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Antihypertensives Are Administered Selectively in Emergency Department Patients with Subarachnoid Hemorrhage

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Abstract

Elevated blood pressure is common in patients with acute subarachnoid hemorrhage (SAH). American Heart Association guidelines do not specify a blood pressure target, but limited data suggest that systolic blood pressure (SBP) ≥ 160 mmHg is associated with increased risk of rebleeding and neurologic decline. In a population-based study, we determined the frequency of antihypertensive therapy in emergency department (ED) patients with SAH and the proportion of those patients with SBP ≥ 160 mmHg who received this therapy. In 2005, nontraumatic SAH cases were retrospectively ascertained at 16 hospitals in our region by screening for International Classification of Diseases Ninth Revision diagnostic codes 430–436. Blood pressure was recorded at ED presentation and also before and after any treatment with antihypertensives. Hypotension was defined as SBP <100 mmHg. The Mann-Whitney *U* test and χ^2 test were used for comparisons. Our cohort comprised 82 patients with SAH presenting to an ED; 4 patients were excluded. The median age of the included patients was 54 years, 74.4% were female, 29.5% were black, and 31 (39.7%) had SBP ≥ 160 mmHg. Antihypertensive therapy was given to 22 of 31 patients (70.9%) with SBP ≥ 160 mmHg and to 4 of 47 patients (8.5%) with SBP <160 mmHg. No patients became hypotensive after receiving treatment. Age, sex, Glasgow Coma Scale score, and National Institutes of Health Stroke Scale score were similar between treated and untreated patients. In the absence of definitive evidence, current blood pressure management in local EDs appears reasonable. Further studies of blood pressure management in acute SAH are warranted.

Keywords

Blood pressure; hypertension; stroke; hemorrhagic stroke

Each year in the United States, approximately 30,000 persons suffer nontraumatic subarachnoid hemorrhage (SAH),^{1,2} and the mortality rate after SAH is as high as 45%.^{3,4} As many as half of all patients with SAH are found to have hypertension on arrival to the

emergency department (ED).³ Evidence from observational studies suggests a decreased risk of rebleeding in patients with aneurysmal SAH treated with antihypertensive agents compared with those not treated,⁵ and systolic blood pressure (SBP) >160 mmHg has been associated with increased risk of rebleeding in patients admitted to the hospital with SAH.⁶ Rebleeding is a feared consequence of SAH that is associated with increased morbidity and mortality; however, there is little published research on controlling blood pressure in patients with SAH in a presumed unsecured aneurysm.

The most recent American Heart Association guidelines for the management of SAH recommend that “blood pressure should be monitored and controlled to balance the risk of stroke, hypertension-related rebleeding, and maintenance of cerebral perfusion pressure.”¹ Current practice regarding blood pressure management in ED patients with SAH is poorly described, and how closely these guidelines are being followed is unknown.

In this study, we examined the frequency of antihypertensive administration in ED patients with nontraumatic SAH, and the proportion of those patients with SBP ≥160 mmHg who received antihypertensive therapy. We hypothesized that antihypertensive agents would be frequently administered in the ED to patients presenting with SAH independent of SBP ≥160 mmHg because of concerns about rebleeding in a presumed unsecured aneurysm.

Methods

In the Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS), a large population-based epidemiologic study of stroke, the study population is defined as the 1.3 million residents of the greater Cincinnati/northern Kentucky region, which includes 2 southern Ohio counties and 3 contiguous northern Kentucky counties that border the Ohio River. This area includes 16 hospitals. Although residents of nearby counties seek care at these 16 hospitals, only residents of the 5 GCNKSS study area counties were included as cases. The study period extended from January 1, 2005, to December 31, 2005.

The methods of case ascertainment and data collection have been reported previously.^{7,8} In brief, study nurses retrospectively reviewed the records of every inpatient in our large, biracial population with a primary or secondary stroke-related International Classification of Diseases, Ninth Revision discharge diagnosis (codes 430–436) from the 16 acute care hospitals in the GCNKSS study area. When a case of stroke or transient ischemic attack was identified, a study nurse abstracted the medical record using a standardized case report form. Abstracted data included symptoms and duration, point of first health care provider contact (eg, 911/emergency medical services, ED, primary doctor), ED physical examination findings including vital signs, medical and surgical history, home medications, social history/habits, pre-event and postevent functional status measured using the modified Rankin Scale (mRS) score, retrospective National Institutes of Health Stroke Scale (NIHSS) from chart review, Glasgow Coma Scale (GCS) score, diagnostic tests performed in the ED or during hospitalization, treatments, and 30-day mortality.

