Hypertensive Emergencies Remain a Clinical Problem and are Associated with High Mortality

Weranuj Roubsanthisuk MD*,
Unticha Wongsurin RN*, Peera Buranakitjaroen MD, PhD*

**Division of Hypertension, Department of Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand*

Objective: *We suspect that hypertensive emergencies remain a clinical problem and data on their long-term prognosis are lacking. This study was conducted in order to determine the frequency, management, and outcome of hypertensive emergencies in this era, in which hypertension treatment is more effective than in the past.*

Material and Method: *We reviewed the medical records of patients with hypertensive emergencies admitted to the medical wards of Siriraj Hospital in 2003 and collected data on their characteristic, management, investigations, and follow-ups through 31 December 2007.*

Results: *There were 184 patients included. Hypertension has been previously diagnosed in 89% of cases. Nearly half also had diabetes mellitus and around a quarter had chronic kidney failure. Mean ± SD of blood pressure at presentation was 205.96 ± 21.36/114.60 ± 20.59 mmHg. Cardiac complications and stroke accounted for 71% and 23% of all target organ damage, respectively. Intravenous nitroglycerine and furosemide were most frequently prescribed. Additional investigations to search for the causes of hypertension were performed in only 55 cases. The average hospital stay was 9.8 days. The in-hospital mortality rate was 15%. Some 26% of patients were lost to follow-up and another 20% died later. Only 19% of patients had regular follow-ups until the end of 2007 and remained on an average of 2.4 antihypertensive drugs.*

Conclusion: *Hypertensive emergencies are usually found in patients with a history of hypertension and diabetes mellitus or kidney failure. Recommended investigations usually failed to identify the cause of hypertension. The mortality rate of these patients was extremely high while their adherence to treatment was extremely poor.*

Keywords: *Hypertension, Emergencies, Diabetes mellitus, Kidney failure, Heart failure*

J Med Assoc Thai 2010; 93 (Suppl. 1): S54-61
Full text. e-Journal: *http://www.mat.or.th/journal*

Chronic hypertension (HT) is the leading cause of stroke, coronary artery disease, heart failure, and other cardiovascular complications. In 2001, 13.5% of total mortality and 6% of disability-adjusted life years worldwide were attributable to high blood pressure (BP)[1]. Therefore, health organizations around the world are trying hard to prevent the development of both HT and its complications. The availability of many effective antihypertensive medications nowadays has led to the substantial improvement of BP control in HT as compared with the past[2]. Numerous landmark studies in hypertensive subjects have proved that BP lowering is effective in preventing the development of stroke[3,4], heart failure[4], acute myocardial infarction[4], and end stage renal disease (ESRD)[5].

In contrast, cases with severe HT and acute or progressive target organ damage (TOD), which are defined as hypertensive emergencies, are much less studied. Most studies usually described only the clinical characteristics of the patients at presentation[6,7]. Data on long-term prognosis of these cases are lacking[8]. Some physicians believe that hypertensive emergencies are rare. However, we have seen these cases

Correspondence to: Roubsanthisuk W, Division of Hypertension, Department of Medicine Siriraj Hospital, Mahidol University, 2 Prannok Road, Bangkoknoi, Bangkok 10700, Thailand. Phone: 0-2419-7790-1, Fax: 0-2419-7790. E-mail: siwro@mahidol.ac.th

from time to time. We suspect that hypertensive emergencies remain a clinical problem in this era, in which effective BP lowering medications are more available than in the past.

We reviewed medical records at a tertiary care university hospital to determine the frequency, the management, and the outcome of hypertensive emergencies.

