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RESEARCH

Association between left ventricular hypertrophy and the peak times of the R and P waves in hypertensive patients

Hipertansif hastalarda sol ventrikül hipertrofisi ile R ve P dalgalarının zirve zamanları arasındaki ilişki

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Abstract

Purpose: This study aimed to investigate the relationship between left ventricular hypertrophy (LVH), assessed by echocardiographic left ventricular mass index (LVMI), and electrocardiographically evaluated R wave peak time (RWPT) and P wave peak time (PWPT) in hypertensive patients.

Materials and Methods: A cross-sectional study was conducted involving 100 hypertensive patients, excluding those with specific medical conditions. Clinical and demographic data were collected, and electrocardiographic and echocardiographic examinations were performed. RWPT and PWPT were assessed along with other parameters.

Results: LVH was present in 33 out of 100 patients. Patients with LVH exhibited significantly higher RWPT (45.6 ± 4.9 vs. 35.0 ± 5.5) and PWPT (55.5 ± 15.8 vs. 49.1 ± 12.5) compared to those without LVH. Additionally, LVH patients showed increased left atrium anteroposterior diameter, and prolonged deceleration time. QRS fragmentation was more common in the LVH group.

Conclusion: The study highlights the association between electrocardiographic parameters (RWPT and PWPT) and LVH in hypertensive patients. These findings underscore the potential utility of electrocardiogram as a non-invasive tool for LVH assessment in clinical practice in hypertensive patients, aiding in risk stratification and therapeutic decision-making.

Keywords:. Electrocardiography, hypertension, P wave peak time, R wave peak time

Öz

Amaç: Bu çalışmada hipertansif hastalarda ekokardiyografik olarak değerlendirilen sol ventrikül kitle indeksi (SVKİ) ile belirlenen sol ventrikül hipertrofisi (SVH) ile elektrokardiyografik olarak değerlendirilen R dalgası tepe zamanı (RDTZ) ve P dalgası tepe zamanı (PDTZ) arasındaki ilişkinin araştırılması amaçlandı.

Gereç ve Yöntem: Spesifik tıbbi durumları olanlar hariç, 100 hipertansif hastayı kapsayan kesitsel bir çalışma yürütüldü. Klinik ve demografik veriler toplandı, elektrokardiyografik ve ekokardiyografik incelemeler yapıldı. RDTZ ve PDTZ diğer parametrelerle birlikte değerlendirildi.

Bulgular: 100 hastanın 33'ünde SVH mevcuttu. SVH'li hastalar, SVH olmayanlara kıyasla anlamlı derecede daha yüksek RDTZ (45.6 ± 4.9' e karşı 35.0 ± 5.5) ve PDTZ (55.5 ± 15.8' e karşı 49.1 ± 12.5) değerleri gösterdi. Ek olarak, SVH hastalarında sol atriyum ön-arka çapında artış ve deselerasyon zamanında uzama görüldü. QRS fragmantasyonu SVH grubunda daha sık görüldü.

Sonuç: Çalışma, hipertansif hastalarda elektrokardiyografik parametreler (RDTZ ve PDTZ) ile SVH arasındaki ilişkiyi vurgulamaktadır. Bu bulgular, elektrokardiyogramın klinik uygulamada hipertansif hastalarda risk sınıflandırmasına ve terapötik karar vermeye yardımcı olacak SVH değerlendirmesi için invaziv olmayan bir araç olarak potansiyel faydasının altını çizmektedir.

Anahtar kelimeler: Elektrokardiyografi, hipertansiyon, P dalgası tepe zamanı, R dalgası tepe zamanı

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INTRODUCTION

Over a billion people worldwide suffer from hypertension, a common public health issue. Because it can harm target organs, such as the heart, it is linked to higher rates of morbidity and mortality. Left ventricular hypertrophy (LVH) is a common cardiovascular condition associated with hypertension and is a substantial predictor of adverse cardiovascular events. Its presence correlates with an increased likelihood of congestive heart failure, coronary heart disease, and stroke occurrences[1]. LVH is characterized by structural adaptation of the heart to sustained high pressure and an increase in the left ventricular wall's size and thickness[2].

