Severe inpatient hypertension prevalence and blood pressure response to antihypertensive treatment

Lama Ghazi MD, PhD1 | Fan Li PhD2 | Xinyuan Chen PhD3
Michael Simonov MD1 | Yu Yamamoto MS1 | Aditya Biswas MS1
Jonathan Hanna MD4 | Tayyab Shah MD4 | Raymond Townsend MD5
Aldo Peixoto MD6,† | F. Perry Wilson MD, MSCE1,†

1 Department of Internal Medicine, Clinical and Translational Research Accelerator, Yale University, New Haven, Connecticut, USA
2 Department of Biostatistics, Yale School of Public Health, New Haven, Connecticut, USA
3 Department of Mathematics and Statistics, Mississippi State University, Mississippi State, Mississippi, USA
4 Department of Internal Medicine, Yale School of Medicine, Yale University, New Haven, Connecticut, USA
5 Department of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, USA
6 Department of Internal Medicine, Section of Nephrology, Yale School of Medicine, and the Hypertension Program, Yale New Haven Hospital Heart and Vascular Center, New Haven, Connecticut, USA

Correspondence
F. Perry Wilson MD, MSCE, 60 Temple St, Ste 6C, New Haven, CT 06510, USA.
Email: francis.p.wilson@yale.edu

†These authors contributed equally to this work.

Funding information
National Institutes of Health, Grant/Award Numbers: P30DK079310, R01DK113191; American Heart Association Postdoctoral Fellowship award, Grant/Award Number: 829804; Robert E. Leet and Clara Guthrie Patterson Trust Mentored Research Award

Abstract
Severe hypertension (HTN) that develops during hospitalization is more common than admission for HTN; however, it is poorly studied, and treatment guidelines are lacking. Our goal is to characterize hospitalized patients who develop severe HTN and assess blood pressure (BP) response to treatment. This is a multi-hospital retrospective cohort study of adults admitted for reasons other than HTN who developed severe HTN. The authors defined severe inpatient HTN as the first documented BP elevation (systolic BP > 180 or diastolic BP > 110) at least 1 hour after admission. Treatment was defined as receiving antihypertensives (intravenous [IV] or oral) within 6h of BP elevation. As a measure of possible overtreatment, the authors studied the association between treatment and time to mean arterial pressure (MAP) drop ≥ 30% using the Cox proportional hazards model. Among 224,265 hospitalized adults, 10% developed severe HTN of which 40% were treated. Compared to patients who did not develop severe HTN, those who did were older, more commonly women and black, and had more comorbidities. Incident MAP drop ≥ 30% among treated and untreated patients with severe HTN was 2.2 versus 5.7/1000 person-hours. After adjustment, treated versus untreated patients had lower rates of MAP drop ≥ 30% (hazard rate [HR]: 0.9 [0.8, 0.99]). However, those receiving only IV treatment versus untreated had greater rates of MAP drop ≥ 30% (1.4 [1.2, 1.7]). Overall, the authors found that clinically significant MAP drop is observed among inpatients with severe HTN irrespective of treatment, with greater rates observed among patients treated only with IV antihypertensives. Further research is needed to phenotype inpatients with severe HTN.

KEYWORDS
antihypertensive therapy, blood pressure response, electronic health records, hypertension, inpatient
Hypertension (HTN) is common in hospitalized patients with prevalence rates up to 72%. In patients who are admitted for severe HTN (systolic/diastolic blood pressure [SBP/DBP] > 180/110 mm Hg) with acute end organ damage, that is, hypertensive emergency, immediate intravenous (IV) pharmacotherapy under intensive care monitoring is recommended to limit progressive injury. However, far more common is incident severe HTN in already hospitalized patients occurring during an admission unrelated to HTN. Increased blood pressure (BP) in ambulatory patients is associated with increased risk of cardiovascular disease (CVD) related outcomes including stroke, myocardial infarction and coronary artery disease increases, but treatment of severe inpatient HTN in the absence of acute target organ injury is currently not directed by guidelines. Additionally, recent studies have found that treatment of severe inpatient HTN resulted in greater BP drops, and higher rates of acute kidney injury and myocardial injury. However, these studies used different study populations (Rastogi and coworkers excluded cardiac admissions), used a wide range of blood pressure (BP) thresholds, and did not account for patient level factors (eg, pain, anxiety).

Because severe HTN that develops during hospitalization is poorly studied and management remains arbitrary, understanding the actual real-world practice of identifying and treating severe HTN is essential. To address this evidence gap and limitations of previous study, we conducted a retrospective cohort study of adults admitted to five teaching hospitals in Connecticut. Our goal was to leverage data from this large healthcare system to determine prevalence of severe inpatient HTN, to characterize how severe HTN is managed, and to understand how often antihypertensive treatment leads to excessive BP reduction (Figure 1).

Severe inpatient HTN was defined as the first documented severe BP elevation (SBP > 180 or DBP > 110 mm Hg) reported after admission to the floor and did not include BPs captured in the emergency department. To exclude falsely elevated measures, we excluded patients whose BP dropped to SBP < 180 mm Hg or DBP < 110 mm Hg within 1 hour of the index severe BP elevation without administration of antihypertensive medications over the same interval. If no repeat BP measurement was available within 1 hour of severe BP elevation, we considered the patient to have severe HTN.

Covariates

Demographics, vital signs, body mass index, comorbidities prior to admission (defined per the Elixhauser comorbidity index based on ICD-10 codes9), antihypertensive medications, and laboratory results were extracted from the EHR. We defined mean arterial pressure (MAP) as \( \frac{1}{3} \text{SBP} + \frac{2}{3} \text{DBP} \). We included coefficient of variation (standard deviation of BP/mean BP of BP measurements obtained before severe HTN developed) of MAP, SBP, and DBP to account for BP variability. We also included narcotics, sedatives, benzodiazepines, non-steroidal anti-inflammatory drugs (NSAID), corticosteroids, and crystalloid IV fluids; these are markers of pain, anxiety, inflammation, or hypovolemia and have been associated with BP effects. Race and ethnicity were extracted from the patient-reported demographic information in the EHR and were included as they have been independently associated with HTN.

Antihypertensive treatment was defined as receiving any oral or IV medication class (angiotensin converting enzyme inhibitors/angiotensin receptor blockers, calcium channel blockers, beta blockers, diuretics, renin inhibitors and vasodilators) within 6 hours of developing severe HTN. Antihypertensive treatment and route were assigned as time-varying covariates within the longitudinal dataset.