Material and Method

Design and Subjects

We reviewed medical records of hypertensive emergencies admitted to medical wards of our hospital in 2003. Hypertensive emergencies were defined as 1) severe HT [systolic BP (SBP) $\geq$ 180 or diastolic BP (DBP) $\geq$ 120 mmHg] and 2) acute or progressive TOD as defined in the guidelines for the management of HT[2,9]. Major acute TOD in hypertensive emergencies included acute left ventricular failure, acute myocardial infarction, unstable angina, acute aortic dissection, accelerated-malignant HT, stroke, hypertensive encephalopathy, progressive renal failure, severe preeclampsia, and eclampsia. The presence of these conditions was based on the definitions written in major practice guidelines[10-17]. Data on patients' characteristics, in hospital management, and further investigations to identify the cause of HT were collected. We also collected long-term follow-up data from hospital discharge until 31 December 2007. Two investigators reviewed each medical record. Disparities of data were resolved among the two reviewers. Data collected were filled in predefined case record form. The Ethical Committee of Siriraj Hospital approved this study protocol.

Statistical analysis

Data were analyzed using SPSS 10.0 (SPSS Cooperation, Chicago, Illinois, USA) and presented as percentages and means or medians with standard deviations as appropriate.

Results

There were 184 hypertensive emergencies included into the study. Of these, 160 were for patients admitted once and 24 were admitted more than one time. This made up a total of 216 admissions. There was a total of 6230 medical admissions in 2003. Therefore, hypertensive emergencies were accounted for 3.5% of all medical admissions in that year. Most of the readmissions were for the recurrent attack of unstable angina or acute left ventricular failure. Table 1 demonstrated the characteristics of all subjects. Only 10 patients with known HT had never received antihypertensive medication. Cardiac symptoms were the most common and neurological deficits were the second most common presenting symptoms. Other manifestations included edema, oliguria, headache, blurred vision, epistaxis, fall, seizure, dizziness, vertigo, loss of appetite, nausea, vomiting, abdominal discomfort, and fatigue (Table 2). The median duration of the presenting symptoms was 12.5 hours (range 10 minutes-168 hours). Forty-nine percent of subjects had significant renal impairment as defined by serum creatinine greater than 1.5 mg/dl in men or 1.4 mg/dl in women[13]. Moreover, 80% of cases in which the urinalysis was available had proteinuria ranging from trace to 4+.

Acute target organ damage and the initial management

Only one instance of TOD appeared in an individual patient in 148 admissions. Two TODs pre-

Table 1. Clinical characteristics of all 184 hypertensive emergency cases

Characteristics	
Age (years)	60.5 $\pm$ 14.9 (23-99)
Sex: female (%)	50.5
Underlying diseases	
Hypertension (%)	88.9
Duration of hypertension (years)	7.4 $\pm$ 6.0 (1 week-30 years)
Current antihypertensive drugs (items)	2.4 $\pm$ 1.1 (1-6)
History of non-compliance before admission (%)	24.2
Diabetes mellitus (%)	45.7
End-stage renal disease (%)	22.8
On regular dialysis (%)	19
Admission SBP (mmHg)	205.96 $\pm$ 21.36 (160-260)
Admission DBP (mmHg)	114.60 $\pm$ 20.59 (66-170)
BUN (mg/dl)	40.84 $\pm$ 37.80 (6-217)
Creatinine (mg/dl)	4.46 $\pm$ 6.67 (0.5-47.7)
Urinary protein detected by semiquantitative method (%)	
Negative	20.4
Trace	0.6
1+	17.3
2+	16.0
3+	22.2
4+	23.5

Data shown were mean $\pm$ SD (range) unless otherwise specified

sented in the other 67 admissions. Most were unstable angina or acute myocardial infarction with acute left ventricular failure. Three TODs presented in one admission. Therefore, total TODs in all 216 admissions were 285 cardiovascular events. Cardiac complications were the first and stroke was the second most common TODs in our study (Table 3).

