Psychiatric Hospitalization Increases Short-Term Risk of Stroke

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Background and Purpose—Recent evidence suggests that psychological distress, including the symptoms of psychiatric illness, may acutely increase the risk of stroke. However, existing studies are limited by small sample sizes, inherent recall bias, and poorly defined criteria for what constitutes psychological distress.

Methods—We analyzed administrative data from the Healthcare Cost and Utilization Project for the state of California from 2007 to 2009 using a case-crossover design. Conditional logistic regression was used to estimate the odds ratios (ORs) and 95% confidence intervals (95% CIs) for combined hemorrhagic and ischemic stroke risk occurring within 15, 30, 90, 180, and 365 days of a hospitalization for a psychiatric diagnosis (as defined by *International Classification of Diseases, Ninth Revision*, code) among adults.

Results—Psychiatric hospitalizations within 1 year before stroke were found in 2585 (5.3%) of 48 558 stroke patients. Hospitalization for a psychiatric condition was associated with increased risk of stroke within all 5 time periods, with the highest odds of stroke occurring within 15 days (0–15 days: OR, 3.5; 95% confidence interval [CI], 2.6–4.8; 0–30 days: OR, 3.0; 95% CI, 2.4–3.8; 0–90 days: OR, 2.3; 95% CI, 2.0–2.7; 0–180 days: OR, 2.2; 95% CI, 2.0–2.5; and 0–365 days: OR, 2.6; 95% CI, 2.4–2.8).

Conclusions—Psychiatric hospitalization increases the short-term risk of stroke, particularly within the 15-day period after hospitalization. **(*Stroke*. 2017;48:1795-1801. DOI: 10.1161/STROKEAHA.116.016371.)**

Key Words: confidence intervals ■ hospitalization ■ hypertension ■ sample size ■ stroke

Stroke remains a leading cause of morbidity and mortality in the United States.[1] Although medical comorbidities, such as hypertension and diabetes mellitus, have been researched extensively as risk factors for stroke, the role of acute precipitants of stroke, or triggers, remains poorly understood.[2] Research into novel risk factors or triggers for stroke is necessary,[3] with emerging research suggesting that psychosocial stress, including the symptoms of psychiatric illnesses, such as depression and anxiety, may acutely increase the risk of stroke.[4,5]

The relationship between chronic psychosocial stress and stroke risk is poorly understood. Prospective data, for example, revealed that depression is associated with long-term risk of incident stroke (mean follow-up 10 years).[6] A recent meta-analysis of 14 studies showed that perceived psychosocial stress (those exposed to general or work stress or to stressful life events) is independently associated with an increased risk of stroke.[7] Others, however, have demonstrated no significantly increased risk of stroke in patients with higher levels of stressful life events or greater degrees of negative affect.[8]

Recently, however, research has focused on whether or not stress may acutely, and transiently, increase the risk of stroke, thus acting as a stroke trigger.[9,10] Indeed, identifiable triggers were found in >44% of patients in 1 recent observational cross-sectional study, and psychological stress, more so than alcohol or infection, represented the most common precipitant.[11] Yet existing trigger studies are limited by sample size, recall bias, and poorly defined criteria for what constitutes stress.

In an effort to reduce the effect of recall bias, we used a case-crossover design, in which participants act as their own controls, with well-defined criteria for stress to investigate the relationship between psychosocial stress and stroke risk in a large administrative data set. We hypothesized that the risk of stroke would be highest immediately after psychiatric hospitalization, when psychological stress is likely to be most acute, and that the magnitude of association would decrease as time since the psychiatric hospitalization increases.

Methods

Study Setting and Inclusion Criteria

Publicly available data from the California State Inpatient Database, from the Healthcare Cost and Utilization Project 2007 to 2009, and from the Agency for Healthcare Research and Quality were used. This

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included the emergency department visit database (State Emergency Department Databases). Under Healthcare Cost and Utilization Project, claims data for each hospital discharge are collected, deidentified, and standardized from various states and then made available to researchers. The California State Inpatient Database contains data for all patients hospitalized in nonfederal acute care California hospitals. Data elements include demographic information, such as age, sex, race, and insurance payer. For each hospitalization, discharge diagnosis code (≤25 *International Classification of Diseases, Ninth Revision [ICD-9]* codes), month of discharge, length of stay in hospital, and Agency for Healthcare Research and Quality comorbidity measures are available. Primary diagnosis designations and indicators for conditions present on hospital arrival for each *ICD-9* code are included in the data sets to account for preexisting conditions as opposed to a complication that occurs during a hospitalization. Tracking of patients over time through multiple hospital admissions is possible through the use of a deidentified patient identification code classified as the visitlink variable.