Outcome

The primary study outcome was time to MAP drop ≥30% within 6 hours from the time of developing severe HTN. For most hypertensive emergencies (patients admitted for severe HTN), guidelines recommend that MAP be decreased gradually by 10–20% in the first hour and a further 5–15% over the next 23 hours to conserve cerebral perfusion and avoid ischemic damage to the vascular beds that have been habituated to elevated BPs. Additionally, severe BP reductions have been associated with increased risk of death. Therefore, we used a MAP cutoff of 30% to reflect a clinically relevant BP drop that
Severe Inpatient Hypertension and Blood Pressure Response to Treatment

2.6 Statistical analysis

Characteristics between patient groups were compared using the $\chi^2$ test for proportions and Wilcoxon rank sum test for continuous variables. Class and number of antihypertensive medications were described. We used Cox proportional hazards models with time-varying covariates to study the association between antihypertensive treatment and time to MAP drop $\geq 30\%$. Secondary outcomes included time to SBP drop $\geq 30\%$ and DBP drop $\geq 30\%$. All time-to-event comes were administratively censored at 6 hours. We fit an unadjusted model, reduced adjusted model (with covariates previously shown to be confounders2,10–14,19 and having a $p$ value $\leq 0.05$ in our data) and a fully adjusted model (all covariates). We performed similar analysis using the following exposures: (1) treatment with IV antihypertensives versus untreated, (2) treatment with oral antihypertensives versus untreated, and (3) treatment with IV versus oral antihypertensives. For secondary outcomes, we used a linear mixed effect model with random intercept and random slope at the patient level to study the association between treatment and slope of MAP, SBP and DBP change over 6 hours from the time of severe HTN development in unadjusted and adjusted models.

2.7 Sensitivity analysis

We conducted several sensitivity analyses. First, severe inpatient HTN was defined as having two consecutive severe BP measurements (SBP $> 180$ mm Hg or DBP $> 110$ mm Hg) within 3 hours [sustained BP elevation]. If no repeat BP measurement was available within 3 hours, patient was not considered to have severe inpatient HTN. Second, knowing that antihypertensive medications are required in patients who are admitted with CVD diagnosis such as acute coronary symptoms (ACS) or stroke,20–22 and patients with heart failure and atrial fibrillation may be treated with antihypertensive medications such as loop diuretics and beta blockers23–25; we excluded loop diuretics and betablockers from antihypertensive medication list and subsequently stratified by whether patients were admitted for CVD diagnosis (ACS, stroke, heart failure, or atrial fibrillation) or not. Third, treatment was
304,695 adult (≥18 years) inpatient encounters between 1/6/2016 and 3/31/2020 at 5 YNHHS hospitals

224,265 unique inpatients

23,147 with severe inpatient hypertension (SBP>180 OR DBP >110)

9,166 treated with antihypertensive medication within 6 hours of developing severe inpatient hypertension

13,753 not treated with antihypertensive medications within 6 hours of developing severe inpatient hypertension

Excluded:
- 3,078 admitted for hypertensive emergency
- 22,737 admitted to the ICU or maternity ward or research unit
- 1,083 had no BP measured
- 44,766 patients whose length of stay <2 days and >30 days
- 8,766 patients who had severe hypertension on admission or in emergency department

Excluded:
- 228 patients receiving vasopressors 0-6 hours before developing severe inpatient hypertension

FIGURE 2  Study flow diagram. YNHHS: Yale New Haven Health System; ICU: intensive care unit; BP: blood pressure

defined as receiving a new antihypertensive medication (ie, not a standing medication: medication not prescribed/day ≥90% of their hospital stay). Fourth, we excluded patients admitted to the surgical wards and thus excluded BPs obtained preoperatively and postoperatively. Fifth, we limited our analysis to patients admitted to the medical ward with sustained hypertension and defined treatment as receiving a new medication. Finally, we manually reviewed 100 charts to validate BP measurements, medications and other covariates recorded in our dataset.

Statistical significance was defined by a 2-sided $P < .05$. We conducted our analyses using R, version 4.0.0 (R Project for Statistical Computing).

3  RESULTS

3.1  Cohort characteristics

Of the 304,695 adult patient encounters within YNHHS, we identified 224,265 unique patient encounters of which 23,147 developed severe HTN (Figure 3). The median number of BP measurements available was 33 [interquartile range (IQR): 22, 57] during a median length of stay of 4.7 [3.1, 7.4] days. Compared to patients who did not develop severe HTN, those who did were older and more likely to be female, black and be admitted to a medical ward (Table 1). Additionally, those with severe HTN had higher prevalence of comorbidities (Elixhauser score: 6[3, 9] vs. 5[2,9]). Baseline laboratory values were similar though patients with severe HTN had lower estimated glomerular filtration rate (eGFR) [63 vs. 80 ml/min/1.73m²] [26, 27]. Admission BPs were higher among patients who developed severe HTN compared to those who did not (MAP: 105 [93, 117] vs. 95 [84, 105]).

After excluding 228 patients who received vasopressors, 9166 received antihypertensive medications while 13,753 were left untreated within 6 hours following severe BP elevation. Median time from admission to first recorded severe BP elevation was 8 [1.3, 49.4] hours overall, 10 [2.2, 53.1] hours among those treated and 7 [1.0, 47.6] among those untreated. Patients who developed severe HTN and received treatment were older, had similar BPs on admission, and had more comorbidities compared to those who were not treated (Table S1). Additionally, treated compared to untreated severe hypertensive patients had similar MAP at time of diagnosis (122 [115, 129]; $p$-value = .04) and were less likely to have received steroids, NSAIDs, crystalloids, or narcotics before severe BP elevation. Moreover, MAP and SBP drop after 6 hours of developing severe BP elevation was higher in untreated than treated inpatients (Figure 3, Table S2). Moreover, treated patients received overall a median of 1 [1, 1.75] antihypertensive; and a median of 0 [0, 1] of new antihypertensives. The most commonly used agents were beta blockers, calcium channel blockers, and angiotensin converting enzyme inhibitors/angiotensin receptor blockers (Table S3). Of the 9166 inpatients who developed severe HTN and were treated within 6 hours, 1912 received IV medications, 5756 received oral medications; and 1498 received both.
3.2 | Antihypertensive treatment and severe BP drop (≥30%) 