Study physicians reviewed each abstract and determined whether a stroke or transient ischemic attack had occurred. The physicians assigned a stroke subtype and mechanism to each verified case based on all available information, using published definitions.^{7,9} The analysis reported here included patients aged 18 or older diagnosed with SAH and evaluated in an ED within the GCNKSS study area. Blood pressure was recorded at ED presentation, as well as before and after any antihypertensive treatment (up to a maximum of 3 doses of medication). Hypertension was defined as SBP ≥160 mmHg; hypotension, as SBP <100 mmHg. Institutional Review Board approval was obtained from all participating hospitals.

Data management and descriptive and comparative analyses were performed using SAS versions 8.02 and 9.2 (SAS Institute, Cary, NC), and SPSS version 18.0 (SPSS Inc, Chicago, IL). The χ^2 and Mann-Whitney *U* tests were used for comparisons.

Results

In 2005, a total 94 cases of SAH were recorded in the GCNKSS study area. Of these 94 patients, 82 were seen in an ED, 7 were inpatients, 1 was a direct admission from a nursing home, 2 were direct admissions from a primary care office, 1 died during emergency medical system transport, and 1 was identified at the coroner's office who had been managed as an outpatient. Two patients were under age 18 years. Another 2 patients initially presented to a hospital outside of the GCNKSS study areas.

Table 1 presents demographic data for the 78 included patients. Median age was 54 years (range, 22–95 years), 74% were female, 30% were black, median GCS score was 15 (range, 3–15), and median NIHSS score was 1 (range, 0–37). Twenty-six patients (33%) were treated with an antihypertensive agent in the ED, and the other 52 patients (66%) were not treated. There were no differences in age, sex, race, median NIHSS and GCS scores, history of hypertension, other medical history, and 30-day mortality between the patients who received anti-hypertensive therapy and those who did not (Table 2). Of the 26 treated patients, 22 had SBP $\geq$ 160 mmHg at ED presentation. Four of 47 (9%) patients with SBP <160 mmHg on arrival at the ED received antihypertensive therapy. Thirty-one patients (40%) had SBP $\geq$ 160 mmHg at ED presentation, 22 (71%) of whom received antihypertensive therapy (Table 3). In these patients, the median SBP at ED presentation was 192 mmHg (range, 165–245 mmHg) in those who received antihypertensive therapy, compared with 183 mmHg (range, 162–241 mmHg) in those who did not ($P = .685$).

None of the 26 patients who received antihypertensive therapy became hypotensive after treatment. However, 4 of the 26 patients who were treated did not have a post-treatment blood pressure recorded in the ED. In the treated patients, labetalol ($n = 14$) and nitroprusside ($n = 11$) were the most frequently used agents. Hydralazine was used in 3 patients, nicardipine in 2 patients, nifedipine in 2 patients, and quinapril in 1 patient.

Discussion

To the best of our knowledge, this is the first report to describe population-wide practice patterns for ED blood pressure management in patients with SAH. We found that 40% of the patients with SAH had SBP $\geq$ 160 mmHg at ED presentation. Two-thirds of these patients received antihypertensive therapy in the ED, compared with only 9% of patients with SBP <160 mmHg received antihypertensive therapy. Thus, the ED physicians in our community appeared to administer antihypertensive therapy selectively to patients with SAH with SBP $\geq$ 160 mmHg in the ED. This is contrary to our hypothesis that antihypertensives would be administered independent of presenting blood pressure because of concerns about rebleeding in a presumed unsecured aneurysm. In the absence of evidence-based guidelines for blood pressure management in SAH, we believe the practice pattern we observed to be reasonable.