The study population consisted of patients hospitalized with an ischemic or a hemorrhagic stroke in any nonfederal acute care hospital in California in the year 2009. We used a case-crossover design (Figure 1), in which each patient serves as his/her own control, to assess the relationship between the hospitalization or emergency department visit for a psychiatric condition and stroke in adults over the age of 18. This design is useful in studying acute events, such as stroke, brought on by exposures that transiently increase the risk for having an event, in this case transiently elevated psychiatric symptoms that brought on a psychiatric hospitalization.[12] Psychiatric hospitalizations within 15, 30, 90, 180, and 365 days of stroke onset (risk periods) were compared with control periods of equivalent duration in the 1 and 2 calendar years preceding the defined risk period. Psychiatric hospitalizations would be considered triggers of stroke if they were present more frequently in the risk period compared with the control period. In this study design, cases act as their own controls, and thus, the design inherently controls for interindividual variability and confounding.[5] Patients with preexisting depression and psychosis, as defined by the Agency for Healthcare Research and Quality comorbidity measure of depression and history of psychosis, were excluded from the analysis. Although such individuals are at increased risk of psychiatric hospitalization compared with those who are otherwise well, this analysis focuses on the acute effects of psychiatric hospitalization as opposed to the well-established effects of chronic mental illness as a risk factor for stroke. Cases in which both stroke and psychiatric diagnoses were present on arrival were also excluded. The Institutional Review Board at Columbia University Medical Center approved this study.

Exposure and Covariates

Psychiatric conditions were identified using validated *ICD-9* codes and included the following diagnoses: 296.20 to 296.25 (major depressive disorder, single episode), 296.30 to 296.35 (major depressive disorder, recurrent episode), 300.4 (dysthymic disorder), 311 (depressive disorder, not otherwise specified), 296.5 (bipolar I disorder, now depressed), 296.6 (bipolar I disorder, mixed), 296.82 (atypical depressive disorder), 296.90 (unspecified episodic mood disorder), 309.0 (adjustment reaction, including adjustment disorders and reaction to chronic stress), 309.1 (prolonged depressive reaction), 309.28 (adjustment disorder with mixed anxiety and depressed mood), 309.81 (post-traumatic stress disorder), 308.3 (acute stress disorder), E967.3 (abuse of spouse or partner by ex-spouse or ex-partner), 309.24 (adjustment disorder with anxiety), and 300.00 to 300.02 (anxiety states).[13] Patients were only classified as hospitalized with a psychiatric condition if psychiatric condition was the primary reason for hospitalization and presentation to the emergency department.

Psychiatric hospitalization before stroke was considered as an exposure event. Risk time periods assessed were 0 to 15, 0 to 30, 0 to 90, 0 to 180, and 0 to 365 days. In addition, we assessed the risk of stroke within the mutually exclusive time intervals 16 to 30, 31 to 90, 91 to 180, and 181 to 365 days.

Outcomes

Ischemic stroke was defined using *ICD-9* codes 433.x1 (x, the fourth digit, can vary to specify a specific arterial distribution), 434 (excluding 434.x0), or 436 present at any diagnostic position between DX1 and DX12. Cases were excluded if any traumatic brain injury *ICD-9*-Clinical Modification (CM) code (800–804, 850–854) or rehabilitation care *ICD-9-CM* code (V57) was present as the primary diagnosis. Hemorrhagic stroke was defined with *ICD-9* codes 430 to 432 present at any diagnostic position.

The California State Inpatient Database does not provide separable dates for each *ICD-9* code within the same hospitalization, potentially limiting the assignment of temporal relationships among events within each admission. Therefore, all admissions for which any kind of stroke and psychiatric condition occurred in the same admission during all 3 years of data were omitted. Thus, using the restricted data, all comparisons were only between separate admissions for stroke and psychiatric conditions.