Incident MAP drop ≥30% among treated and untreated patients with severe HTN was 2.22/1000 person-hours versus 5.73/1000 person-hours (p-value < .001) (Table S2). Among inpatients who developed severe HTN, treatment was associated with lower rates of MAP and DBP drop ≥30% (Table 2) in the fully adjusted model. This is consistent for patients treated with oral medications versus untreated. However, patients who were treated with IV only medications had a 38% (HR, 95%CI: 1.4 [1.2, 1.7]), 43% (1.4 [1.2, 1.7]), and 32% (1.3 [1.1, 1.6]) greater rate of MAP, SBP and DBP drop ≥30% compared to untreated inpatients, respectively, after adjusting for demographic and clinical characteristics. Severe BP drop (≥30%) was also greater among patients treated with IV versus oral medications. The following patient characteristics were associated with greater risk of MAP drop ≥30%: increase in age, congestive heart failure, cardiac arrhythmia, peripheral vascular disease, and receiving crystalloids or sedatives.

In sensitivity analysis, in which severe HTN was defined as having sustained BP elevation, we similarly observed that patients who were treated with IV antihypertensives had greater rate of MAP, SBP, and DBP drop ≥30% compared to other groups (Table S4). We then defined treatment as receiving any class of antihypertensive medications except loop diuretics and beta blockers and subsequently stratified our analysis by CVD admission diagnosis (Table S5). In CVD and non-CVD admissions, treatment was associated with a lower rate of DBP drop ≥30% in the fully adjusted models. Treatment with IV medications was associated with greater severe BP drops when compared to other groups. The following sensitivity analyses yielded similar results to the primary analysis: defining treatment as having received a new antihypertensive medication (Table S6), excluding patients admitted to the surgical ward (Table S7), and when considering only patients admitted to the medical ward with sustained hypertension treated with new antihypertensives and adjusting for CVD admission (Table S8).

3.3 | Antihypertensive treatment and BP response 

Patients who develop severe HTN and were treated had greater absolute decrease in MAP (-0.6 [−1.0, −0.2]) and DBP (-1.2 [−1.6, −0.8]) compared to untreated inpatients in the fully adjusted model (Table 3). Similarly, patients who received oral antihypertensives compared to no
TABLE 1  Baseline characteristics of study cohort on admission overall and among those who did and did not develop severe inpatient hypertension

| Demographics                  | Overall N = 224 265 | Among inpatients who developed severe hypertension N = 23 147 (10.3%) | Among inpatients who did not develop severe hypertension N = 201 118 (89.7%) |
|-------------------------------|---------------------|---------------------------------------------------------------------|------------------------------------------------------------------|
| Age, years                    | 64.7 (18.4)         | 71.4 (16.3)                                                         | 63.9 (18.5)                                                      |
| Male                          | 107 130 (47.8)      | 10 316 (44.6)                                                      | 96 814 (48.1)                                                   |
| Black                         | 37 441 (16.7)       | 4576 (19.8)                                                        | 32 865 (16.3)                                                   |
| Hispanic or Latino            | 25 839 (11.5)       | 2341 (10.1)                                                        | 23 498 (11.7)                                                   |
| Service admitted to           |                     |                                                                    |                                                                |
| Medical                       | 178 917 (79.8)      | 19 021 (82.2)                                                      | 159 896 (79.5)                                                 |
| Surgical                      | 45 348 (20.2)       | 4126 (17.8)                                                        | 41 222 (20.4)                                                  |
| Comorbidities                 |                     |                                                                    |                                                                |
| Congestive heart failure      | 59 559 (26.6)       | 7163 (30.9)                                                        | 52 396 (26.1)                                                  |
| Cardiac arrhythmia            | 87 137 (38.9)       | 9546 (41.2)                                                        | 77 591 (38.6)                                                  |
| Valvular disease              | 46 083 (20.5)       | 4976 (21.5)                                                        | 41 107 (20.4)                                                  |
| Pulmonary circulation disorder| 28 282 (12.6)       | 2835 (12.2)                                                        | 25 447 (12.7)                                                  |
| Peripheral vascular disease   | 49 986 (22.3)       | 6036 (26.1)                                                        | 43 950 (21.9)                                                  |
| Hypertension                  | 146 976 (65.5)      | 18 493 (79.9)                                                      | 128 483 (63.9)                                                 |
| Paralysis                     | 8938 (4.0)          | 1122 (4.8)                                                         | 7816 (3.9)                                                      |
| Other neurological disorder   | 46 929 (20.9)       | 6440 (27.8)                                                        | 40 489 (20.1)                                                  |
| Chronic pulmonary disorders   | 82 129 (36.6)       | 8581 (37.1)                                                        | 73 548 (36.6)                                                  |
| Diabetes                      | 73 770 (32.9)       | 9791 (42.3)                                                        | 63 979 (31.8)                                                  |
| Hypothyroidism                | 43 942 (19.6)       | 5222 (22.6)                                                        | 38 720 (19.3)                                                  |
| Renal failure                 | 53 097 (23.7)       | 7578 (32.7)                                                        | 45 519 (22.6)                                                  |
| Liver disease                 | 35 717 (15.9)       | 3390 (14.6)                                                        | 32 327 (16.1)                                                  |
| Peptic ulcer disease (no bleeding) | 10 411 (4.6) | 1263 (5.5)                                                         | 9148 (4.5)                                                      |
| AIDS/HIV                      | 3290 (1.5)          | 346 (1.5)                                                          | 2994 (1.5)                                                      |
| Malignancy                    | 51 275 (22.9)       | 4600 (19.9)                                                         | 46675 (23.2)                                                   |
| Rheumatoid arthritis/collagen disorders | 17 565 (7.8) | 1894 (8.2)                                                         | 15 671 (7.8)                                                   |
| Coagulopathy                  | 34 507 (15.4)       | 3133 (13.5)                                                        | 31 374 (15.6)                                                  |
| Obesity                       | 57 692 (25.7)       | 5870 (25.4)                                                        | 51 822 (25.8)                                                  |
| Weight loss                   | 40 080 (17.9)       | 4250 (18.4)                                                        | 35 830 (17.8)                                                  |
| Fluid and electrolyte disorders | 103 802 (46.3) | 12 244 (52.9)                                                      | 91 558 (45.5)                                                  |
| Blood loss anemia             | 15 447 (6.9)        | 1748 (7.6)                                                         | 13 699 (6.8)                                                   |
| Iron deficiency anemia        | 42 258 (18.8)       | 4965 (21.4)                                                        | 37 293 (18.5)                                                  |
| Alcohol use disorder          | 28 829 (12.9)       | 3034 (13.1)                                                        | 25 795 (12.8)                                                  |
| Drug abuse                    | 30 101 (13.4)       | 3245 (14.0)                                                        | 26 856 (13.4)                                                  |
| Psychosis                     | 11 526 (5.1)        | 1413 (6.1)                                                         | 10 113 (5.0)                                                   |
| Depression                    | 68 409 (30.5)       | 7829 (33.8)                                                        | 60 580 (30.1)                                                  |
| Elixhauser score              | 5 [2, 9]            | 6 [3, 9]                                                           | 5 [2, 9]                                                       |

