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ORIGINAL ARTICLE
ORİJİNAL ARAŞTIRMA

Investigation of Thrombotic Tendency in Hypertensive Urgencies

Hipertansif Acillerde Trombotik Yatkınlığın Araştırılması

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ABSTRACT

Aim: Hypertension is a significant risk factor for the development of thrombotic events. Mean platelet volume is a marker that correlates closely with platelet activity in uncontrolled hypertension. In this study, we aimed to monitor changes in MPV and other laboratory parameters during a 2-hour follow-up in patients diagnosed with hypertensive urgency and who were administered captopril or amlodipine treatment.

Material and Method: In this study, a total of 100 patients who were considered to have hypertensive urgency in the ED were separated into two groups in a randomized controlled manner. To reduce their blood pressure, 25 mg captopril tablets were orally given to fifty patients and 5 mg amlodipine tablets were orally given to fifty patients. MPV and other laboratory parameters were recorded at 0 minutes, 60 minutes, and 120 minutes.

Results: There was no difference between the two groups in terms of the patients' first MPV values (p>0.05). MPV was increased after 60 minutes in the captopril group. However, despite the evidence of a decrease at the end of 120 minutes, the difference was not found (p>0.05). MPV was decreased in the amlodipine group at the end of 60 and 120 minutes, but no difference was found (p>0.05).

Conclusion: In this study, the decrease in MPV values caused by amlodipine was more remarkable than that caused by captopril. We believe that for patients who present with hypertensive urgency, MPV values should be reduced in the early period for the prevention of thrombosis.

Keywords: Hypertensive urgency, mean platelet volume, thrombosis

ÖZ

Amaç: Hipertansiyon, trombotik olayların gelişimi için önemli bir risk faktörüdür. Ortalama trombosit hacmi, kontrolsüz hipertansiyonda trombosit aktivitesi ile yakından ilişkili bir belirteçtir. Bu çalışmada, hipertansif aciliyet tanısı alan ve kaptopril veya amlodipin tedavisi uygulanan hastalarda 2 saatlik takipte ortalama trombosit hacmi ve diğer laboratuvar bulgularındaki değişiklikleri izlemeyi amaçladık.

Gereç ve Yöntem: Bu çalışmada acil serviste hipertansif ivedi durum olduğu düşünülen toplam 100 hasta randomize kontrollü olarak iki gruba ayrıldı. Tansiyonlarını düşürmek için elli hastaya 25 mg kaptopril tablet ve elli hastaya da 5 mg amlodipin tablet oral yolla verildi. 0 dakika, 60 dakika ve 120 dakikadaki MPV ve diğer laboratuvar parametrelerindeki değişiklikler kaydedildi.

Bulgular: Hastaların başlangıç ortalama trombosit hacimleri açısından iki grup arasında fark yoktu (p>0.05). MPV, kaptopril grubunda 60 dakika sonra yükseldi. Ancak 120 dakika sonunda azalma olduğuna dair kanıtlara rağmen fark bulunamadı (p>0.05). 60 ve 120 dakika sonunda amlodipin grubunda MPV azaldı, ancak fark bulunmadı (p>0.05).

Sonuç: Bu çalışmada, amlodipinin neden olduğu MPV değerlerindeki düşüş, kaptoprilden daha fazla dikkat çekiciydi. Hipertansif ivedi durum ile başvuran hastalarda trombozun önlenmesi için erken dönemde MPV değerlerinin düşürülmesi gerektiğini düşünmekteyiz.

Anahtar Kelimeler: Hipertansif aciller, ortalama trombosit hacmi, tromboz

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INTRODUCTION

Hypertension is an independent risk factor for coronary artery disease (CAD), heart failure (HF), stroke, and renal failure (RF) and can cause death (1). According to the WHO, cardiovascular (CV) causes take the first place among the "preventable causes of death." In addition, hypertension is considered to be the most important disease among the preventable causes of death in the world (2).

Worldwide, a total of 1.38 billion people were reported to have hypertension in 2010. The prevalence of hypertension is 30.1% in women and 31.9% in men (3). Approximately 75% of patients with hypertension are aware of their high blood pressure (BP) and receive appropriate treatment. Proper management of hypertension greatly reduces the risk of stroke and mortality (4). It has been shown that most of the patients who applied to the ED with high BP measurements had an undetected chronic BP elevation (5). Acute elevation of BP is defined as a hypertensive crisis and is classified as a hypertensive emergency (HE) in the presence of end-organ damage and hypertensive urgency (HU) in the absence of it. Accurate diagnosis and appropriate treatment in patients with hypertension are of critical importance (6,7).