Most of the cases were initially treated with intravenous antihypertensive medications. The most frequently used drugs were nitroglycerine and furosemide (Table 4). Most cases received more than one medication. Nitroglycerine and furosemide were the most common drug combinations. Sodium nitroprusside or nicardipine was usually used when nitroglycerine did not decrease BP to the target. Hydralazine was given once via bolus injection. A case with eclampsia received continuous infusion of magnesium sulphate. The median durations of nitroglycerine and sodium nitroprusside infusions were 36 hours (range 1-148.5 hours) and 20 hours (range 1.5-159 hours), respectively. Intravenous antihypertensive agent was not prescribed in 48 admissions. Thirty-eight of these cases suffered from acute strokes. BP could be controlled with oral drugs after hospitalization in the other cases. Short-acting nifedipine was prescribed in 34 admissions; via oral route in 30 admissions, via sublingual administration in 3 admissions and via both oral and sublingual administrations in one admission. Nifedipine did not induce a precipitous fall of BP in any cases. Oral antihypertensive medications were prescribed within 24, 24-48, and 48-72 hours after admission in 68.8, 11.6, and 3.3% of all admissions, respectively. However, 7% of cases received oral agents later than 72 hours after admission and 9.3% of cases did not received oral drug while they stayed in the hospital.

Acute dialysis, in addition to BP lowering, was performed in 31 admissions. Ninety percent of cases who needed acute dialysis had ESRD as an underlying condition and only 29% of these cases have been previously placed on regular hemodialysis. Ten percent of cases who received dialysis in the hospital had renal recovery after BP was better controlled.

Investigations to search for the causes of hypertension

Additional investigations were performed to search for the causes of HT in only 55 cases (Table 5). More than one test was performed in some cases. Common investigations were renal ultrasonography and investigations for renal artery stenosis (RAS). There was a variability in selecting the first investigation to

Table 2. The presenting symptoms in 216 admissions of 184 hypertensive emergency cases

Symptoms	Number (%)
Dyspnea	122 (56.5)
PND	84 (38.9)
Chest pain	61 (28.2)
Edema	29 (13.4)
Oliguria	12 (5.6)
Weakness	46 (21.3)
Deterioration of consciousness	27 (12.5)
Headache	12 (5.6)
Dysarthria	9 (4.2)
Blurred vision	5 (2.3)
Epistaxis	1 (0.5)
Others	27 (12.5)

Table 3. Details of 285 acute target organ damages found in 216 admissions of 184 hypertensive emergency cases

Target organ damage	Number (%)
Cardiac complications	
Acute left ventricular failure	131 (46)
Unstable angina	37 (13)
Acute myocardial infarction	34 (11.9)
Acute aortic dissection	2 (0.7)
Accelerated-malignant HT	13 (4.6)
Stroke or transient ischemic attack (TIA)	
Ischemic stroke	39 (13.7)
Hemorrhagic stroke	23 (8.1)
Stroke (unspecified type)	1 (0.4)
TIA	1 (0.4)
Progressive renal failure	2 (0.7)
Eclampsia	1 (0.4)
Epistaxis	1 (0.4)
Total	285 (100)

Table 4. Prescription of parenteral antihypertensive agents in 216 admissions

Intravenous blood pressure lowering agents	Number (%)
Nitroglycerine	139 (64.4)
Furosemide	139 (64.4)
Injection	111 (51.4)
Continuous drip	3 (1.4)
Injection followed by continuous drip	25 (11.6)
Sodium nitroprusside	18 (8.3)
Nicardipine	5 (2.3)
Hydralazine	1 (0.5)
$MgSO_4$	1 (0.5)

demonstrate the anatomy of renal artery. The most common one was Doppler ultrasonography. Renal ultrasonography showed multiple cysts in both kidneys compatible with adult polycystic kidney disease in one patient and small renal calculi in 3 patients. Six patients had renal biopsy with renal pathology demonstrating glomerulosclerosis with or without tubulointerstitial fibrosis in 5 cases and glomerulosclerosis with fibrinoid necrosis of blood vessels, substantiating the presence of malignant HT in one case. Two patients had slight elevation of urinary catecholamines or vanillylmandelic acid (VMA). However, I^{131}-metaiodobenzylguanidine (MIBG) scan did not show any abnormal uptake of the tracer in both cases. Aldosterone/renin ratio (ARR) was determined in 4 cases. No patient had suppressed plasma renin activity, which did not confirm the presence of primary aldosteronism. In summary, 4 cases had RAS, all of which the diagnosis was confirmed by renal angiography.