Admission characteristics, median [IQR]

| MAP                         | 95.7 [85.0, 106.3] | 105.3 [93.0, 117.0] | 94.7 [84.3, 105.0] |
| SBP (mm Hg)                 | 134.0 [118.0, 150.0] | 153.0 [134.0, 172.0] | 132.0 [117.0, 147.0] |
| DBP (mm Hg)                 | 76.0 [66.0, 86.0] | 80.0 [69.0, 92.0] | 76.0 [66.0, 85.0] |
| Heart Rate (bpm)            | 85.0 [73.0, 100.0] | 82.0 [70.0, 97.00] | 85.0 [73.0, 100.0] |
| BMI (kg/m²)                 | 27.6 [23.5, 32.9] | 27.4 [23.3, 32.5] | 27.6 [23.6, 32.9] |

(Continues)
treatment had greater reduction in MAP and DBP after adjusting for demographic and clinical characteristics. Being older, obese, and having valvular disease were associated with lower MAP drop over time.

In sensitivity analysis, we defined severe HTN as having sustained BP elevation, treatment with IV antihypertensives compared to oral antihypertensives or no treatment was associated with greater BP reduction (Table S9). After defining treatment as receiving any antihypertensive medication class except loop diuretics and beta blockers, we observed that only among non-CVD admission treatment was associated with greater MAP reduction compared to no treatment (Table S10). However, we found that neither treatment administration or route was associated with MAP or SBP changes when defining treatment as receiving new antihypertensive medications (Table S11). Following exclusion of patients admitted to the surgical ward, we found that treatment overall and with IV medications compared to no treatment was associated with lower BP (Table S12). After implementing all exclusion criteria, treatment irrespective of type resulted in greater DBP reduction (Table S13).

### 4 | DISCUSSION

In this multi-hospital retrospective cohort study, we found that among adults admitted for reasons other than HTN, severe HTN developed in 10% of which 40% received antihypertensive treatment, primarily oral medications. We found that MAP drop ≥30% within 6 hours after severe inpatient HTN development was observed in both untreated and treated patients. After adjusting for demographic and clinical characteristics, untreated patients had greater rates of MAP drop ≥30% compared to treated patients. This association, however, differed by treatment route. Those treated only with oral antihypertensives within 6 hours of developing severe HTN had lower rates of MAP drop ≥30% compared to untreated patients. In contrast, those treated with IV antihypertensives compared to untreated inpatients had greater rates of MAP drop ≥30%. Absolute reduction in MAP following severe HTN development was slightly greater by 0.6 mm Hg in patients who received treatment (any or oral antihypertensives) compared to untreated.

We found that treatment with oral antihypertensives might be safer than no treatment as it resulted in a lower rate of MAP drop ≥30%. This finding persisted even after including only new medication orders. We hypothesize that a possible mechanism in which oral antihypertensives might cause a lower BP drop compared to no treatment is via blunting of rapid BP response. The most common oral antihypertensives given in this group include metoprolol, amlodipine, and hydralazine. Metoprolol and amlodipine increased baroreflex sensitivity in small scale studies. With improved baroreceptor sensitivity, it is possible that systemic arterial pressure elevation leads to decreased discharge of sympathetic neurons thus resulting in reduced heart rate, decreased cardiac contractility, decreased peripheral vascular resistance, a lower drop in BP, and overall less variation in hemodynamics.

### Table 1 (Continued)

| Admission laboratory values median [IQR] | Overall N = 224 265 | Among inpatients who developed severe hypertension N = 23 147 (10.3%) | Among inpatients who did not develop severe hypertension N = 201 118 (89.7%) |
|----------------------------------------|----------------------|--------------------------------------------------------------------------|--------------------------------------------------------------------------|
| Serum sodium (meq/L)                   | 139.0 [136.0, 141.0]  | 139.0 [136.0, 141.0]                                                    | 139.0 [136.0, 141.0]                                                    |
| Serum potassium (meq/L)                | 4.1 [3.8, 4.05]       | 4.2 [3.8, 4.6]                                                          | 4.1 [3.8, 4.5]                                                          |
| Serum chloride (meq/L)                 | 102.0 [98.0, 105.0]   | 102.0 [98.0, 105.0]                                                     | 102.0 [98.0, 105.0]                                                     |
| Serum bicarbonate (meq/L)              | 24.0 [22.0, 27.0]     | 24.7 [22.0, 27.0]                                                       | 24.0 [22.0, 27.0]                                                       |
| BUN (mg/dl)                            | 18.0 [12.0, 27.0]     | 21.0 [14.0, 32.0]                                                       | 17.0 [12.0, 26.0]                                                       |
| Serum creatinine (mg/dl)               | 0.9 [0.7, 1.3]        | 1.1 [0.8, 1.6]                                                          | 0.9 [0.7, 1.3]                                                          |
| eGFR (ml/min/1.73m²)                   | 78.5 [49.7, 108.7]    | 63.3 [38.3, 91.7]                                                       | 80.3 [51.4, 110.4]                                                      |
| White blood cell count (x1000/ul)      | 9.1 [6.8, 12.3]       | 9.0 [6.8, 12.4]                                                         | 9.2 [6.8, 12.4]                                                         |
| Platelet count (x1000/ul)              | 223.0 [171.0, 285.0]  | 224.0 [174.0, 284.0]                                                    | 223.0 [171.0, 286.0]                                                    |
| Hemoglobin, g/dl                       | 12.0 [10.4, 13.5]     | 12.0 [10.4, 13.5]                                                       | 12.0 [10.4, 13.5]                                                       |
| Hematocrit, %                          | 36.9 [32.3, 41.0]     | 37.0 [32.5, 41.2]                                                       | 36.9 [32.2, 41.0]                                                       |