Statistical Analysis

Conditional logistic regression stratified on the variable visitlink, a variable created within the database to link patients in the sample without identifying information, was used to compute odds ratios (ORs) and 95% confidence intervals (95% CIs) for any hospital admission with stroke within 0 to 15, 0 to 30, 0 to 180, and 0 to 365 days after exposure. It is important to note that the case-crossover analysis does not require adjustment of the ORs because each patient serves as his or her own control. We further assessed risk of stroke post-psychiatric condition hospital visit at nonoverlapping time intervals after psychiatric condition hospital visit by investigating the risk at 16 to 30, 31 to

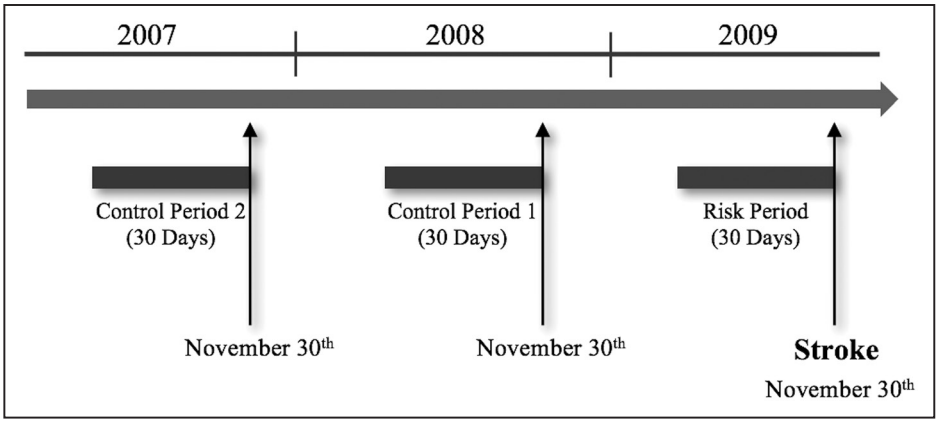

Figure 1. Case-crossover study design. Psychiatric hospitalization within specified time period (eg, 30 d) of stroke onset (risk period) is compared with control periods of equivalent duration in the 1 and 2 calendar years preceding the defined risk period. (In this example, stroke onset occurred on November 30.)

90, 91 to 180, and 181 to 365 days. Ischemic and hemorrhagic strokes were studied separately for each time period. Interactions between psychiatric condition and age and sex were conducted. Subsequent stratification by age was performed to assess whether psychiatric condition hospital visit is a trigger for stroke in the young.

Results

A total of 48 558 strokes were identified. There were 36 185 (74.5%) ischemic strokes and 12 373 (25.5%) hemorrhagic strokes. Psychiatric conditions diagnosed within 1 year of stroke (either ischemic or hemorrhagic) were found in 2585 (5.3%) patients. Patients with a recent psychiatric hospitalization (within 1 year) were more likely to be women (60% versus 50%) and to have had a longer stroke hospital course (median 5 versus 4 days) than patients without a recent psychiatric hospitalization. These patients also had increased proportions of Agency for Healthcare Research and Quality comorbidity measures, including diabetes mellitus, renal failure, and congestive heart failure (Table 1).

Hospitalization for or presenting to the emergency department with a psychiatric condition was associated with increased odds of stroke within all 5 predefined time periods (Table 2), with the highest odds of stroke occurring in those who most recently experienced a psychiatric visit (0–15 days: OR, 3.5; 95% confidence interval [CI], 2.6–4.8; 0–30 days: OR, 3.0; 95% CI, 2.4–3.8; 0–90 days: OR, 2.3; 95% CI, 2.0–2.7; 0–180 days: OR, 2.2; 95% CI, 2.0–2.5; and 0–365 days: OR, 2.6; 95% CI, 2.4–2.8). Thus, although the odds of stroke were highest in the short-term period, the risk of stroke remained elevated ≤1-year post-psychiatric hospitalization.