Mortality rate

The average length of hospital stay was 9.8 ± 9.2 days (range 1-62 days). Mean ± SD of BP before discharge was 136.98 ± 21.83/81.30 ± 13.02 mmHg. The in-hospital mortality rate was 15%. Sixty percent of deaths were related to stroke complicated with brain herniation. Most of the discharged cases received 2-3 items of antihypertensive drugs. Frequently prescribed drugs were diuretics, β-blockers, calcium antagonists, and angiotensin-converting enzyme inhibitors (ACEIs). Surprisingly, only 2.9% of cases received angiotensin II receptor blockers (ARBs). Unlike the usual practice in HT, the most frequently prescribed diuretics at hospital discharge were loop diuretics. This may be due to the high proportion of patients with heart failure in this study. Because of their rapid BP lowering effects[18], the prescriptions of dihydropyridine (DHP) calcium antagonists were far more often than those of non-DHP calcium antagonists.

Twenty percent of cases were referred to other hospitals for long-term care. Forty-seven cases (26%) were lost to long-term follow-up. Fifteen of them had no hospital visit after discharge. An average duration of follow-up among the other thirty-two cases was 14 ± 13 months (range 0.5-44 months). Twenty percent of cases died later. Complicated infections after prolonged hospitalization such as ventilator-associated pneumonia or catheter-associated infection were the major cause of death during this period (Table 6). The average duration from hospital discharge to death was 18 ± 17 months (range 1-55 months). Only 19% of cases still had regular visit till the end of 2007. Mean ± SD of BP at the last hospital visit was 144.20 ± 15.60/79.30 ± 9.40 mmHg. These patients had an average duration of follow-up of 52 ± 3.8 months (range 44-59 months) and remained on an average of 2.4 ± 0.9 antihypertensive drugs (range 1-4 drugs). The use of diuretics was much less than the initial period and thiazide-type diuretics were the majority instead of loop diuretics. The frequency of ARBs prescribed rose from 2.9% at hospital discharge to 26.5% at the last hospital visit. Table 7 summarized the prescription of each group of antihypertensive agents.

Table 5. Special investigations to identify the causes of hypertension in 55 cases

Investigations	n
Captopril renography	5
Doppler ultrasonography	22
MRA of renal arteries	7
Renal angiography	5
Renal ultrasonography	43
Renal biopsy	6
Urinary vanillylmandelic acid or catecholamines	7
I^{131}- Metaiodobenzylguanidine scan	2
Aldosterone/renin ratio	4

Discussion

We found 216 hypertensive emergency admissions in 1-year period. This indicated that hyper-

Table 6. Causes of death during the first hospitalization and the follow-up period

Cause of death	Number (%)		
	In the 1st admission	During follow-up	Total
Stroke	17 (60.7)	2 (5.4)	19 (29.2)
Acute myocardial infarction	1 (3.6)	3 (8.1)	4 (6.2)
ESRD	2 (7.1)	7 (18.9)	9 (13.8)
Infection	5 (17.9)	19 (51.4)	24 (36.9)
Pulmonary embolism	1 (3.6)	0	1 (1.5)
Upper GI hemorrhage	0	1 (2.7)	1 (1.5)
Pneumothorax	0	1 (2.7)	1 (1.5)
Unknown	2 (7.1)	4 (10.8)	6 (9.2)
Total	28 (100)	37 (100)	65 (100)

GI = gastrointestinal

tensive emergencies remain a clinical problem in spite of the availability of many effective BP lowering agents. A preliminary report of the on-going STAT registry in the US, which was aimed to include subjects with acute HT, supports our findings. There were 982 cases enrolled from 21 hospitals and 58% of them had evidence of TODs associated with hypertensive episodes[19]. Most of our cases had a history of HT as did cases in other studies[6,7]. Interestingly, 46% and 23% of our patients also had diabetes mellitus (DM) and ESRD, respectively. Since most of our cases with ESRD did not get renal replacement therapy, inadequate natriuresis and volume overload might have induced BP elevation and finally the occurrence of hypertensive emergencies. The fact that heart failure was more frequently found in cases with than without ESRD supports this hypothesis. The frequency of heart failure among all TODs was 65.2% in subgroup with ESRD as compared with 39.8% in those without ESRD.