The degree of LVH can be assessed non-invasively using echocardiography, which allows measurement of left ventricular mass index (LVMI). LVMI is an established predictor of cardiovascular morbidity and mortality in hypertensive patients[3,4].

In clinical settings, the electrocardiogram (ECG) typically serves as the initial diagnostic tool for assessing LVH. Various established criteria are utilized for this assessment, such as the Sokolow–Lyon, Cornell voltage, Cornell voltage QRS duration product criteria, the Gubner index, and the Romhilt-Estes score[1]. While these criteria demonstrate high specificities and effectively identify individuals with significant LVH and heightened risk, their sensitivities are relatively low[1]. Consequently, a normal ECG result does not necessarily rule out the presence of LVH, underscoring the necessity for alternative ECG parameters to accurately detect this condition.

The R wave peak time (RWPT) on an ECG is the interval from the onset of the QRS complex to the peak of the R wave. It represents the time it takes for electrical activation to propagate from the endocardial surface to the epicardial surface of the ventricles[5]. LVH leads to electrical and structural changes in the heart, including changes in the depolarization process. In patients with LVH, RWPT may be prolonged, indicating delayed or abnormal electrical activation of the left ventricle[6]. This delay in activation may be attributed to the increased distance the electrical impulse has to travel through the hypertrophied myocardium[6].

P wave peak time (PWPT) is a parameter that represents the time measured from the onset to the peak of the P wave in ECG[7]. To date, there is no evidence of a direct association between PWPT and LVH. However, P-wave abnormalities can sometimes be observed in the presence of left atrial abnormalities such as hypertrophy or dilatation. This may suggest an indirect relationship between P wave changes involving PWPT and LVH, because LVH can often lead to left atrial enlargement.

In this study, we aimed to investigate whether there is a relationship between the presence of LVH as assessed by echocardiographic LVMI and electrocardiographically evaluated RWPT and PWPT in hypertensive patients. This study provides novel insights into the association between RWPT and PWPT and LVH in hypertensive patients. By demonstrating significant relationships between these ECG parameters and LVH, our findings suggest that RWPT and PWPT could serve as valuable non-invasive markers for LVH, enhancing early diagnosis and management of hypertensive patients at risk of adverse cardiovascular events. We hypothesized that hypertensive patients with LVH would exhibit prolonged RWPT and PWPT compared to those without LVH, indicating delayed or abnormal electrical activation associated with structural changes in the heart.

MATERIALS AND METHODS

Study design and sample

The Adana City Training and Research Hospital's ethics committee accepted the cross-sectional study (approval number: 23/11/2023-2955). Every action made throughout the study involving human participants complied with the Helsinki Declaration and the ethical guidelines established by national research committees. Informed consent was also supplied by the individuals. The study was conducted at the cardiology outpatient clinic of Adana City Training and Research Hospital from December 2023 to February 2024. The hospital is renowned for its comprehensive cardiovascular care services, adhering to stringent protocols to ensure data reliability. The clinical practices were carried out by a team of experienced cardiologists and trained technicians, all of whom are certified in advanced cardiovascular diagnostics and management.

This study comprised patients admitted to the cardiology outpatient clinic of Adana City Training and Research Hospital between December 2023 and February 2024 who had previously been diagnosed with hypertension and were receiving

antihypertensive medication. The study population consisted of 100 patients in total after exclusion criteria. Every patient had their clinical and demographic information recorded, including age, gender, body surface area, diabetes, smoking, hyperlipidemia, systolic and diastolic blood pressure, antihypertensive medications including beta-blocker, angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, calcium channel blocker, as well as blood laboratory parameters (complete blood count, creatinine, potassium, calcium, and total cholesterol levels). Echocardiography and electrocardiography were performed on each patient. Echocardiograms of the patients were performed immediately after the patients' electrocardiograms were taken. Exclusion criteria encompassed patients with prior cerebrovascular disease, atrial fibrillation, bundle branch block on electrocardiography, ejection fraction below 50%, established heart failure, severe valvular disease, chronic renal failure, utilization of antiarrhythmic drugs excluding beta-blockers and calcium channel blockers, and individuals below the age of eighteen.