Values are presented as count (percent) or median (IQR). Abbreviations: BMI, body mass index; BP, blood pressure; MAP, mean arterial pressure; SBP, systolic BP; DBP, diastolic BP; bpm, beats per minute; BUN, Blood Urea Nitrogen; eGFR, Estimated glomerular filtration rate.
p-values are < .0001 for all covariates except chronic pulmonary disorders (p-value = .135), obesity (p = .182), iron deficiency anemia (p-value = .229), AIDS/HIV (p-value = .732), alcohol use disorder (p-value = .229), drug abuse (.005), and platelet count (p-value = .02).
by a plateau thus the acute effect on BP drop might be transient. Our findings might also reflect BP variability during hospitalization; this is influenced by several factors not readily captured in the EHR such as agitation, pain and movement. Additional research to understand the physiological effect of antihypertensives on BP in the inpatient setting are needed.

A recent study of 10 hospitals within the Cleveland Clinic healthcare system assessed whether treatment of inpatient HTN (SBP ≥140 mm Hg) affects outcomes among patients hospitalized for non-CVD reasons. They found that 78% of inpatients had an elevated SBP reading and 33% were treated (IV antihypertensive or a new class of oral antihypertensives). Treatment was associated with greater odds of composite outcome (acute kidney injury [AKI], myocardial injury, stroke) irrespective of treatment route (IV or oral). They observed that among patients with SBP > 160 mm Hg treated and untreated patients had similar rates of SBP decline > 20 mm Hg (58% and 61%, respectively). Similarly, we observed a SBP decrease of -32 [−49, −18] and -25 [−53, −21] mm Hg among treated and untreated inpatients in our cohort. In the Cleveland Clinic cohort 47% of patients with HTN were treated, compared to 40% of our cohort. There are key differences between our studies. First, our definition for severe HTN relied on higher BP values (SBP > 180 or DBP > 110 mm Hg). In the Cleveland Clinic cohort only 7.7% of untreated and 17.7% of treated patients had a SBP >180 (n = 2,139 vs. 23,147 inpatients in our cohort). Second, we included CVD-related admissions. Third, we defined treatment as receiving any antihypertensive medication within 6 hours of severe HTN development. The main finding from their study was that treatment of elevated BP in the hospital was not beneficial and potentially harmful. Our analyses focused on MAP drop of ≥30% and not on end-organ damage; however, a severe reduction in BP is associated with increased risk of death. Unlike Rastogi and coworkers we observed that treatment with IV only medications infers additional harm (MAP drop ≥30%) compared to untreated individuals or those treated with oral antihypertensives. Of note, in both cohorts a BP drop was observed irrespective of treatment. This suggests that a one-size-fits-all approach to treatment of severe inpatient HTN is not necessary.

### Table 2: Association of treatment with MAP, SBP, DBP drop ≥30% over 0–6 hours from time of severe inpatient HTN development

| HR (95% CI) | Model 1 (Unadjusted) | Model 2 | Model 3 |
|------------|----------------------|---------|---------|
| **MAP drop ≥ 30%** | | | |
| Treated versus untreated | 0.86 [0.80, 0.94] | 0.89 [0.82, 0.96] | 0.89 [0.80, 0.99] |
| Treated with IV only versus untreated | 1.49 [1.33, 1.68] | 1.49 [1.32, 1.68] | 1.38 [1.15, 1.67] |
| Treated with oral only versus untreated | 0.59 [0.53, 0.66] | 0.61 [0.54, 0.68] | 0.69 [0.61, 0.79] |
| Treated with IV only versus oral only | 2.57 [2.22, 2.98] | 2.48 [2.13, 2.91] | 2.06 [1.65, 2.57] |
| **SBP drop ≥ 30%** | | | |
| Treated versus untreated | 0.92 [0.86, 0.99] | 0.93 [0.86, 1.00] | 0.96 [0.87, 1.05] |
| Treated with IV only versus UNTREATED | 1.44 [1.29, 1.62] | 1.42 [1.26, 1.59] | 1.43 [1.19, 1.71] |
| Treated with oral only versus untreated | 0.69 [0.62, 0.76] | 0.69 [0.63, 0.77] | 0.78 [0.69, 0.88] |
| Treated with IV only versus oral only | 2.11 [1.84, 2.42] | 2.08 [1.80, 2.41] | 1.87 [1.53, 2.29] |
| **DBP drop ≥ 30%** | | | |
| Treated versus untreated | 0.79 [0.74, 0.85] | 0.82 [0.76, 0.89] | 0.79 [0.72, 0.88] |
| Treated with IV only versus untreated | 1.34 [1.20, 1.50] | 1.37 [1.22, 1.54] | 1.32 [1.11, 1.58] |
| Treated with oral only versus untreated | 0.52 [0.47, 0.58] | 0.55 [0.49, 0.61] | 0.60 [0.53, 0.69] |
| Treated with IV only versus oral only | 2.66 [2.30, 3.08] | 2.59 [2.22, 3.02] | 2.25 [1.82, 2.78] |