The choice of initial antihypertensive medications seemed appropriate. Parenteral drugs in our hospital were nitroglycerine, sodium nitroprusside, nicardipine, diltiazem, hydralazine, and furosemide. Cardiac complications contributed around 70% of all TODs. Nitroglycerine is the drug of choice for BP lowering in these situations. Loop diuretics are also favorable in heart failure. The high frequency of cardiac complication rate (70%) was close to the rate at which nitroglycerine and furosemide were prescribed (64.4%). Sodium nitroprusside, which is more potent than nitroglycerine, was used in only 8.3% of cases. Light sensitivity of sodium nitroprusside makes its preparation more difficult than the other agents and this could be one of the reasons why sodium nitroprusside was not frequently used. Only 5 cases received nicardipine because the cost of nicardipine in our hospital was many times higher than nitroglycerine or sodium nitroprusside. The inappropriate use of short-acting nifedipine was found in 34 admissions. The administration of nifedipine by either route was not effective for BP lowering in this study, whereas serious adverse effects have been reported from the precipitous fall of BP after nifedipine administration[20]. Because of its unpredictable efficacy, nifedipine should not be used in hypertensive emergencies, which requires treatment in a few hours to limit the extent of TOD.

Intravenous BP lowering agents were not prescribed in 22.2% of all admissions, mostly because of the presence of acute stroke. Most of the patients with acute stroke have reactive HT, which usually resolves spontaneously[21]. Aggressive BP lowering in these patients may compromise cerebral perfusion. However, markedly elevated BP in acute stroke is associated with cerebral edema and a worsening prognosis[22,23]. It is generally recommended to lower BP in these patients only when BP is extremely high[24,25].

After initial management, the causes of HT were searched for in 30% of cases. Investigations revealed positive results in 9% of those tested. The main objective of the investigations was to look for the pathology of renal parenchyma or renal arteries. This may be the result of the fact that half of our cases had renal impairment. Glomerulosclerosis obtained from renal biopsies could not clarify the cause of HT. The lowest serum creatinine among these cases was 3.5 mg/dl suggesting that renal biopsies were performed too late when active renal pathology might not exist. Pheochromocytoma was rarely looked for as a result of the absence of typical symptoms in most of the cases. ARR was the least common test performed. The prevalence of primary aldosteronism is much higher after the introduction of ARR as a screening test[26,27]. According to the non-specific manifestations of primary aldosteronism, ARR must be performed whenever secondary HT is suspected. Although primary aldosteronism rarely presented as hypertensive emergencies[28], the ARR done here in only 4 cases might underestimate the presence of primary aldosteronism.

Table 7. The prescription of oral antihypertensive medications at hospital discharge and at the last hospital visit during the follow-up period

Drugs	Number (%)	
	Hospital discharge	Last visit
Diuretics		
Thiazide	19 (10.9)	8 (23.5)
Loop diuretics	89 (51.1)	1 (2.9)
Spironolactone	1 (0.6)	0
β-blockers	82 (47.1)	19 (55.9)
Calcium antagonists		
Dihydropyridine (DHP)	72 (41.4)	20 (58.8)
Non-dihydropyridine (non-DHP)	11 (6.3)	1 (2.9)
DHP and non-DHP	4 (2.3)	1 (2.9)
Angiotensin-converting enzyme inhibitors	89 (51.1)	13 (38.2)
Angiotensin II receptor blockers	5 (2.9)	9 (26.5)
α-blockers	23 (13.2)	4 (11.8)
Direct vasodilators	64 (36.8)	1 (2.9)
Central α_2 agonists	10 (5.7)	3 (8.8)