Platelets are activated in uncontrolled hypertension and contribute immensely to the increased tendency to thrombosis. MPV is one of the indicators closely related to platelet activity (8). It has been shown that MPV is higher in patients with hypertension (9).

In our study, we aimed to monitor the changes in MPV and other laboratory parameters levels, which is one of the indicators of susceptibility to thrombosis, by controlling the BP in patients who reported to the ED with any complaints, had high BP, and were thought to have HU. In addition, we ensured that patients with high BP were referred to hypertension outpatient clinics and received treatment at the earliest to prevent possible thromboembolic events in the future.

MATERIAL AND METHOD

The study involved 100 patients, whose written consent was obtained, in the Emergency Medicine Clinic of Kayseri Training and Research Hospital between Jun 1, 2013, and March 31, 2014. Nontraumatic patients with hypertension who applied to the ED as an outpatient or were brought in by an ambulance constituted the study group. Patients who were thought to have a diagnosis of HE with end-organ damage were not included in the study, whereas those with a diagnosis of HU were the target population of the study. Before the study, permission was obtained from the Ethics Committee of Erciyes University with the number 2013/332 dated May 7, 2013.

Data Collection and Patient Selection

The study included nontraumatic, conscious patients who came to the ED with any complaints had a systolic BP (SBP) of >140 mmHg and/or a diastolic BP (DBP) of >90 mmHg measured in both arms and were thought to have HU. There was no sex discrimination in the selection of the patients, and all patients with hypertension between the ages of 18 and 90 years who were eligible for the study were included after obtaining their verbal and written consent. The selection of the patients was performed with a controlled, randomized, and double-blind study design. The patients were divided into two groups as captopril angiotensin-converting enzyme inhibitor-ACEI) and amlodipine (calcium channel blocker-CCB) groups of 50 people. The demographic data, vital signs, and examination findings of the participants were recorded. To lower the BP, 25 mg captopril tablets were given to the patients in the first group, with the drug name covered. The name of the drug given to the patients was revealed during their discharge from the ED. With the same method, 5 mg amlodipine tablets were given orally to the second group of patients, and the name of the drug was revealed during their discharge from the ED. Complete blood count, biochemical markers, and lipid levels were measured in the patients belonging to both groups in the hospital laboratory a total of three times, i.e., before the treatment (0 minutes), at the 1st hour, and the 2nd hour. The obtained MPV and other blood parameters were recorded. SBP, DBP, mean arterial pressure (MAP), and heart rate of the patients were also recorded.

At the beginning of the study, those with a history of type II diabetes mellitus, dyslipidemia, CAD, obesity (body mass index>30 kg/m^2), chronic RF, liver failure, stroke, major surgery, or disease in the last 6 months, history of smoking, severe aortic insufficiency or stenosis, or used drugs that affect platelet function (heparin, acetylsalicylic acid, Clopidogrel, or warfarin) were excluded. During the study, which lasted approximately 14 months, 2785 (1.08%) of the 256,945 patients who applied to the ED on an outpatient basis or a stretcher were accepted as having hypertensive attacks. Of these, 1868 patients were excluded from the study according to the exclusion criteria stated above. The remaining 817 (29.3%) were diagnosed with HE and, hence, excluded from the study. After applying the exclusion criteria, 100 patients with HU (3.59%) were finally included in the study.

Statistical Analysis

The SPSS v 21.0(Statistical Package for Social Sciences) program was used for statistical analyses. Kolmogorov-Smirnov test was used for the normality analysis of the variables. Normally distributed continuous variables were presented as mean values and standard deviations. Analysis of parametric variables between groups was done with a student's t-test. Mann-Whitney U test was used for non-parametric variables that did not show normal

distribution. Qualitative variables were summarized as percentages. Chi-square tests were employed to compare categorical expressions. Correlation analysis was performed to determine the relationship between the variables. The paired-sample t-test was used to analyze the distribution of variables over time. Pearson's and Spearman's correlation analysis was used to evaluating the relationship between variables. p<0.05 was accepted as statistical significance in all tests.