The initial pool included 148 patients. However, 48 patients were excluded based on the following criteria: prior cerebrovascular disease (4 patients), atrial fibrillation (11 patients), bundle branch block on electrocardiography (5 patients), ejection fraction below 50% (12 patients), established heart failure (5 patients), severe valvular disease (4 patients), chronic renal failure (4 patients), utilization of antiarrhythmic drugs excluding beta-blockers and calcium channel blockers (2 patients), and individuals below the age of eighteen (1 patient). Consequently, 100 patients were included in the final analysis.

Electrocardiographic examination

For every patient involved in the study, a standard 12-lead surface ECG (25 mm/s, 10 mm/mV) was acquired. Electrocardiography recordings were scanned for a comprehensive evaluation of ECG characteristics, and an image processing application (imagej.net/ij/) was utilized for analysis. Heart rate, R wave peak time, P wave peak time, QRS duration, P wave duration, and QRS fragmentation were among the electrocardiographic characteristics that were assessed. RWPT was determined from the precordial lead V6 (Figure 1) and was defined as the duration from the start of the QRS complex to the R wave peak[5]. P wave peak time, which is the interval between the P wave's beginning and peak, was determined from the D2 lead (Figure 1)[7]. Additional spikes or notches inside the QRS complex were classified as QRS fragmentation[8].

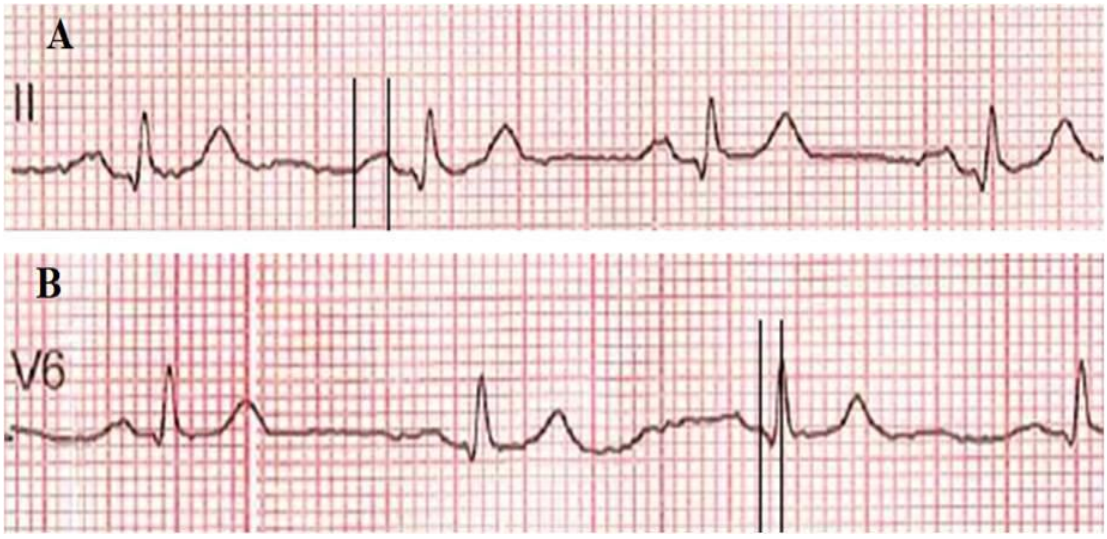

Figure 1A. Measurement of P wave peak time.

Figure 1B. Measurement of R wave peak time.

Echocardiographic examination

The American Society of Echocardiography established guidelines that were followed by the Standard 2D, M-mode, and Doppler assessments[9]. Evaluation was done on echocardiographic measures such as deceleration time, left atrium anteroposterior diameter, trans-mitral E/A, and LVMI. The Devereux Formula was utilized to compute the left ventricular mass[10]. By comparing left ventricular mass to body surface area, the LVMI was computed. An LVMI above 95 in women and above 115 in men was considered as LVH.

Statistical analysis

The statistical analysis was performed using IBM Corp.'s (Armonk, NY, USA) SPSS 21.0 program. A statistical significance threshold of $p < 0.05$ was employed to ascertain significance. The Shapiro-Wilk test was used to evaluate if the parameters had a normal distribution. For variables with normal distributions, the Student's t-test was used to compare continuous data between patients with and without LVH. Specifically, the Student's t-test was applied to compare the following variables: Age, body surface area, hemoglobin, RWPT, PWPT, LVMI.