Of the 9,166 inpatients who developed severe HTN and were treated, 1,912 were treated only with IV medications, 5,756 were treated only with oral medications and 1,498 were treated with a combination of IV and oral medications. 13,753 inpatients developed severe HTN and were not treated. **Model 1**: unadjusted; **Model 2**: age, sex, race, ethnicity, ward, comorbidities (congestive heart failure, cardiac arrhythmia, peripheral vascular disease, hypertension, diabetes, hypothyroidism, renal failure, AIDS/HIV, cancer, alcohol use, drug use, psychosis, depression), baseline laboratory values (sodium, potassium, chloride, bicarbonate, BUN, eGFR, WBCC, platelet count, hemoglobin, hematocrit), NSAID use 0–6 hours before time of severe inpatient HTN, crystalloid use 0–6 hours before time of severe inpatient HTN, steroid use 0–6 hours before time of severe inpatient HTN, sedative use 0–6 hours before time of severe inpatient HTN, hospital. **Model 3**: age, sex, race, ethnicity, ward, comorbidities (congestive heart failure, cardiac arrhythmia, valvular disease, pulmonary circulation disorder, peripheral vascular disease, hypertension, paralysis, other neurological disorders, chronic pulmonary disease, diabetes, hypothyroidism, renal failure, liver disease, peptic ulcer disease excluding bleeding, AIDS/HIV, lymphoma, cancer, rheumatoid arthritis/collagen disorder, coagulopathy, osteomyelitis, weight loss, fluid and electrolyte disorders, blood loss anemia, deficiency anemia, alcohol use, drug use, psychosis, depression), baseline laboratory values (sodium, potassium, chloride, bicarbonate, BUN, eGFR, WBCC, platelet count, hemoglobin, hematocrit), NSAID use 0–6 hours before time of severe inpatient HTN, crystalloid use 0–6 hours before time of severe inpatient HTN, steroid use 0–6 hours before time of severe inpatient HTN, narcotic use 0–6 hours before time of severe inpatient HTN, sedative use 0–6 hours before time of severe inpatient HTN, maximum MAP before time of severe inpatient HTN development, minimum MAP before time of severe inpatient HTN development, coefficient of variation of MAP before time of severe inpatient HTN development, hospital.

Abbreviations: HR, hazard ratio; CI, confidence interval; MAP, mean arterial pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure, HTN, hypertension; IV, intravenous.
appropriate, rather certain patients may especially benefit or be harmed from antihypertensives. Additionally, before initiating treatment we should have further knowledge on how BP is measured in the hospital setting. In our study as well as others looking at inpatient HTN, steroid use 0–6 hours before time of severe inpatient HTN, narcotic use 0–6 hours before time of severe inpatient HTN, sedative use 0–6 hours before time of severe inpatient HTN, hospital.

**Model 3:** age, sex, race, ethnicity, ward, comorbidities (congestive heart failure, cardiac arrhythmia, peripheral vascular disease, hypertension, diabetes, hypothyroidism, renal failure, AIDS/HIV, cancer, alcohol abuse, drug abuse, psychosis, depression), baseline laboratory values (sodium, potassium, chloride, bicarbonate, BUN, eGFR, WBCC, platelet count, hemoglobin, hematocrit), NSAID use 0–6 hours before time of severe inpatient HTN, crystalloid use 0–6 hours before time of severe inpatient HTN, steroid use 0–6 hours before time of severe inpatient HTN, narcotic use 0–6 hours before time of severe inpatient HTN, sedative use 0–6 hours before time of severe inpatient HTN, hospital.

**Abbreviations:** CI, confidence interval; MAP, mean arterial pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure.

---

**TABLE 3** Association of antihypertensive treatment among those who developed severe inpatient hypertension with MAP, SBP, and DBP change within 6 hours of developing severe inpatient hypertension

| β (95% CI) | Model 1 (Unadjusted) | Model 2 | Model 3 |
|-----------|----------------------|---------|---------|
| **Slope of MAP** | | | |
| Treated versus Untreated | −0.78 [−1.13, −0.43] | −0.61 [−0.98, −0.25] | −0.56 [−0.97, −0.16] |
| Treated with IV only versus untreated | −0.93 [−1.73, −0.13] | −0.57 [−1.39, 0.25] | 0.42 [1.49, 0.65] |
| Treated with oral only versus untreated | −1.02 [−1.47, −0.58] | −0.78 [−1.24, −0.32] | −0.56 [−1.04, −0.08] |
| Treated with IV only versus oral only | 0.51 [−0.31, 1.33] | 0.45 [−0.39, 1.29] | 0.23 [−0.03, 0.86] |
| **Slope of SBP** | | | |
| Treated versus Untreated | 0.07 [−0.44, 0.58] | −1.07 [−0.63, 0.42] | 0.17 [−0.41, 0.76] |
| Treated with IV only versus untreated | −1.03 [−2.19, 0.13] | −1.16 [−2.35, 0.03] | −1.09 [−2.63, 0.44] |
| Treated with oral only versus untreated | 0.33 [−0.32, 0.99] | 0.26 [0.41, 0.94] | 0.66 [0.39, 1.37] |
| Treated with IV only versus oral only | −1.48 [−2.71, −0.25] | −1.51 [−2.77, −0.24] | −2.14 [−3.68, −0.62] |
| **Slope of DBP** | | | |
| Treated versus Untreated | −1.34 [−1.68, −0.99] | −1.03 [−1.38, −0.68] | −1.23 [−1.63, −0.83] |
| Treated with IV only versus untreated | −1.18 [−1.96, −0.40] | −0.58 [−1.37, 0.22] | −0.58 [−1.64, 0.47] |
| Treated with oral only versus untreated | −1.85 [−2.29, −1.41] | −1.46 [−1.91, −1.01] | −1.46 [−1.93, −0.98] |
| Treated with IV only versus oral only | 1.39 [0.61, 2.18] | 1.41 [0.60, 2.21] | 1.40 [0.43, 2.39] |

**Model 1:** unadjusted.

**Model 2:** age, sex, race, ethnicity, ward, comorbidities (congestive heart failure, cardiac arrhythmia, peripheral vascular disease, hypertension, diabetes, hypothyroidism, renal failure, AIDS/HIV, cancer, alcohol abuse, drug abuse, psychosis, depression), baseline laboratory values (sodium, potassium, chloride, bicarbonate, BUN, eGFR, WBCC, platelet count, hemoglobin, hematocrit), NSAID use 0–6 hours before time of severe inpatient HTN, crystalloid use 0–6 hours before time of severe inpatient HTN, steroid use 0–6 hours before time of severe inpatient HTN, narcotic use 0–6 hours before time of severe inpatient HTN, sedative use 0–6 hours before time of severe inpatient HTN, hospital.

**Model 3:** age, sex, race, ethnicity, ward, comorbidities (congestive heart failure, cardiac arrhythmia, valvular disease, pulmonary circulation disorder, peripheral vascular disease, hypertension, paralysis, other neurological disorders, chronic pulmonary disease, diabetes, hypothyroidism, renal failure, liver disease, peptic ulcer disease excluding bleeding, AIDS/HIV, lymphoma, cancer, rheumatoid arthritis/collagen disorder, coagulopathy, obesity, weight loss, fluid and electrolyte disorders, blood loss anemia, deficiency anemia, alcohol abuse, drug abuse, psychosis, depression), baseline laboratory values (sodium, potassium, chloride, bicarbonate, BUN, eGFR, WBCC, platelet count, hemoglobin, hematocrit), NSAID use 0–6 hours before time of severe inpatient HTN, crystalloid use 0–6 hours before time of severe inpatient HTN, steroid use 0–6 hours before time of severe inpatient HTN, narcotic use 0–6 hours before time of severe inpatient HTN, sedative use 0–6 hours before time of severe inpatient HTN, maximum MAP before time of severe inpatient HTN development, minimum MAP before time of severe inpatient HTN development, coefficient of variation of MAP before time of severe inpatient HTN development, hospital.