RESULTS

Twenty (40%) patients in the captopril group and 13 (26%) patients in the amlodipine group were men. Into the groups in terms of age, sex, biochemical parameters, and blood lipid values were no significant differences (p>0.05), but body mass index (BMI) was significantly higher in the amlodipine group (p<0.05) (**Table 1**).

Table 1. Demographic, biochemical, and blood lipid characteristics of the participants

	Reference range	Captopril group n = 50	Amlodipine group n = 50	p value
Age (years)	18-90	60.2± 13.5	61.8± 13.9	0.56
BMI	18.5-29.9	26.2±2.7	27.3±2.2	0.039
Sex (male)		20(40%)	13(26%)	0.137
Glucose (mg/dL)	70-110	154.7±80.6	143.8±69.9	0.049
BUN (mg/dL)	7-20	18.8±7.2	16.8±6.1	0.132
Creatinine (mg/dL)	0.6-1.3	1.02±0.8	0.8±0.2	0.049
ALT U/L	0-45	21±14.3	20.5±13.4	0.863
AST U/L	0-41	22.7±7.9	23.7±10.8	0.608
Triglyceride (mg/dL)	35-150	202.4±122.2	224±159.7	0.451
Total cholesterol (mg/dL)	0-200	209.1±44.7	216.6±50.5	0.432
LDL cholesterol (mg/dL)	0-135	125.7±33.9	133.4±40.4	0.306
HDL cholesterol (mg/dL)	40-60	45.9±9.5	46.3±8.9	0.552
D- Dimmer (ng/mL)	0-500	193.8±144.6	307.3±550.6	0.162
Troponin I (ng/mL)	0-0.1	0.2±0.04	0.2±0.07	0.736
CK-MB (U/L)	0-25	18.5±11.7	16.4±6.4	0.262

BUN: blood urea nitrogen, ALT: alanine aminotransferase, AST: aspartate aminotransferase, LDL: low-density lipoprotein, HDL: high-density lipoprotein, CK-MB: creatine kinase-myocardial band

Baseline parameters (0 minutes) such as hemoglobin, white blood cell (WBC) count, platelet count, red blood cell distribution width (RDW), platelet distribution width (PDW), hematocrit values, MPV and neutrophil groups/lymphocyte (N/L) ratio there was no significant difference between the groups. (p>0.05) (**Table 2**).

Baseline parameters (0 minutes) such as MPV, N/L ratio, SBP, DBP, pulse pressure (PP), MAP, and heart rate (HR) values there was no significant difference between the groups (p>0.05) (**Table 3**). While there was a significant difference in the captopril group in terms of SBP, DBP, MAP, and PP values at the end of the first hour (p<0.05), there was no significant difference in MPV, N/L ratio, and HR values (p>0.05). In the captopril group, there was a statistically significant difference in N/L ratio, SBP, DBP, MAP, PP, and HR at the end of the 2nd hour (p<0.05), but no significant difference was found in the MPV values (p>0.05) (**Table 4**).

Table 2. Baseline (0 min) biochemical and hematological characteristics of the participants

	Reference range	Captopril group n = 50	Amlodipine group n = 50	P value
Hemoglobin (Hgb)	13-17 g/ dL	14± 1.57	14 ± 1.62	0.975
WBC	4.6-10.2 (10^3/uL)	8.27±2.81	8.72±2.83	0.428
Platelet value	130-400 (10^3/uL)	237.2±58.4	245.9±68.3	0.513
MPV value	7.2-11.1 fL	8.7±1.4	8.9± 1	0.471
PDW value	12 -26%	15.9±2.3	16.1±0.44	0.61
RDW value	37% -54%	39.3±8.9	40.9±3.02	0.243
Hematocrit value (Htc)	40% -52%	41.4±7.64	42.7±4.56	0.316
N/L ratio		3.6±4.4	2.6±1.9	0.145

WBC: white blood cell, MPV: mean platelet volume, N/L: neutrophil /lymphocyte ratio

Table 3. Patients' baseline (0 minutes) blood pressure and heart rate values

	Captopril group n = 50	Amlodipine group n = 50	P value
MPV (fL)	8.7±1.4	8.9± 1	0,471
N/L ratio	3,6±4,4	2,6±1,9	0,145
SBP	172,1±22,3	172±18,1	0,984
DBP	101±12,4	99±11,7	0,411
MAP	120,7±20,9	123,7±12,3	0,378
PP	71,4±19,9	72,6±15,4	0,754
HR	88±20,5	86,8±25,4	0,795