For variables without normal distributions, the Mann-Whitney U test was used. This test was employed for comparing non-normally distributed variables such as: Systolic blood pressure, diastolic blood pressure, white blood cell count, blood glucose, creatinine, potassium, calcium, total cholesterol, heart rate, QRS duration, P wave duration, left atrium anteroposterior diameter, deceleration time, left ventricular ejection fraction, trans-mitral E/A.

Categorical variables were compared using the chi-squared test or Fisher's exact test, as appropriate. The chi-squared test or Fisher's exact test was used for comparing the prevalence of: Male gender, diabetes, smoking, hyperlipidemia, QRS fragmentation, R wave peak time ≥ 40 msec, P wave peak time ≥ 60 msec, use of beta-blockers, renin-angiotensin system inhibitors, calcium channel blockers. Continuous data were presented as mean ± standard deviation for normally distributed data or median and range for non-normally distributed data. Categorical variables were presented as frequencies and percentages.

The sample size was calculated based on a power analysis. For RWPT, we aimed to detect a medium to large effect size (Cohen's d = 0.6) with a power of 80% and a significance level of 0.05. The power analysis indicated that a minimum of 50 subjects per group would be required to achieve statistical significance. Similarly, for PWPT, aiming to detect a medium to large effect size (Cohen's d = 0.7) with the same power and significance level, the analysis indicated that at least 45 subjects per group were needed. Therefore, we included a total of 100 patients to ensure robust results and account for potential exclusions.

RESULTS

LVH was present in 33 out of 100 patients. The clinical and laboratory findings of the patients are presented in Table 1. The groups with and without LVH were statistically similar in terms of age, gender, and body surface area ($p=0.060$, $p=0.877$, and $p=0.081$, respectively). In addition, the proportions of diabetes, smoking, and hyperlipidemia were similar between the groups ($p>0.05$ for all). Beta-blockers, renin-angiotensin system inhibitors, and calcium channel blockers were used at similar rates in both groups ($p>0.05$ for all). There were no statistically significant differences between the groups in any of the laboratory findings including hemoglobin, white blood cell count, blood glucose, creatinine, potassium, calcium, and total cholesterol levels ($p>0.05$ for all). Systolic and diastolic blood pressures were also similar between the groups ($p>0.05$ for all).

The patients' echocardiography and electrocardiography findings are presented in Table 2. Left ventricular mass index, left atrium anteroposterior diameter, and deceleration time were statistically significantly higher in the group with LVH ($p<0.001$, $p=0.020$, and $p=0.040$, respectively). Trans-mitral E/A was lower in the LVH group ($p=0.001$). There was no difference in terms of ejection fraction between the groups. RWPT and PWPT were statistically significantly higher in the LVH group ($p<0.001$, and $p=0.031$, respectively). When utilizing the established 40 ms cut-off value for RWPT in previous researches[11], there was a notably increased occurrence of patients with RWPT equal to or exceeding 40 ms within the LVH group ($p<0.001$). When considering the 60 ms cut-off value for PWPT established in prior studies[12], the quantity of patients exhibiting PWPT of 60 ms or greater was significantly higher in the LVH group ($p=0.009$). While there was no significant difference between the groups in terms of P wave duration ($p>0.05$), it was observed that the

QRS duration was significantly longer in the LVH group (p=0.029). While QRS fragmentation was significantly more common in the LVH group (p=0.035), heart rates were similar between the groups (p>0.05).

Table 1. Clinical and laboratory findings of patients according to LVH groups with a p-value.