BP was well controlled with 2 to 3 oral drugs prescribed before discharge. Severe HT with TODs usually need multidrug regimens. ARBs were rarely used at hospital discharge but their prescription rate was 10 times higher during follow-up. The cost of ARBs in Thailand is much higher than ACEIs as a result of the generic ACEIs. Therefore, clinicians usually prescribe ACEIs first whenever renin angiotensin system blockers are indicated. If the patients are intolerant to ACEI-induced cough, found in 25% of our population[29], ARBs will be used instead. Hydralazine, which is no longer a major antihypertensive drug[13], was prescribed to 37% of cases at hospital discharge. This rate dropped to 3% at the last visit. The frequent use of hydralazine tablets immediately after the use of parenteral BP lowering agents may be explained by its rapid onset of action. The antihypertensive regimen was later adjusted according to patients' risk factors and TOD. Likewise, loop diuretics were the most common diuretics prescribed at the beginning because of the presence of heart failure in the majority of cases. However, thiazide-type diuretics, which are more suitable for the treatment of chronic HT due to their more vasorelaxant effects on arterioles, were used more frequently later.

The mortality rate of patients in this study is extremely high even when compared with high-risk hypertensive cases. Patients older than age 55 who were at high risk for cardiovascular events in HOPE[30] and ONTARGET[31] studies had a total mortality rate of 10-12% after 4-5 years of follow-up. In our study the mortality rate was 15% during hospitalization and 20% during follow-up. This very high mortality rate is in accordance with a study from Brazil. Sobrinho S, et al. showed the 5-month mortality of 21% among subjects with hypertensive emergencies[32]. Patients who died in the follow-up period had an average duration from the first hospital discharge to death of only 18 months. These findings indicated that hypertensive emergency cases remained at extremely high risk for death after the initial management. This may be the result of the unknown pathophysiology of these conditions[33] leading to non-specific treatment. Why acute TOD develops only in certain but not all cases with severe HT is not clearly known. Twenty-five percent of cases both in our study and in the STAT registry[19] were not taking the medications before the events. Therefore, compliance of these high risk patients needed to be greatly improved.

In conclusion, hypertensive emergencies remain a clinical problem at our institution. Most of the cases had histories of HT and DM or ESRD. Recommended investigations failed to identify the cause of HT. These patients had extremely high mortality rate while their adherence to treatment was extremely poor. All strategies to improve awareness, control, and compliance in hypertensive patients should be promoted to prevent their occurrence. Adequate long-term renal replacement therapy in patients with chronic kidney failure should also be helpful.

References

1. Lawes CM, Vander Hoorn S, Rodgers A. Global burden of blood-pressure-related disease, 2001. Lancet 2008; 371: 1513-8.
2. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, Jr., et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003; 42: 1206-52.
3. Staessen JA, Fagard R, Thijs L, Celis H, Arabidze GG, Birkenhager WH, et al. Randomised double-blind comparison of placebo and active treatment for older patients with isolated systolic hypertension. The Systolic Hypertension in Europe (Syst-Eur) Trial Investigators. Lancet 1997; 350: 757-64.
4. Adler AI, Stratton IM, Neil HA, Yudkin JS, Matthews DR, Cull CA, et al. Association of systolic blood pressure with macrovascular and microvascular complications of type 2 diabetes (UKPDS 36): prospective observational study. BMJ 2000; 321: 412-9.
5. Brenner BM, Cooper ME, de Zeeuw D, Keane WF, Mitch WE, Parving HH, et al. Effects of losartan on renal and cardiovascular outcomes in patients with type 2 diabetes and nephropathy. N Engl J Med 2001; 345: 861-9.
6. Martin JF, Higashiama E, Garcia E, Luizon MR, Cipullo JP. Hypertensive crisis profile. Prevalence and clinical presentation. Arq Bras Cardiol 2004; 83:131-6; 25-30.
7. Zampaglione B, Pascale C, Marchisio M, Cavallo-Perin P. Hypertensive urgencies and emergencies. Prevalence and clinical presentation. Hypertension 1996; 27: 144-7.
8. Cherney D, Straus S. Management of patients with hypertensive urgencies and emergencies: a systematic review of the literature. J Gen Intern Med 2002; 17: 937-45.
9. 2003 European Society of Hypertension-European Society of Cardiology guidelines for the management of arterial hypertension. J Hypertens 2003;