SBP: systolic blood pressure, DBP: diastolic blood pressure, MAP: mean arterial pressure or mean blood pressure, PP: pulse pressure, HR: heart rate

Table 4. Changes in the captopril group at the 1st and 2nd hour compared with the baseline parameters

	Baseline (0 hour) values n = 50	1st-hour values n = 50	2nd-hour values n = 50	P1 value *	P2 value **
MPV (fL)	8,69± 1,4	8.73± 1.5	8,65± 1,5	0,623	0,623
N/L ratio	3,59±4,4	3,76±3,8	4,39±4,9	0,712	0,712
SBP	172±22,3	141,6±20,1	127,7±16,2	<0,001	<0,001
DBP	101±12,4	82,3±12,5	76,7±9,3	<0,001	<0,001
MAP	120,7±20,92	101,7±13,8	91,1±16,6	<0,001	<0,001
PP	71,4±19,9	59,4±16,3	51±14,2	<0,001	<0,001
HR	88±20,5	85,4±14,8	82,3±12,2	0,126	0,126

MPV: mean platelet volume, N/L: neutrophil/lymphocyte ratio, SBP: systolic blood pressure, DBP: diastolic blood pressure, MAP: mean arterial pressure, NB: pulse pressure, HR: heart rate
*Comparison of baseline (0 minutes) parameters and 1st-hour values
** Comparison of baseline (0 minutes) parameters and 2nd-hour values

Furthermore, in the amlodipine group, there was no significant difference in baseline (0. min) MPV, N/L ratio, and HR (p>0.05), but there was a significant difference in SBP, DBP, MAP, and PP values (p<0.05). Although there was a significant difference in the N/L ratio, SBP, DBP, MAP, and PP values at the end of the 2nd hour in the amlodipine group (p<0.05), there was no difference in MPV and HR (p>0.05) (**Table 5**).

Table 5. Changes in the amlodipine group at the 1st and 2nd hour compared with the baseline parameters

	Baseline (0 hour) values n = 50	1st-hour values n = 50	2nd-hour values n = 50	P1 value *	P2 value **
MPV (fL)	8.87± 1	8.85±0.94	8,81± 0,96	0,754	0.450
N/L ratio	2,58±1,96	3,25±2,2	3,25±2,2	0,038	0,038
SBP	172±18,07	136,9±20,9	128,2±18,4	<0,001	<0,001
DBP	99±11,7	79,5±12,9	73,5±10,1	<0,001	<0,001
MAP	123,7±12,3	98,6±14,2	91,6±11,8	<0,001	<0,001
PP	71,4±19,9	59,4±16,3	54,7±13,3	<0,001	<0,001
HR	86,8±25,4	83,2±13,7	82,3±12,1	0,256	0,177

MPV: mean platelet volume, N/L: neutrophil/lymphocyte ratio, SBP: systolic blood pressure, DBP: diastolic blood pressure, MAP: mean arterial pressure, PP: pulse pressure, HR: heart rate
*Comparison of baseline (0 minutes) parameters and 1st-hour values
** Comparison of baseline (0 minutes) parameters and 2nd-hour values

When the MPV change between the groups at the 1st and 2nd hour was examined, although there was a slightly higher decrease in MPV at the end of the 2nd hour in the amlodipine group compared with the captopril group, there was no significant difference (p>0.05) (**Figure 1**).

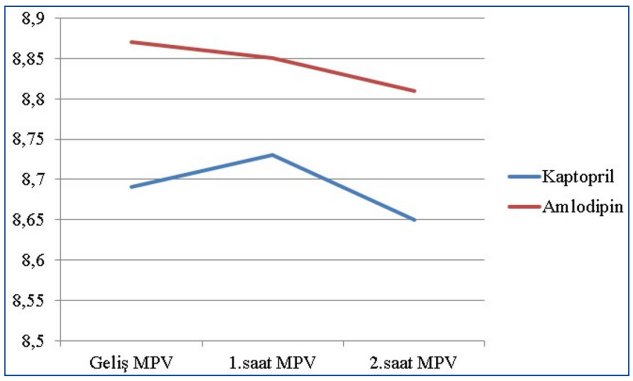

Figure 1. Mean platelet volume change with treatment between Captopril and Amlodipine groups (Baseline MPV, 1st hour MPV, 2nd hour MPV)

In the correlation evaluations, no correlation was found between the MPV change at the 1st hour and the decrease in the SBP value. Moreover, there was no correlation between the MPV change at the 2nd hour and SBP (p>0.05).