Nonoverlapping time periods were also analyzed (ie, 0–15 days, 15–30 days, etc) to better understand how the odds of stroke after psychiatric hospitalization change over time. This revealed (Figure 2) a time-dependent decrease in the risk of stroke that seemed to level off between 30 and 90 days (0–15 days: OR, 3.5; 95% CI, 2.6–4.8; 15–30 days: OR, 2.9; 95% CI, 2.1–4.0; 30–90 days: OR, 2.0; 95% CI, 1.7–2.4; 90–180 days: OR, 2.1; 95% CI, 1.7–2.5; and 180–365 days: OR, 1.9; 95% CI, 1.7–2.2).

Subgroup analyses based on stroke type revealed that the odds of hemorrhagic strokes in the 15-day time period were higher than the odds of ischemic strokes (0–15 days hemorrhagic stroke OR, 4.3; 95% CI, 2.4–7.8; and 0–15 days ischemic stroke OR, 3.2; 95% CI, 2.2–4.7); however, the odds of ischemic strokes were higher in the 30- and 90-day time periods (Table 2).

We found evidence of an interaction between psychiatric condition and age on the odds of stroke because the association of psychiatric diagnosis with stroke risk was higher in younger patients between the ages of 18 and 45 (*P* for interaction=0.09). To further explore the effects of this potential age interaction, the analyses were stratified by predefined age categories. Patients between the ages of 18 and 45 had higher odds of stroke in the 15-day period after psychiatric hospitalization (OR, 14.0; 95% CI, 1.7–113) compared with those between the ages of 45.1 and 64 (OR, 3.6; 95% CI, 2.1–6.0) and those older than age 65 (OR, 3.2; 95% CI, 2.1–4.8; Table 3). When the analyses were stratified by sex, there was no significant difference in stroke risk between female and male patients (180 days: OR, 2.06; 95% CI, 1.77–2.41 for women; and OR, 2.47; 95% CI, 2.03–3.01 for men; *P*=0.1609).

Discussion

In this case-crossover analysis of stroke patients from a large administrative data set, we found that patients hospitalized for a psychiatric condition have increased risk of stroke and that these odds are highest within the 15-day period after hospitalization.

The magnitude of this effect is particularly pronounced in younger patients (ages 18–45) for whom the odds of stroke were 14× greater in the 15-day time period after a psychiatric hospitalization. Although the CI was wide, this subgroup analysis dramatically decreases the sample size because of the lower prevalence of stroke in this age range (n=8).

The time-dependent decrease in the risk of stroke (Figure 2), with apparent equilibration of risk between 30 and 365 days, may indicate a transition from severe psychiatric illness acting as an acute trigger to more of a chronic risk factor. This is, to our knowledge, the first analysis to explore the dynamic nature of a risk factor over time.

Although the role of psychosocial stress on the short-term risk of acute myocardial infarction has been well established,[12,14] very few studies have examined the triggering effects of acute stress on stroke.[2] The lack of data in this domain is at least partly attributable to the lack of consensus on what distinguishes a trigger from more traditional, chronic risk factors.[4] As previously described, a trigger may be defined as an event that produces short-term physiological changes, which may lead directly to the onset of acute stroke. The hazard period, a related concept, is defined as the time interval after trigger initiation for which there is an associated increase in the risk of stroke.[15] To our knowledge, there are just 3 previous studies that have examined the role of psychosocial distress as a stroke trigger as defined by these criteria.

Using a case-crossover approach, Guiraud et al[16] found that stressful life events (bereavement, health problems, etc) nearly tripled the odds of stroke within the following month. Koton et al,[17] also using the case-crossover method, found that both negative emotions and anger increase the odds of stroke within the subsequent 2-hour period. Last, Yoo et al[18] identified a significant association between psychological distress (within the 3 days before stroke onset) and stroke, with an increased risk of hemorrhagic versus ischemic events. Our findings are thus consistent with these previous investigations.

In our analysis, younger patients (ages 18–45) had dramatically increased odds of stroke compared with older patients. Because of the paucity of data on psychosocial stressors as triggers of stroke, it is worth highlighting parallels with other well-described triggers. Previous research, for example, has shown that young adults (under 50 years of age) presenting with acute stroke had higher rates of febrile infection in the preceding month than age-matched controls.[19] Recent data from our group indicate that infections present on admission in young stroke patients are closely associated with National Institutes of Health Stroke Scale and subsequent poor outcomes at discharge.[20] Thus, younger patients may be

Table 1. Baseline Characteristics of Stroke Patients With Hospitalization for Psychiatric Conditions Within 1 Year of Hospitalization for Stroke

