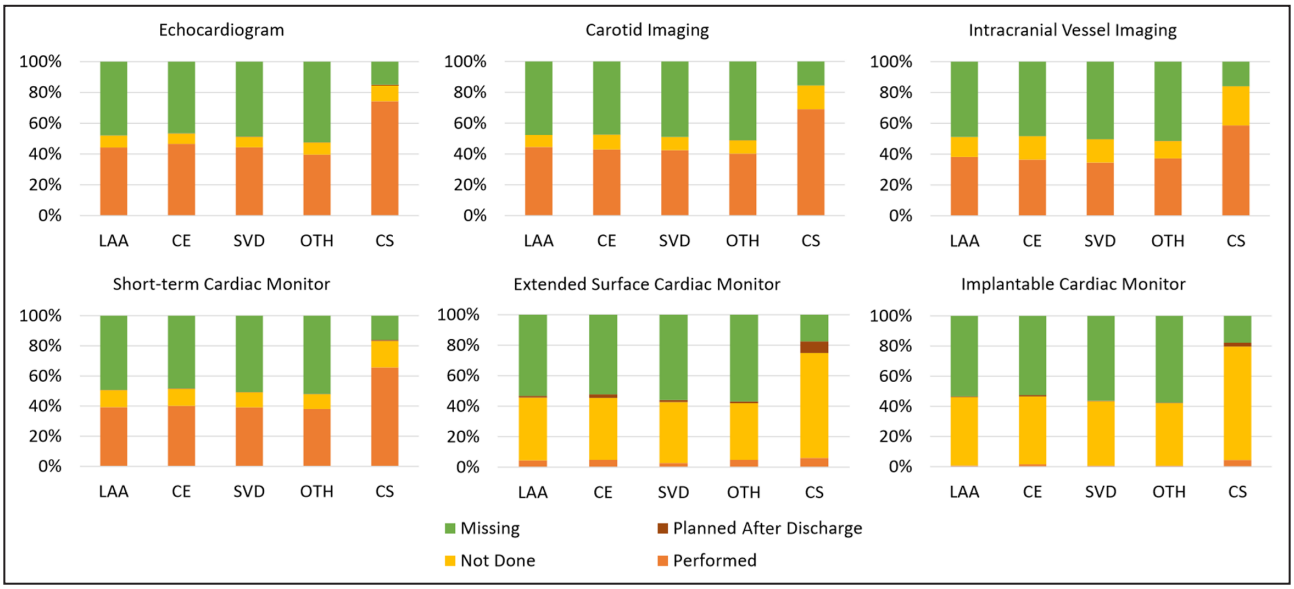

Figure 3. Diagnostic testing by IS etiologic subtype.
CE indicates cardioembolic; CS, cryptogenic stroke; IS, ischemic stroke; LAA, large-artery atherosclerosis; OTH, stroke of other determined pathogenesis; and SVD, small-vessel disease.

interventions that comprise GWTG achievement and quality measures. When comparing patients with a missing etiologic diagnosis and those with a documented diagnosis, there were minor differences in age, sex, and race and ethnicity. Of these, only race and ethnicity had a statistically meaningful effect size by standardized difference, and given the small absolute differences between groups, these differences may or may not be clinically meaningful. The Brain Attack Coalition's recent Inequalities in Care symposium identified disparities in the use of secondary stroke prevention strategies and noted that more data are required to describe and understand those disparities.[18] GWTG-Stroke has been an invaluable tool for studying and improving stroke care.[7,19] Given the importance of diagnostic testing to optimize secondary prevention, an effort to validate etiologic stroke subtype and diagnostic testing within GWTG should be undertaken. With validated data the GWTG-Stroke registry would become a powerful tool to identify potential disparities in evaluation and secondary stroke prevention and could be used to create targeted quality improvement initiatives to reduce those disparities.

Major strengths of our study include the large sample size and the use of a prospectively recorded database that has been demonstrated to have a high rate of accuracy in abstracted data. A potential weakness might stem from the lack of validation of stroke subtype abstraction in GWTG-Stroke, but that would not change the relevance of the findings related to the missing pathogenesis and relatively low reported rates of diagnostic testing. This study used data from the first 21 months after coding instructions for IS subtype were finalized. It is possible that coding of these variables could improve over time as data abstractors gain experience. Additional limitations include not having data regarding workup that may have occurred after discharge and long-term clinical outcomes. Further, these findings may not apply to hospitals and patients who differ from those participating in GWTG-Stroke.

CONCLUSIONS

In a large, contemporary, nationwide cohort, IS subtype was reported in just over half of records. Missing data were more common in older adults, women, and patients with Black/Hispanic race and ethnicity, although differences were small. Patients with missing etiologic subtype less frequently received evidence-based interventions. Reported rates of diagnostic testing were low, even among patients with CS, and only a minority of patients with CS received long-term cardiac monitoring. More research is needed to fully understand how patients with stroke are evaluated and whether accurate determination of pathogenesis is occurring, as these issues constitute important opportunities for quality improvement in the care of patients with stroke.

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