DISCUSSION

Hypertension is a serious health problem, and its proper management greatly reduces the risk of stroke and mortality (10). Among more than 145 million emergency patients in the USA each year, the estimated prevalence of high BP is close to 45% (11). It is estimated that globally approximately 1 billion people have hypertension and 7.1 million people die annually (6). In a multicenter study, 1546 of 333,407 patients admitted to the ED were found to have a hypertensive crisis (0.46%), and 391 of these patients (25.3%) were found to have HE (12). Similarly, in our study, 2785 (1.08%) of 256,945 patients who applied to the ED were found to have a hypertensive crisis and 817 (29.3%) of these patients were diagnosed with HE.

In hypertension, platelets are activated for various reasons. Platelets secrete numerous substances, such as β-thromboglobulin, PF4, P-selectin, thrombospondin, and glycoprotein Ia, IIa, and IIIb, which are important mediators of coagulation, thrombosis, inflammation, and atherosclerosis. Platelets of patients with hypertension are more sensitive to angiotensin II and catecholamines, which are potent stimulants of activation and aggregation of circulating platelets (8,13). Furthermore, platelets of patients with hypertension express more alpha2-adrenoceptors (14). MPV alone is also considered a platelet activation marker. Changes in MPV are very important for the early diagnosis of prothrombotic and thrombotic events. Because the larger platelets are metabolically more active, the volume of platelets is one of the determinants of their functions (15). Li Gang et al. reported that higher MPV values were an independent risk factor for the increased incidence of hypertension, supporting the role of platelet activation (16). MPV has been identified as an independent risk factor for hypertension, stroke, myocardial infarction (MI), pulmonary thromboembolism (PE), DM, preeclampsia, and deep vein thrombosis (17-19). In addition, increased MPV levels in patients with hypertension have been established to be an independent predictor of major cardiac side effects (19). In a study on the variability of MPV in the acute stroke phase, it was documented that MPV values increased as the size of the infarction increased (20). In light of all these studies, the changes in baseline MPV values recorded in the ED and the alterations in MPV values during hospitalization support the hypothesis that it can provide an idea about platelet reactivation and prognosis.

With antihypertensive treatment, hypertension-induced thrombocyte activation and prothrombotic status can be improved. It has been supported by many studies that endothelial dysfunction, platelet activation, and hemostasis disturbances are regressed with the treatment of hypertension (8,21). Several studies have indicated that most of the patients admitted to the ED with high BP had an undetected chronic high BP. It is of critical importance to reduce the morbidity and mortality of patients who are thought to have hypertension, with the correct diagnosis and appropriate treatment approach (5,6). In our study, we aimed to reduce the thrombotic process by regressing the existing endothelial dysfunction and platelet activation. This was achieved by facilitating the decrease in MPV in the acute period by administering 25 mg captopril or 5 mg amlodipine. Demirtunc et al. examined the effect of amlodipine on MPV in metabolic

syndrome and found that the daily administration of 10 mg of amlodipine did not have a significant effect on MPV (22). No study has been conducted so far investigating the effects of captopril and amlodipine on MPV. In our study, although there was a tendency of increased MPV in the captopril group at the end of the 1st hour compared with the baseline values of the participants, no statistical significance was found. A slight decrease was observed in the amlodipine group compared with the baseline values of the participants, but no difference was found. The reason for the increase in the MPV value in the captopril group at the end of the 1st hour is unknown, but the MPV value at the end of the 2nd hour decreased compared with the baseline values of the participants. Nonetheless, the decrease was not statistically significant. In the amlodipine group, there was a decrease in the MPV value compared with the baseline values of the participants at the end of the 2nd hour. However, there was no statistically significant difference.