Variable	Stroke Admissions With Recent Hospitalization for Psychiatric Conditions (n=2585)	Stroke Admissions Without Recent Hospitalization for Psychiatric Conditions (n=45 973)
Age, y	70.27	70.39
SD (range)	14.85 (15.00–99.00)	15.69 (0–104.00)
No. of chronic conditions, mean (SD)	7.28 (2.95)	6.43 (2.83)
Median (range)	7.00 (1.00–20.00)	6.00 (1.00–20.00)
Length of stay, d, mean (SD)	8.30 (14.27)	6.77 (11.43)
Median (range)	5 (0–300.00)	4 (0–356)
Women, n (%)	1542 (59.67)	22 735 (49.67)
Race n (%)		
White	1505 (60.66)	25 277 (57.72)
Black	275 (11.08)	4269 (9.75)
Hispanic	485 (19.55)	8557 (19.54)
Asian/pacific Islander	178 (7.17)	4768 (10.89)
Native American	2 (0.08)	22 (0.05)
Other	36 (1.45)	897 (2.05)
AHRQ comorbidity measures		
Valvular disease	225 (8.70)	3521 (7.66)
Solid tumor without metastasis	53 (2.05)	906 (1.97)
Metastatic cancer	53 (2.05)	887 (1.93)
Renal failure	537 (20.77)	6509 (14.16)
Congestive heart failure	475 (18.38)	5971 (12.99)
Chronic pulmonary disease	486 (18.80)	6086 (13.24)
Coagulopathy	153 (5.92)	2355 (5.12)
Peripheral vascular disorders	226 (8.74)	3815 (8.30)
Paralysis	235 (9.09)	3174 (6.90)
Pulmonary circulation disorders	83 (3.21)	1207 (2.63)
Other neurological disorders	137 (5.30)	1713 (3.73)
Hypertension (combine uncomplicated and complicated)	1978 (76.52)	35 459 (77.13)
Drug abuse	97 (3.75)	1262 (2.75)
Diabetes mellitus with chronic complications	285 (11.03)	3410 (7.42)
Diabetes mellitus, uncomplicated	728 (28.16)	11 063 (24.06)
Lymphoma	28 (1.08)	279 (0.61)
Weight loss	151 (5.84)	2178 (4.74)
Rheumatoid arthritis/collagen vascular diseases	70 (2.71)	994 (2.16)
Alcohol abuse	124 (4.80)	2118 (4.61)
Obesity	253 (9.79)	3669 (7.98)
Acquired immune deficiency syndrome	8 (0.31)	65 (0.14)

AHRQ indicates Agency for Healthcare Research and Quality.

particularly susceptible to the damaging effects of stress and infection that confer an acutely increased risk of stroke.

The mechanism(s) through which severe psychiatric illness increases the stroke risk are incompletely understood. Stress hormone receptors were first identified in the brain >40 years ago, and stress has been shown to have significant effects on hippocampal remodeling, including myriad roles in modifying neuronal architecture, altering gene expression, and maintaining the cytoskeleton.[21] How stress influences the neurovasculature, and stroke risk in particular, is less clear. Direct effects

Table 2. Cumulative Association of Hospitalization for Psychiatric Condition With Risk of Overall Stroke, Ischemic Stroke, and Hemorrhagic Stroke

Hospitalization for Psychiatric Condition	All Stroke, OR (95% CI)	Ischemic Stroke, OR (95% CI)	Hemorrhagic Stroke, OR (95% CI)
Within 15 days before stroke	3.52 (2.57–4.84); n=169	3.20 (2.20–4.66); n=117	4.29 (2.37–7.76); n=52
Within 30 days before stroke	3.01 (2.39–3.80); n=308	3.11 (2.35–4.11); n=216	2.74 (1.80–4.19); n=92
Within 90 days before stroke	2.33 (2.01–2.71); n=725	2.41 (2.00–2.89); n=502	2.17 (1.66–2.84); n=223
Within 180 days before stroke	2.21 (1.96–2.50); n=1174	2.15(1.85–2.48); n=823	2.35 (1.89–2.93); n=351
Within 365 days before stroke	2.57 (2.40–2.75); n=2585	2.48 (2.28–2.69); n=1934	2.78 (2.45–3.16); n=651

