Late potentials and insulinemia in patients with hypertension*

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A relationship between hypertension and insulin resistance state or fasting insulinemia has been proposed. Insulin could be one of the causes of myocardial hyperthrophy. Left ventricular hypertrophy (L VH) is a risk factor for the patients with hypertension. Signal-averaged electrocardiography identifying late potentials might be useful to detect a subgroup of patients with echocardiographic left ventricular hypertrophy who are at high risk of sudden death, acute myocardial infarction, heart failure and arrhythmias. In order to investigate the relationships among all these factors, we examined 70 patients with primary (essential) hypertension. They were divided into 2 groups: Group 1:35 patients with left ventricular hypertrophy, Group 2:35 patients without left ventricular hypertrophy. They all did not have diabetes mellitus or coronary artery disease. Fasting insulin levels were measured, left ventricular mass was calculated by echocardiography, and late potentials were detected by signal-averaged ECG.

Results: late potentials were found to be significantly higher in Group 1 (p<05), there were no relations between left ventricular hypertrophy and fasting insulin levels (p>.05), and left ventricular hypertrophy was not related to mean blood pressure (p>.05). In conclusion, LVH has potential arrhythmia substrate and insulin does not appear to involve in the development of LVH. [Turk J Med Res 1992:10(6):321-323]

Keywords: Hypertension, Insulin resistance, Hypertrophy, Electrocardiography

The etiopathogenetic role of insulin resistance and/or hyperinsulinemia in essential hypertension is well known (1-5). Modan et al. have shown that the association between hypertension and insulin resistance is obesity-independent (1). Insulin resistance is usually represented by measuring fasting or stimulated (after glucose load) insulin levels rather than the complicated euglycemic insulin-clamp technique (1-3). Fasting insulin levels provide a crude measure of peripheral insulin resistance (5). On the other hand insulin has been claimed as a possible cause of myocardial hypertrophy (6,7), and myocardial hypertrophy is strongly predictive of ventricular arrhythmias (8). Signal-averaged ECG (SAECG) is beneficial to detect these patients who have high risk due to myocardial hypertrophy (9). The aim of this study was to show the relationships among the fasting insulin levels (FI), myocardial hypertrophy, and signal-averaged ECG findings in patients with essential hypertension.

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MATERIALS AND METHODS

We examined 70 patients with primary (essential) hypertension who were not on drug therapy were examing. They did not have diabetes mellitus or coronary artery disease assessed by clinical and laboratory findings. These patients were divided into 2 groups:

Group 1: 35 patients (16 male, 19 female) with left ventricular hypertrophy (LVH)

Group 2: 35 patients (14 male, 21 female) without LVH.

Mean age for Group 1 was 58±7, for Group 2 was 50±10 years . Mean blood pressure (MBP) was 134±19 mmHg in Group 1,125+12 mmHg in Group 2.

Fasting insulin level was measured by an Insulin-radioimmunoassay kit (INS-RIA-100) from Medgenix, Belgium. Normal values for this test range from<4 to 25 uU/ml.

LVH was assessed by M-mode and two-dimensional echocardiography (HP 7702 A model machine)

Table 1. The clinical and laboratory findings in patients

	Group I	Group 2	p value
Mean age	58+7	50+10	<.001
Mean blood pressure	134±19	125±12	>.05
Fasting insulin (mU/ml)	7±6	5+4	>.05
Late potentials (present)	16 (46%)	6(17%)	<05*
Late potentials (absent)	19(54%)	29 (83%)	<.05*

*x^2-test

according to the recommendations of the American Society of Echocardiography and the Penn Convention. Left ventricular mass index was calculated for each patient by dividing left ventricular mass by body surface area. 125 g/m was chosen as a cutoff point to detect LVH (10).

Signal averaged ECGwas recorded by Marquette Electronics Hi-resolution ECG, with vector magnidude filtered at 40-250 Hz with a noise level less than 1 uV. Normal values for SAECG parameters were these:

1. Filtered QRS duration <120 ms

2. The duration of Low Amplitude Signals <40mV—<40 ms

3. Root-mean-square amplitude of the terminal 40 ms->>25 uV. Late potentials were considered present if 2 of these 3 parameters were abnormal.

Statistical comparisons were made by Student t test and Chi-squared test.

RESULTS

The findings for all patients and the statistical comparisons between the two groups are listed in Table 1. It can be seen that insulin does not appear to involve in the development of LVH, because fasting insulin levels in both groups are statistically significant (p>.05). There is no relation between MBP and LVH (p>.05). on the other hand the presence of the late potentials (LP) is higher in Group 1 than in Group 2 (p<.05) and the absence of LP is higher in Group 2 than in Group 1 (p<05).

DISCUSSION

The present study simply indicates several features in patients with primary hypertenison. First, the fasting insulin levels in both groups are not different. Fasting insulin levels in our study seem to be lower than the figures reported in the literature (1,3), but we did not measure the insulin levels in our normotensive controls. However, if we could have measured the stimulated insulin levels in our patients, we would have found higher levels. But these figures show that insulin does not appear to involve in the development of LVH, because there is no statistical difference between the two gruops. Second, the presence of the late potentials is higher in the Group 1 as shown in other studies (9,11,12). This indicates potentials arrhytmia substrate for the LVH group. But that remaines to be proved, because there are a few studies recently published showing that the presence of late potentials does not predict or corelate to the complex arrhythmias seen in the hypertensive patients with LVH (11). The, mean blood pressures in both groups were similar and there was no relation between mean blood pressure and LVH.

Hipertansiyonda geç potansiyeller ve insulinemi

İnsülin rezistansı veya açlık insülinemisi ile hipertansiyon arasında ilişki tespit edilmiştir. İnsülinin ayrıca hipertrofi yapıcı etkisi üzerinde durulmaktadır. Sol ventrikül hipertrofisi hipertansiyonda önemli bir risk faktörüdür: ani ölüm, akut miyokard infarktüsü, kalp yetmezliği ve aritmiler için. Geç potansiyelleri tespit eden Signal-averaged EKG ise bu yüksek riski taşıyan hasta grubunu tespit edebilir. Bütn bu faktörler arasındaki ilişkiyi tespit etmek üzere 70 primer hipertansiyonlu hasta iki gruba ayrılarak incelendi.

Grup 1:35 hasta, sol ventrikül hipertrofisi (+)

Grup 2:35 hasta, sol ventrikül hipertrofisi (—)

Hastaların hiçbirinde diabetes mellitus ve ASKH yoktu. Açlık insülin seviyesi ölçüldü, ekokardiyografi ile sol ventrikül kitle hesabı yapıldı, geç potansiyellerde Signal-averaged EKG ile değerlendirildi.

Neticeler: Grup Vde geç potansiyeller önemli oranda fazla bulundu (p<0.05), insülin seviyesi ile sol ventrikül hipertrofisi arasında bir ilişki yoktu (p>0.05), ortalama kan basıncı ile sol ventrikül kitlesi arasında ilişki bulunamadı (p>0.05).

Sonuç olarak, sol ventrikül hipertrofisinde potansiyel bir aritmi kaynağının olduğu ve insülinin sol ventrikül hipertrofisinin oluşumunda bir rolünün olmadığı gösterildi.

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Anahtar Kelimeler:Hipertansiyon, İnsülin rezistansı, Hipertrofi, Elektrokardiyografi

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