21: 1011-53.
10. Nieminen MS, Bohm M, Cowie MR, Drexler H, Filippatos GS, Jondeau G, et al. Executive summary of the guidelines on the diagnosis and treatment of acute heart failure: the Task Force on Acute Heart Failure of the European Society of Cardiology. Eur Heart J 2005; 26: 384-416.
11. Van de Werf F, Ardissino D, Betriu A, Cokkinos DV, Falk E, Fox KA, et al. Management of acute myocardial infarction in patients presenting with ST-segment elevation. The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. Eur Heart J 2003; 24: 28-66.
12. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, et al. Diagnosis and management of aortic dissection. Eur Heart J 2001; 22: 1642-81.
13. Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R, Germano G, et al. 2007 Guidelines for the Management of Arterial Hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). J Hypertens 2007; 25: 1105-87.
14. Rosei EA, Salvetti M, Farsang C. Treatment of hypertensive urgencies and emergencies. ESH Scientific Newsletter: Update on Hypertension Management. 2006; 7: No. 28.
15. Vaughan CJ, Delanty N. Hypertensive emergencies. Lancet 2000; 356: 411-7.
16. Phillips RA, Greenblatt J, Krakoff LR. Hypertensive emergencies: diagnosis and management. Prog Cardiovasc Dis 2002; 45: 33-48.
17. Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. Am J Obstet Gynecol 2000; 183: S1-S22.
18. Julius S, Kjeldsen SE, Weber M, Brunner HR, Ekman S, Hansson L, et al. Outcomes in hypertensive patients at high cardiovascular risk treated with regimens based on valsartan or amlodipine: the VALUE randomised trial. Lancet 2004; 363: 2022-31.
19. Hughes S. Acute hypertension: An undertreated condition. 11 Feb 2008 [cited 15 Aug 2009]. Available from: http://www.medscape.com/viewarticle/570027.
20. Peters FP, de Zwaan C, Kho L. Prolonged QT interval and ventricular fibrillation after treatment with sublingual nifedipine for malignant hypertension. Arch Intern Med 1997; 157: 2665-6.
21. Morfis L, Schwartz RS, Poulos R, Howes LG. Blood pressure changes in acute cerebral infarction and hemorrhage. Stroke 1997; 28: 1401-5.
22. Vemmos KN, Tsivgoulis G, Spengos K, Zakopoulos N, Synetos A, Kotsis V, et al. Association between 24-h blood pressure monitoring variables and brain oedema in patients with hyperacute stroke. J Hypertens 2003; 21: 2167-73.
23. Willmot M, Leonardi-Bee J, Bath PM. High blood pressure in acute stroke and subsequent outcome: a systematic review. Hypertension 2004; 43: 18-24.
24. Adams HP, Jr., del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, et al. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. Stroke 2007; 38: 1655-711.
25. Broderick J, Connolly S, Feldmann E, Hanley D, Kase C, Krieger D, et al. Guidelines for the management of spontaneous intracerebral hemorrhage in adults: 2007 update: a guideline from the American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group. Stroke 2007; 38: 2001-23.
26. Young WF, Jr. Minireview: primary aldosteronism—changing concepts in diagnosis and treatment. Endocrinology 2003; 144: 2208-13.
27. Kaplan NM. The current epidemic of primary aldosteronism: causes and consequences. J Hypertens 2004; 22: 863-9.
28. Bortolotto LA, Cesena FH, Jatene FB, Silva HB. Malignant hypertension and hypertensive encephalopathy in primary aldosteronism caused by adrenal adenoma. Arq Bras Cardiol 2003; 81: 97-100, 93-6.
29. Buranakitjaroen P, Sriussadaporn S, Phoojaroenchanachai M, Sangprasert P, Saravich S. Angiotensin converting enzyme inhibitor induced cough: experience in Siriraj Hospital. J Med Assoc Thai 2003; 86: 647-54.
30. Yusuf S, Sleight P, Pogue J, Bosch J, Davies R, Dagenais G. Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events