CI indicates confidence interval; and OR, odds ratio.

of stress on the vasculature, through inflammation, oxidative stress, and immune dysfunction, encompass 1 possible explanation.[22] Indirect effects, via catecholamine release and thus increased sympathetic activation, have also been proposed.[23]

As shown in Table 1, patients with a recent psychiatric hospitalization have more medical comorbidities (renal failure, congestive heart failure, diabetes mellitus, obesity, etc) compared with those without. Although these differences could represent some sort of systematic bias, the finding is consistent with previous research demonstrating that patients with affective, anxiety, and depressive disorders have higher rates of chronic medical conditions than those without a psychiatric illness.[24,25] Because each patient is compared with himself or herself in the case-crossover analysis, any confounding effects of these medical comorbidities on stroke risk are mitigated if not removed completely. It should be noted, however, that the increased risk of stroke with older age is not controlled for in the case-crossover study design. Thus, one cannot exclude the possibility that the likelihood of stroke during the risk period (Figure 1) is higher than that during the control periods because the patient is simply older. Nevertheless, we think that the increased odds of stroke after psychiatric hospitalization cannot be fully explained by this marginal increase in patient age.

Behavioral mechanisms, such as medication noncompliance, could also play a role. When compared with those without, patients with depression are 3× more likely to be noncompliant with medical treatment recommendations.[26] This degree of noncompliance could significantly increase one's risk of cardiovascular disease. For example, among patients treated with drug-eluting stents for acute myocardial infarction, those discontinuing thienopyridine therapy in the first 30 days after myocardial infarction had significantly higher mortality compared with those who did not.[27] Further research is needed to address how the diagnosis of a psychiatric condition affects medication adherence in patients at risk for stroke.

The strengths of this study include utilization of a large data set with many patients, strictly defined criteria for what constitutes psychiatric stress given *ICD-9* diagnoses, and minimal ambiguity in terms of defining time periods. Whereas previous trigger studies have been limited by recall bias, our study relied on *ICD-9* codes entered by the medical team at the time of arrival as opposed to the patient's recollection of how they were feeling at some distant time in the past. This allows for a more accurate assessment of the relationship between the onset of psychiatric symptoms and the onset of stroke. It also establishes a minimum threshold for the severity of psychiatric distress because all patients included in the analyses required hospitalization. Our approach thus represents the most rigorous to date in examining the role of severe psychiatric illness as a trigger of acute stroke.

Our study is not without limitations. Most importantly, although there was a strong association between psychiatric hospitalization and subsequent risk of stroke, we are unable to comment on whether there is a direct, causal relationship between the 2. In addition, detailed clinical information and

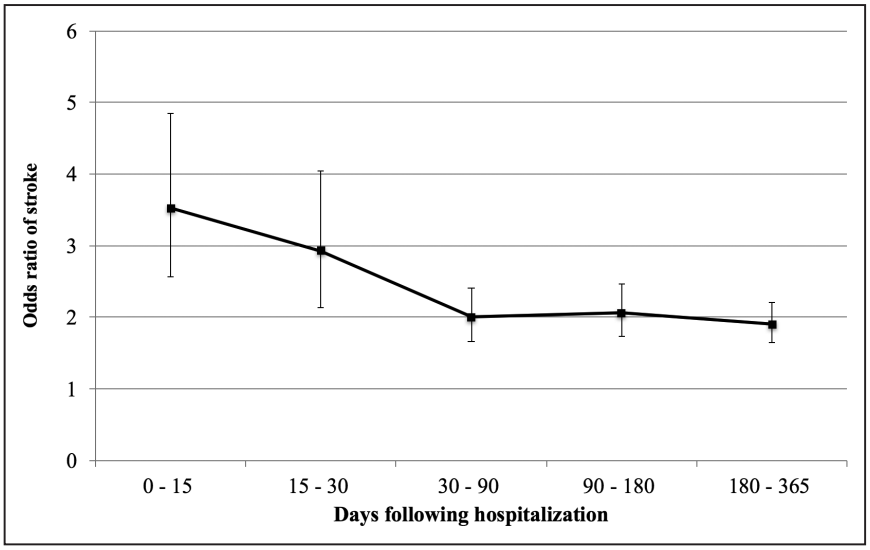

Figure 2. Association of hospitalization for psychiatric condition at each specific time interval with risk of stroke. Error bars represent 95% confidence intervals. Horizontal axis not to scale.