**Abbreviations:** CI, confidence interval; MAP, mean arterial pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure.
higher 1-year risk of CVD events among patients who had their antihypertensive treatment intensified during inpatient admissions. Additional research is needed to better understand if and when treatment is needed.

Our study has several strengths. To our knowledge, this is the largest cohort of inpatients with severe HTN. We used a set BP cutoff of 180/110 to define severe BP elevation. Participants are well-characterized clinically and encompassed patients admitted for various reasons, allowing for adjustments of many possible confounders (such as comorbidities, medications that affect BP). Results were consistent across multiple sensitivity analysis. We validated our data by manual chart review. There were also limitations: (1) We did not account for outpatient BP before hospitalization. However, we adjusted for history of HTN and for the range of BP fluctuation during the admission before development of severe inpatient HTN; (2) We do not have data on how BP was measured (eg, device, cuff placement). Data represents real word practice; (3) Data is from a single healthcare system and findings might not be generalizable across other healthcare systems; (4) We assessed the effect of antihypertensive treatment by route irrespective of treatment indication, dose, and whether antihypertensive was a home medication. In sensitivity analysis we excluded loop diuretics and beta blockers from antihypertensive medication list as they can be given for other CVD indications and stratified by CVD admission status. Additionally, to account for outpatient antihypertensives, we repeated our analyses in which we excluded standing medications that most likely reflect patients home medications; (5) BP management differs by medical or surgical service patient is admitted to. We excluded patients admitted to the surgical ward in sensitivity analysis; and (6) Other unmeasured covariates might confound the association between treatment of severe HTN and outcomes. We are unable to capture the clinical decision-making reasons for treatment or no treatment (ie, selection bias), and who ordered the antihypertensives. However, we have accounted for all available confounders in the EHR for our analysis. Thus, the greater MAP drop among the untreated may reflect clinical intuition on the part of the providers, that is, providers correctly anticipate BP improvement, that we can’t fully capture.

5 | CONCLUSIONS

We found that in a cohort of hospitalized patients admitted for reasons other than HTN, 10% of adults developed severe HTN and 40% of these patients were treated with antihypertensives. Paradoxically, treatment (overall and with oral antihypertensives) compared to no treatment resulted in lower rates of MAP drop ≥30%. Patients with severe HTN treated with only IV antihypertensives compared to untreated and treated with oral only antihypertensives had greater rates of MAP drop ≥30%. Our findings suggest that treating severe inpatient HTN with IV antihypertensives should be done conservatively. Upcoming studies will aim at assessing BP reduction following specific antihypertensive drug classes and types and assess the effect of treatment on clinical outcomes, such as stroke, myocardial infarction and AKI, specifically among those with a significant MAP drop. Given that both untreated and treated patients with severe HTN had a significant reduction in MAP, a one-size-fits-all approach is not appropriate to treating severe HTN. Additionally, conducting prospective studies using standardized BP measurement to assess frequency of severe HTN as well as the role of treatment on outcomes are critical. Finally, further research is needed to phenotype hospitalized patients with severe HTN based on adverse outcome risk to help establish personalized treatment guidelines.

ACKNOWLEDGMENTS

We would like to thank William B. White, MD at the University of Connecticut School of Medicine for his input on the manuscript. This work was supported by National Institutes of Health grants P30DK079310 (FPW) and R01DK113191 (FPW). Dr. Ghazi was supported by the American Heart Association Postdoctoral fellowship award (829804) and Robert E. Leet and Clara Guthrie Patterson Trust Mentored Research Award. The funders did not play a role in study design, data collection, analysis, reporting, or the decision to submit for publication.

AUTHOR CONTRIBUTION

Lama Ghazi: Design, Analysis, Writing. Fan Li: Supervision, Analysis, Writing. Xinyuan Chen: Analysis, Writing. Michael Simonov: Data acquisition, Writing. Yu Yamamoto: Data acquisition, Writing. Aditya Biswas: Data acquisition, Writing. Jonathan Hanna: Validation, Writing. Tayyab Shah: Validation, Writing. Raymond Townsend: Writing. Aldo Peixoto: Supervision, Writing. F. Perry Wilson: Supervision, Writing.

ORCID

Lama Ghazi MD, PhD https://orcid.org/0000-0002-9930-3575
Fan Li PhD https://orcid.org/0000-0001-6183-1893

REFERENCES

1. Axon RN, Cousineau L, Egan BM. Prevalence and management of hypertension in the inpatient setting: a systematic review. J Hosp Med. 2011;6(7):417–422.
2. Peixoto AJ. Acute severe hypertension. N Engl J Med. 2019;381(19):1843–1852.
3. Janke AT, McNaughton CD, Brody AM, Welch RD, Levy PD. Trends in the incidence of hypertensive emergencies in US emergency department from 2006 to 2013. J Am Heart Assoc. 2016;5(12):e004511.
4. Shah M, Patil S, Patel B, et al. Trends in Hospitalization for Hypertensive Emergency, and Relationship of End-Organ Damage With In-Hospital Mortality. Am J Hypertens. 2017;30(7):700–706.
5. Rastogi R, Sheehan MM, Hu B, Shaker V, Kojima L, Rothberg MB. Treatment and outcomes of inpatient hypertension among adults with non-cardiac admissions. JAMA Intern Med. 2021;181(3):345–352.
6. Axon RN, Garrell R, Pfahl K, et al. Attitudes and practices of resident physicians regarding hypertension in the inpatient setting. J Clin Hypertens (Greenwich). 2010;12(9):698–705.
7. Axon RN, Turner M, Buckley R. An update on inpatient hypertension management. Curr Cardiol Rep. 2015;17(11):94.
8. Mohandas R, Chamarthi G, Bozorgmehr S, et al. Pro Re nata antihypertensive medications and adverse outcomes in hospitalized patients: a propensity-matched cohort study. Hypertension. 2021;78(2):516–524.
9. Quan H, Sundararajan V, Halfon P, et al. Coding algorithms for defining comorbidities in ICD-9-CM and ICD-10 administrative data. Med Care. 2005;43(11):1130–1139.
10. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. *Nat Rev Nephrol*. 2020;16(4):223–237.