In the treated patients, labetalol and vasodilators (hydralazine and nitroprusside) were the most frequently administered agents. Although no antihypertensive medication has been demonstrated to be superior to another, the American Heart Association recommends the use of nicardipine, labetalol, or esmolol and cautions against the use of nitroprusside because of its tendency to raise intracranial pressure and cause toxicity with prolonged infusion.¹ Further studies are warranted to determine whether the use of nitroprusside observed in our study persists, and whether its use has any impact on patient outcomes.

This study is limited by its use of retrospective data collection. Although our sample size was small, this was a complete sampling of every case of SAH occurring in our region over a 1-year period, in a population that is representative of the United States in terms of percentage of black residents, socioeconomic status, and educational level.^{3,8} We also collected only up to 4 blood pressure measurements, and thus may have missed some patients who became hypertensive later in the ED but did not receive therapy. Similarly, blood pressure normalization might explain why 9 hypertensive patients did not receive antihypertensive therapy. Finally, given our small sample size, we were not able to draw conclusions regarding the impact of blood pressure management on patient outcomes. In particular, we did not examine whether antihypertensive therapy decreased the occurrence of rebleeding in our study population. Our goal was to describe practice patterns in the community, not to assess the impact of these practice patterns on outcomes. Any speculation on outcomes and rebleeding rates would be limited by not accounting for aneurysmal size, location, and other factors that also affect the risk of rebleeding. Such an in-depth exploration is beyond the scope of this study.

In conclusion, in the absence of definitive evidence, current BP management in local EDs appears reasonable, with the exception of the high rate of nitroprusside use. Further studies of blood pressure management in patients with acute SAH are warranted.

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Table 1

Demographic data for the patients with SAH (n = 78)

Characteristic	Value
Age, years, median (range)	54 (22–95)
Female, n (%)	58 (74.4)
Black, n (%)	23 (29.5)
Estimated NIHSS score, median (range)	1 (0–37)
GCS, median (range)	15 (3–15)
Prestroke modified Rankin Scale score, median (range)	0 (0–4)
History of unmedicated hypertension, n (%)	16 (20.5)
On antihypertensive medication, n (%)	27 (34.6)
History of diabetes, n (%)	12 (15.4)
History of coronary disease, n (%)	13 (16.7)
History of atrial fibrillation, n (%)	3 (3.8)
30-day mortality, n (%)	20 (25.6)

Table 2

Comparison of treated and untreated patients

	Treated (n = 26)	Untreated (n = 52)	P value
Age, years, median (range)	57 (39–86)	52 (12–95)	.221
Female, n (%)	22 (84.6)	36 (69.2)	.176
Black, n (%)	11 (42.3)	12 (23.1)	.114
Estimated NIHSS score, median (range)	2.5 (0–35)	1 (0–37)	.383
0, n (%)	6 (23)	23 (44.2)	
1–3, n (%)	8 (30.8)	9 (17.3)	
4–10, n (%)	5 (19.2)	6 (11.5)	
11–15, n (%)	3 (11.5)	3 (5.8)	
16+, n (%)	4 (15.4)	11 (21.2)	
GCS, median (range)	14 (3–15)	15 (3–15)	.570
15, n (%)	12 (46.2)	32 (61.5)	
13–14, n (%)	4 (15.4)	3 (5.8)	
8–12, n (%)	4 (15.4)	3 (5.8)	
4–8, n (%)	5 (19.2)	4 (7.7)	
3, n (%)	1 (3.8)	10 (19.2)	
SBP 160 mmHg at presentation, n (%)	22 (84.6)	9 (17.3)	.,001
Initial SBP, median (range)	192 (165–245)	183 (162–241)	.685
30-day mortality, n (%)	5 (19.2)	15 (28.8)	.421

Table 3Comparison of treated and untreated patients with SBP $\geq$ 160 mmHg

	Treated (n = 22)	Untreated (n = 9)	P value
Age, years, median (range)	58 (39–86)	68 (39–86)	.716
Female, n (%)	19 (86.4)	7 (77.8)	.613
Black, n (%)	10 (45.5)	2 (22.2)	.418
Estimated NIHSS score, median (range)	3.5 (0–35)	1 (0–32)	.203
GCS, median (range)	15 (3–15)	14 (3–15)	.356
Initial SBP, mmHg, median (range)	192 (165–245)	183 (162–241)	.685
30-day mortality, n (%)	5 (22.7)	3 (33.3)	.949