Table 3. Cumulative Association of Hospitalization for Psychiatric Condition With Risk of Overall Stroke, Stratified by Age Category

Hospitalization for Psychiatric Condition	Age 18–45, OR (95% CI)	Age 45.1–64, OR (95% CI)	Age ≥65, OR (95% CI)
Within 15 days before stroke	14.00 (1.72–113.79); n=8	3.58 (2.12–6.02); n=63	3.21 (2.13–4.84); n=98
Within 30 days before stroke	4.00 (1.37–11.70); n=15	3.09 (2.06–4.64); n=105	2.90 (2.16–3.90); n=188
Within 90 days before stroke	2.09 (1.06–4.14); n=36	2.46 (1.88–3.23); n=232	2.29 (1.90–2.77); n=457
Within 180 days before stroke	2.02 (1.12–3.62); n=49	2.12 (1.70–2.65); n=361	2.27 (1.95–2.64); n=764
Within 365 days before stroke	3.04 (2.18–4.25); n=117	2.61 (2.30–2.96); n=783	2.52 (2.32–2.75); n=1680

CI indicates confidence interval; and OR, odds ratio.

prospective case ascertainment for stroke were not available. For example, we could not assess the relationship between a specific psychiatric diagnosis (eg, depression, psychosis, etc) and stroke risk. We relied on administrative data diagnosis codes, and information on stroke severity was not available. We were only able to capture patients who were hospitalized or who presented to the emergency department for stroke or a psychiatric condition in the state of California. Patients who had been hospitalized in other states, or who died of an event before being admitted to a hospital, were not captured. Another limitation results from possible observation bias. Patients hospitalized for a psychiatric condition, for example, may have close medical follow-up, which could lead to a higher rate of identification of stroke signs and symptoms.

The case-crossover design also does not account for the increased risk associated with the aging of the patient over time and their concomitant development of new risk factors. To minimize these concerns, however, we limited the control time windows to the previous 1 year. We also assumed that psychiatric hospitalization would serve as a surrogate of psychological distress.

We could also not account for the possibility of medication-related effects. It is unknown, for example, how many patients initiated psychoactive medications as a result of their hospitalization for a psychiatric condition. Antidepressant medications, including selective serotonin reuptake inhibitors and serotonin–norepinephrine reuptake inhibitors, have been associated with an increased risk of both stroke and transient ischemic attack.[28] Moreover, the magnitude of this effect may be dependent on the duration of antidepressant treatment, with new users (but not long-term users) having an increased risk of stroke.[29] The increased risk of stroke after psychiatric hospitalization may therefore be at least partially attributable to the initiation of a new antidepressant medication.

Antipsychotics have also been associated with an increased risk of stroke. Indeed, multiple studies have suggested a time-dependent increase in the risk of stroke in patients recently initiated on psychiatric drugs.[30] Most of these studies were conducted in elderly populations (greater than age 65) in whom antipsychotics were initiated for the treatment of dementia so direct comparison with our patient cohort is not possible. Yet, even in patients without recorded dementia, antipsychotics—and atypical antipsychotics in particular—seem to confer an increased risk of stroke.[31]

Last, it is important to consider the possibility that stroke may actually lead to—rather than result from—psychiatric symptoms. Although poststroke depression occurs in approximately one third of all stroke survivors, isolated psychiatric symptoms as the primary manifestation of stroke are quite rare.[32,33] Moreover, although infarcts involving the pallidum and caudate have been shown to predispose patients to poststroke depression, it is unlikely that a lesion affecting these key subcortical regions would produce isolated psychiatric symptoms at the time of presentation without any concomitant effects on motor function.[34] It is therefore unlikely that patients presenting with covert stroke were misdiagnosed as having a primary psychiatric illness.

Summary and Conclusions

Psychiatric hospitalization increases the short-term risk of stroke, particularly within the 15-day period after the diagnosis. This effect is most pronounced in younger patients, under the age of 45.

Disclosures

Dr Kronish has received career development awards from the American Heart Association and National Heart, Lung, and Blood Institute. The other authors report no conflicts.

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