	Left ventricular hypertrophy (LVH)						
	All patients(n:100)		**Patients without LVH (n:67)**		**Patients with LVH (n:33)**		
Clinical and laboratory findings	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**p-value***
Age (years)	55.8	8.2	54.8	8.1	58.0	8.1	0.060
Body surface area (m^2)	1.9	0.2	2.0	0.2	1.9	0.1	0.081
Hemoglobin (g/dL)	13.2	1.8	13.0	1.9	13.6	1.4	0.097
	%	**n**	**%**	**n**	**%**	**n**	**p-value****
Male gender	42.0	42	43.3	29	39.4	13	0.877
Diabetes	17.0	17	17.9	12	15.2	5	0.950
Hyperlipidemia	20.0	20	17.9	12	24.2	8	0.457
Smoking	14.0	14	11.9	8	18.1	6	0.541
Beta-blockers	34.0	34	29.9	20	42.4	14	0.212
Renin-angiotensin system inhibitors	71.0	71	68.7	46	75.8	25	0.616
Calcium channel blockers	50.0	50	44.8	30	60.6	20	0.137
	median	**min-max**	**median**	**min-max**	**median**	**min-max**	**p-value*****
Systolic blood pressure (mmHg)	140.0	100.0-190.0	140.0	110.0-190.0	140.0	100.0-190.0	0.557
Diastolic blood pressure (mmHg)	82.5	50.0-120.0	90.0	50.0-100.0	80.0	60.0-120.0	0.214
WBC count ($10^3/\mu L$)	6.8	4.0-17.6	6.8	4.0-17.6	7.1	4.0-15.9	0.985
Blood glucose (mg/dL)	97.0	51.0-570.0	97.0	51.0-570.0	97.0	82.0-249.0	0.644
Creatinine (mg/dL)	0.7	0.5-1.3	0.7	0.5-1.3	0.7	0.5-1.3	0.692
Potassium (mEq/L)	4.7	3.5-5.4	4.8	3.5-5.4	4.7	3.7-5.2	0.414
Calcium (mg/dL)	9.3	8.9-10.1	9.3	8.9-10.1	9.3	8.9-10.1	0.610
Total cholesterol (mg/dL)	202.0	124.0-409.0	199.0	124.0-409.0	208.0	144.0-301.0	0.715

Abbreviations: SD, standard deviation; WBC, white blood cell. *Calculated with the Student's t-test. **Calculated with the chi-squared test, Fisher's exact test. *** Calculated with the Mann–Whitney U test.

Table 2. Echocardiography and electrocardiography findings according to LVH groups with a p-value.

	Left ventricular hypertrophy (LVH)						
	All patients(n:100)		Patients without LVH (n:67)		Patients with LVH (n:33)		
Echocardiography findings	Mean	SD	Mean	SD	Mean	SD	p-value*
Left ventricular mass index (g/m^2)	93.2	19.0	83.5	13.7	112.7	12.1	<0.001
	median	min-max	median	min-max	median	min-max	p-value***
Left ventricular ejection fraction (%)	60.0	50.0-70.0	60.0	50.0-70.0	60.0	50.0-70.0	0.184
Left atrium anteroposterior diameter (cm)	3.5	2.7-4.2	3.4	2.7-4.1	3.6	2.9-4.2	0.020
Trans-mitral E/A	0.9	0.4-1.8	1.0	0.4-1.8	0.8	0.6-1.6	0.001
Deceleration time (msec)	215.0	80.0-408.0	208.0	80.0-390.0	229.0	135.0-408.0	0.040
Electrocardiography findings	Mean	SD	Mean	SD	Mean	SD	p-value*
R wave peak time (msec)	38.5	7.3	35.0	5.5	45.6	4.9	<0.001
P wave peak time (msec)	51.2	13.9	49.1	12.5	55.5	15.8	0.031
	%	n	%	n	%	n	p-value**
QRS fragmentation	37.0	37	29.9	20	51.5	17	0.035
R wave peak time ≥ 40 msec	45.0	45	23.9	16	87.9	29	<0.001
P wave peak time ≥ 60 msec	34.0	34	25.4	17	51.5	17	0.009
	median	min-max	median	min-max	median	min-max	p-value***
Heart rate (beats/min)	78.5	57.0-109.0	79.0	60.0-109.0	75.0	57.0-107.0	0.256
QRS duration (msec)	95.0	70.0-115.0	95.0	70.0-110.0	97.0	85.0-115.0	0.029
P wave duration (msec)	108.5	78.0-125.0	105.0	78.0-125.0	110.0	80.0-120.0	0.111

Abbreviations: *Calculated with the Student's t-test. **Calculated with the chi-squared test, Fisher's exact test. *** Calculated with the Mann–Whitney U test.