11. Booth JN, Li J, Zhang L, Chen L, Muntner P, Egan B. Trends in prehypertension and hypertension risk factors in US adults: 1999–2012. *Hypertension*. 2017;70(2):275–284.

12. Parikh NI, Pencina MJ, Wang TJ, et al. A risk score for predicting near-term incidence of hypertension: the Framingham Heart Study. *Ann Intern Med*. 2008;148(2):102–110.

13. Khirsagar AV, Chiu YL, Lombaert AS, et al. A hypertension risk score for middle-aged and older adults. *J Clin Hypertens* (Greenwich). 2010;12(10):800–808.

14. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/ABC/ACPM/AGS/APHA/ASH/ASP/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation*. 2018;138(17):e426–e483.

15. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH guidelines for the management of arterial hypertension: the task force for the management of arterial hypertension of the European Society of Cardiology and the European Society of Hypertension: the task force for the management of arterial hypertension of the European Society of Cardiology and the European Society of Hypertension. *J Hypertens*. 2018;36(10):1953–2041.

16. Elliott WJ. Clinical features in the management of selected hypertensive emergencies. *Prog Cardiovasc Dis*. 2006;48(5):316–325.

17. Mayer SA, Kurtz P, Wyman A, et al. Clinical practices, complications, and mortality in neurological patients with acute severe hypertension: the studying the treatment of acute hyperTension registry. *Crit Care Med*. 2011;39(10):2330–2336.

18. Peacock F, Amin A, Granger CB, et al. Hypertensive heart failure: patient characteristics, treatment, and outcomes. *Am J Emerg Med*. 2011;29(8):855–862.

19. Saguner AM, Dür S, Perrig M, et al. Risk factors promoting hypertensive crises: evidence from a longitudinal study. *Am J Hypertens*. 2010;23(7):775–780.

20. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2019;50(12):e344–e418.

21. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;130(25):2354–2394.

22. O’Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2013;61(4):e78–e140.

23. Yancy CW, Jessup M, Bozkurt B, et al. 2017 ACC/AHA/HFSA focused update of the 2013 ACCF/AHA guideline for the management of heart failure: a report of the american college of cardiology/american heart association task force on clinical practice guidelines and the Heart Failure Society of America. *J Card Fail*. 2017;23(8):628–651.

24. McNamara RL, Tamariz LJ, Segal JB, Bass EB. Management of atrial fibrillation: review of the evidence for the role of pharmacologic therapy, electrical cardioversion, and echocardiography. *Ann Intern Med*. 2003;139(12):1018–1033.

25. January CT, Wann LS, Calkins H, et al. 2019 AHA/ACC/HRS focused update of the 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol*. 2019;74(1):104–132.

26. Inker LA, Eneanya ND, Coresh J, et al. New creatinine- and cystatin C-based equations to estimate GFR without race. *N Engl J Med*. 2021;385(19):1737–1749.

27. Delgado C, Barreja M, Crews DC, et al. A unifying approach for GFR estimation: recommendations of the NKF-ASN task force on reassessing the inclusion of race in diagnosing kidney disease. *Am J Kidney Dis*. 2021.

28. Valsalainen RK, Kantola IM, Airaksinen KE, Tahvanainen KU, Kaila TJ. Vagal cardiac activity in essential hypertension: the effects of metoprolol and ramipril. *Am J Hypertens*. 1998;11(6 Pt 1):649–658.

29. Kirchheim HR. Systemic arterial baroreceptor reflexes. *Physiol Rev*. 1976;56(1):100–177.

30. Siché JP, Baguet JP, Fagret D, Trémel F, de Gaudemaris R, Mailion JM. Effects of amiodarone on baroreflex and sympathetic nervous system activity in mild-to-moderate hypertension. *Am J Hypertens*. 2001;14(5 Pt 1):424–428.

31. Weder AB, Erickson S. Treatment of hypertension in the inpatient setting: use of intravenous labetalol and hydralazine. *J Clin Hypertens* (Greenwich). 2010;12(1):29–33.

32. Campbell P, Baker WL, Bendel SD, White WB. Intravenous hydralazine for blood pressure management in the hospitalized patient: its use is often unjustified. *J Am Soc Hypertens*. 2011;5(6):473–477.

33. Lipari M, Moser LR, Petrovitch EA, Farber M, Flack JM. As-needed intravenous antihypertensive therapy and blood pressure control. *J Hosp Med*. 2016;11(3):193–198.

34. Jacobs ZG, Najafi N, Fang MC, et al. Reducing unnecessary treatment of asymptomatic elevated blood pressure with intravenous medications on the general internal medicine wards: a quality improvement initiative. *J Hosp Med*. 2019;14(3):144–150.

35. Pasik SD, Chiu S, Yang J, et al. Assess before Rx: reducing the overtreatment of asymptomatic blood pressure elevation in the inpatient setting. *J Hosp Med*. 2019;14(3):151–156.

36. Cherney D, Straus S. Management of patients with hypertensive urgencies and emergencies: a systematic review of the literature. *J Gen Intern Med*. 2002;17(12):937–945.

37. Perez MI, Musini VM. Pharmacological interventions for hypertensive emergencies: a Cochrane systematic review. *J Hum Hypertens*. 2008;22(9):596–607.

38. Breu AC, Axon RN. Acute treatment of hypertensive urgency. *J Hosp Med*. 2018;13(12):860–862.

39. Anderson TS, Jing B, Auerbach A, et al. Clinical outcomes after intensive antihypertensive medication regimens among older adults at hospital discharge. *JAMA Intern Med*. 2019;179(11):1528–1536.

**SUPPORTING INFORMATION**

Additional supporting information may be found in the online version of the article at the publisher’s website.

**How to cite this article:** Ghazi L, Li F, Chen X, et al. Severe inpatient hypertension prevalence and blood pressure response to antihypertensive treatment. *J Clin Hypertens*. 2022;24:339–349. [https://doi.org/10.1111/jch.14431](https://doi.org/10.1111/jch.14431)