Multifactorial causes, such as genetic factors, obesity, environmental factors such as lifestyle and excessive salt consumption, alcohol consumption, lack of physical activity, and psychosocial factors, play a role in the etiopathogenesis of hypertension (13). In their study, Doğru et al. expressed the relationship between MS components and MPV in 888 patients who were thought to have metabolic syndrome (MS); nevertheless, they did not find a relationship between the two (23). We found similar results in our study. It has also been stated in the literature that increased MPV values may be associated with complications in patients with hyperlipidemia and hypertriglyceridemia (24,25). In our study, there was no significant difference between the groups in terms of total cholesterol, LDL, TG, and other measured biochemical parameters. We think that the effect of these parameters on MPV is not different between groups. In their study, Muscari et al. observed that body fat percentage was associated with high (>8.4 fL) MPV values (26). In our study, patients with a BMI of ≥30 were excluded. There was a significant difference in BMI in the amlodipine group compared to the captopril group. However, in terms of baseline MPV values of the participants, the mean MPV value in the amlodipine group was higher than that in the captopril group. Interestingly, the decrease in MPV was greater in the amlodipine group at the end of the 2nd hour.

Comparing the MPV level between men and women, Bancroft et al. study showed that there was no difference between the two groups. Also, they showed that the MPV level is higher in young people than in the elderly population (27). It has been reported that while sex differences play a role in the prevalence and determination of prehypertension and hypertension, the rate of BP control is similar between women and men on antihypertensive drugs (28). In our study, the mean volume was found to be higher in women in all groups. However, there was no difference in terms of MPV elevation. Therefore, the data obtained in the present study are compatible with those in the literature.

According to the study conducted by Varol et al., the MPV values were found to be higher in patients with hypertension than in those without hypertension. In addition, MPV values of patients with prehypertension were found to be lower than those of patients with hypertension (29). In our study, there was no significant decrease in MPV 2 hours after the treatment in previously hypertensive participants in all groups. In participants who were not previously hypertensive, the decrease in MPV 2 hours after the treatment was greater. However, since there was no statistically significant difference, no conclusion could be drawn from this observation.

Numerous clinical studies have shown a positive correlation between total blood viscosity and the severity of arterial hypertension. Especially, severe hypertension is associated with red blood cell aggregation. On the contrary, ACEI, CCB, and α or β-adrenoreceptor blockers used to lower BP led to a significant improvement in blood flow or rheology. Abnormal blood flow or rheology is not directly related to arterial hypertension. However, it is associated with genetic or environmental factors, such as physical inactivity, obesity, smoking, and, chronic mental stress (18-20). High RDW may be associated with a risk of cardiovascular events, HF, and death in patients with MI (30). In our study, no difference was found in terms of baseline hematological parameters (hemoglobin, WBC, platelet, MPV, PDW, RDW, hematocrit, and N/L ratio).

Restoration of endothelial functions and reduction of increased platelet aggregation is one of the goals of modern antihypertensive therapy in essential hypertension. Therefore, the effect of ACEI inhibitors on endothelial and platelet functions is important. In a study, it was shown that after 1 week and 1 month of perindopril treatment, there was no significant change in thrombomodulin and β-thromboglobulin levels but a significant decrease in platelet aggregation was detected (31). In another study, it was established that ACE inhibitors did not cause a significant alteration in thrombomodulin and β-thromboglobulin levels and that there was no change in platelet count and size (32). None of the studies in the literature has investigated MPV change in the early (2 hours) period of hypertensive emergencies admitted to the ED. Furthermore, no study has been performed on the acute effects of captopril or amlodipine on MPV. This lack of investigations in the field highlights the importance of our research.

CONCLUSION

Identification and early treatment of hypertensive crisis in the ED are very important to prevent complications. MPV, which is an indicator of platelet activation, plays a role in the pathogenesis of complications related to thrombosis in many diseases. We think that lowering MPV in hypertensive emergencies is important in terms of early prevention of thrombosis. A slight decrease in MPV was observed with the use of captopril and amlodipine in the acute phase (the first 2-hour period). Notably, amlodipine, one of the drugs used in our study, lowered MPV more than captopril. However, because of the small number of participants and the short MPV follow-up of 2 hours, the effects of the drugs may differ in case of larger participation and longer follow-up periods. We suggest that amlodipine should be preferred over captopril for the reduction of MPV, which is an indicator of thrombosis, during the early period in patients diagnosed with HU.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was approved by the Erciyes University Clinical Research Ethics Committee with date May 7, 2013 and number 2013/332.

Informed Consent: Because the study was retrospective, written informed consent wasnot obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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