DISCUSSION

The findings of our study shed light on the potential associations between ECG parameters consisting of RWPT and PWPT, and the presence of LVH in hypertensive patients. We found that both RWPT and PWPT were significantly prolonged in patients with LVH compared to those without LVH. The incidence of RWPT ≥ 40 ms and PWPT ≥ 60 ms, as defined by established cut-off values from prior researches[11,12], were also higher in the LVH group.

LVH prevalence ranges from 20% in mildly hypertensive patients to almost 100% in those with severe or complicated hypertension[1]. LVH was present in 33 of 100 patients in our study. This result was compatible with the literature, since our study population consisted of patients who had previously been diagnosed with hypertension and were using antihypertensive medications.

Alterations in the electrical activation of the heart, as reflected by prolonged RWPT and PWPT, may be indicative of electrical remodeling of the heart and changes in the propagation of electrical conduction due to structural changes associated with LVH in hypertensive patients. Our observation of higher RWPT in the LVH group within our study aligns with previous research demonstrating an association between prolonged RWPT and increased LVMI among hemodialysis patients[6]. A favorable association was found between PWPT and left atrial anterior-posterior diameter in a study examining the relationship between PWPT and left ventricular diastolic function in individuals with coronary slow flow phenomenon[13]. The association between PWPT and left atrial volume index has been established in

hemodialysis patients[12]. It was also demonstrated that there exists an association between PWPT and left ventricular end-diastolic pressure in hypertensive patients[14]. Moreover, a recent study has revealed that PWPT, linked to atrial remodeling, holds substantial prognostic value in forecasting the likelihood of patients developing atrial fibrillation[15]. The fact that we observed statistically significantly higher PWPT, left atrium anteroposterior diameter, deceleration time, and lower trans-mitral E/A in the LVH group in our study supports the findings of the aforementioned studies.

In previous studies, the occurrence of QRS fragmentation on an ECG is linked to elevated left ventricular mass and a heightened likelihood of exacerbated LVH in patients with hypertension[16,17]. Consistent with these studies, we also found an increased incidence of QRS fragmentation in the LVH group. Furthermore, our study identified an association between prolonged QRS duration and LVH. This finding aligns with previous literature suggesting that QRS duration increases with left ventricular mass in cases of LVH, as well as in normal cardiac conditions[18].

It is essential to consider the clinical implications of our findings. Electrocardiography, being both widely accessible and cost-effective, is routinely employed in clinical settings. Its non-invasive nature renders it especially advantageous for screening hypertensive individuals. The identification of the association between electrocardiographic parameters (RWPT and PWPT) and LVH in hypertensive patients holds promise for clinicians, providing crucial insights into the cardiovascular health of hypertensive patients. This knowledge aids in risk assessment, enabling more targeted and aggressive treatment approaches for high-risk patients.

However, it is important to recognize a few of our study's shortcomings. First, given the relatively small sample size of our study, it is imperative that the findings undergo validation through larger-scale studies. Second, our capacity to determine causality or temporal correlations between electrocardiographic anomalies and the onset of LVH is limited by the cross-sectional approach. Third, the participants in our study were hypertensive patients, and more research is necessary to determine whether our findings apply to other patient populations. Finally, our study did not involve a comparison of the identified parameters with existing electrocardiographic criteria for LVH, as this was beyond its scope. Scores and indices, primarily utilizing QRS amplitude parameters as indicators of hypertrophy, were examined. By incorporating QRS duration into these metrics, the sensitivity and specificity for detecting LVH were enhanced[19]. Our study introduced two novel parameters for LVH detection, potentially improving ECG sensitivity and specificity in hypertensive patients. Further research is warranted to validate these findings.

In conclusion, our study provides evidence of an association between electrocardiographic parameters, specifically RWPT and PWPT, and the presence of LVH in hypertensive patients. These results highlight the potential value of ECG as a non-invasive method for evaluating LVH in clinical settings.

Future research should aim to validate these findings in larger, diverse populations and explore the longitudinal implications of prolonged RWPT and PWPT in predicting adverse cardiovascular events. Additionally, comparative studies examining the sensitivity and specificity of RWPT and PWPT against traditional ECG criteria for LVH detection would be beneficial. Investigating the mechanistic pathways linking RWPT and PWPT to LVH could also provide deeper insights into the pathophysiology of hypertension-induced cardiac remodeling.

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