in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. N Engl J Med 2000; 342: 145-53.
31. Yusuf S, Teo KK, Pogue J, Dyal L, Copland I, Schumacher H, et al. Telmisartan, ramipril, or both in patients at high risk for vascular events. N Engl J Med 2008; 358: 1547-59.
32. Sobrinho S, Correia LC, Cruz C, Santiago M, Paim AC, Meireles B, et al. Occurrence rate and clinical predictors of hypertensive pseudocrisis in emergency room care. Arq Bras Cardiol 2007; 88: 579-84.
33. Marik PE, Varon J. Hypertensive crises: challenges and management. Chest 2007; 131: 1949-62.

ผู้ป่วยความดันโลหิตสูงฉุกเฉินมีอัตราตายสูงเมื่อเทียบกับผู้ป่วยความดันโลหิตสูงทั่วไป

วีรนุช รอบสันติสุข, อัญธิฌา วงศ์สุรินทร์, พีระ บูรณะกิจเจริญ

คณะผู้วิจัยได้ตั้งข้อสังเกตว่ายังพบผู้ป่วยความดันโลหิตสูงฉุกเฉินได้เป็นระยะในยุคปัจจุบันที่มียาลดความดันโลหิตที่มีประสิทธิภาพดีและปลอดภัยกว่าในอดีตมาก ร่วมกับยังขาดข้อมูลการพยากรณ์โรคในระยะยาวของผู้ป่วยกลุ่มนี้ จึงได้รวบรวมข้อมูลลักษณะทางคลินิก การรักษา และการสืบค้นเพื่อหาสาเหตุของโรคความดันโลหิตสูง โดยการทบทวนเวชระเบียนของผู้ป่วยที่ได้รับการวินิจฉัยว่ามีภาวะความดันโลหิตสูงฉุกเฉินและรับไว้รักษาในหอผู้ป่วยสามัญ ภาควิชาอายุรศาสตร์ คณะแพทยศาสตร์ศิริราชพยาบาลในปี พ.ศ. 2546 และติดตามผู้ป่วยจนถึงวันที่ 31 ธันวาคม 2550 รวบรวมผู้ป่วยได้ทั้งสิ้น 184 ราย ร้อยละ 90 ของผู้ป่วยทราบว่าเป็นโรคความดันโลหิตสูงอยู่แล้ว ร้อยละ 46 เป็นโรคเบาหวาน และร้อยละ 23 เป็นโรคไตวายเรื้อรังร่วมด้วย ความดันโลหิตเฉลี่ยแรกรับมีค่า 205.96/114.60 มิลลิเมตรปรอท พบภาวะแทรกซ้อนต่อหัวใจในร้อยละ 71 และโรคหลอดเลือดสมองในร้อยละ 23 ของผู้ป่วย ยาลดความดันโลหิตชนิดฉีดที่แพทย์เลือกใช้บ่อย ได้แก่ nitroglycerine และ furosemide มีผู้ป่วยเพียง 55 รายที่ได้รับการสืบค้นเพิ่มเติมเพื่อหาสาเหตุของโรคความดันโลหิตสูง ระยะเวลานอนโรงพยาบาลเฉลี่ย 9.8 วัน ร้อยละ 15 ของผู้ป่วยเสียชีวิตในโรงพยาบาล ร้อยละ 26 ขาดการติดต่อเพื่อรับการรักษา และร้อยละ 20 เสียชีวิตในระยะต่อมา มีเพียงร้อยละ 19 ที่ยังคงมารับการรักษาสม่ำเสมอ และได้รับยาลดความดันโลหิตเฉลี่ย 2.4 ชนิด โดยสรุปภาวะความดันโลหิตสูงฉุกเฉินยังคงเป็นปัญหาในประเทศไทย มักไม่พบสาเหตุของโรคความดันโลหิตสูงจากการสืบค้นที่ทำอยู่ในปัจจุบัน และผู้ป่วยกลุ่มนี้มีอัตราตายสูงเมื่อเทียบกับผู้ป่วยความดันโลหิตสูงทั่วไป