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# Correlation between Dietary Sodium and Electrocardiographic Left Ventricular Hypertrophy Among Hypertensives

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**= Abstract =**

In hypertensives, electrocardiographic left ventricular hypertrophy (LVH) is an independent risk factor for cardiovascular disease. Animal and human studies suggest an association between left ventricular mass and dietary sodium (Na) intake. This study determined if dietary Na intake in a homogenous ethnic population is associated with electrocardiographic LVH (S in V1 + R in V5 $\geq$ 3.5 mV). Blood pressure (BP), body mass index, EKG, and 24 hour urine Na and potassium (K) excretion were determined in 40 otherwise healthy Korean patients with untreated essential hypertension on the standard Korean diet. Among these variables, only Na excretion (mmol/day) was significantly different between those with and without LVH [LVH+ : 357 $\pm$ 50, LVH− : 268 $\pm$ 25 ($p = 0.04$]. Thus, dietary sodium intake may be predictive of electrocardiographic LVH.

**Key words** : sodium, left ventricular hypertrophy, hypertension, korea, nutrition

## Introduction

Left ventricular hypertrophy (LVH) in hypertensive patients is a risk factor for cardiovascular disease independent of the level of the blood pressure [1, 2].

There are several subgroups of hypertensive patients that have a known higher occurrence of LVH than the general hypertensive population, including males [1], the elderly [1], diabetics [3], and black patients [4]. Our experience in a Hypertension Clinic in Pusan, Korea suggests that there is a very high occurrence in Korean hypertensives. In 1,700 hypertensives with a diastolic blood pressure of 90~104 mmHg, the occurrence rate was 18% by electrocardiogram (unpublished observations). This compares with 5% and 12% in whites and blacks respectively in the United States [5].

Until the widespread use of echocardiography, LVH was thought to be an infrequent finding. Data from the Framingham Study comparing echocardiographic LVH with electrocardiographic LVH reveal that the electro-cardiogram is quite specific for LVH ventricular hypertrophy but not sensitive [5]. Whether LVH is measured by electrocardiography or echocardiography, cardiovascular disease risk is high compared to hypertensives without LVH [1, 2, 5].

Until recently, the pathophysiologic mechanism

of LVH was thought to be related only to hemodynamic load[6, 7, 8]. Findings of a poor correlation between the presence of echocardiographically determined LVH and blood pressure level have stimulated studies searching for possible non-hemodynamic influences.

One proposed non-hemodynamic factor is the increased dietary intake of sodium. Studies in the United States, Germany and France have demonstrated an association between dietary sodium intake and left ventricular mass[9, 10]. However, no study demonstrating a statistically significant relationship between dietary sodium intake and electrocardiographic LVH has been reported. This study was designed to compare dietary sodium intake as measured by 24 hour urinary excretion in patients with and without LVH by electrocardiogram.

## Methods

All the patients entering the study were hypertensive Korean males or females ages 21 to 70 years with an average seated diastolic blood pressure of 95 to 119 mmHg. Patients underwent a two week period with no antihypertensive medications before inclusion criteria were considered. Patients were selected from persons attending the Hypertension Clinic of Baptist Hospital, Pusan, Korea. Patients with a known history of any heart disease, left bundle branch block on electrocardiogram, any secondary form of hypertension, severe alcohol abuse, or pregnancy were excluded.

Trained observers measured blood pressure using mercury sphygmomanometers, using the fifth phase (or disappearance) of the Korotkoff sounds in the seated position in accordance with American Heart Association recommendations. Blood pressure was measured two times at five-minute intervals, with the average taken as the subject's blood pressure.

A 12-lead electrocardiogram was performed using standard methods. Interpretation was performed by the study physician. Diagnosis of LVH was made on the basis of the sum of precordial SV1 plus RV5 or RV6 at least 3.5 mV.

Urine sodium and potassium excretion were measured by timed 24 hour urine collection, with measurements done by standard methods in the laboratory of Baptist Hospital, Pusan, Korea. Body height was measured in centimeters with a fixed rule, and body weights were measured on a standard balanced beam scale.

Written, informed consent was obtained from each patient. The protocol for the study was approved by the Hospital Research Committee.

Statistical analysis was performed on a microcomputer. The one-sided t-test for independent data was used to test differences between the two groups. Statistically significant differences were defined as $P < 0.05$ between groups of data.

## Results

Of the 48 patients entered into this study, eight were excluded for inadequate 24 hour urine collections. Of the remaining 40 patients, 12 were found to have LVH by the criteria noted above; 28 were found not to have LVH.

Table 1 compares those with and without LVH. As noted in the table, there were no statistically significant differences between the two groups regarding age, systolic blood pressure, diastolic blood pressure, body mass index, potassium excretion, or sodium/potassium ratio. However, those subjects with LVH had a statistically significant higher sodium excretion than those without LVH.

## Discussion

Among systolic blood pressure, diastolic blood pressure, body mass index, sodium excretion, potassium excretion, and sodium/potassium ratio, only sodium excretion was significantly different in patients in this study population with LVH and those without LVH by electro-

**Table 1.** Ventricular hypertrophy and various factors

| | $LVH^+$ (n = 12) | $LVH^-$ (n = 28) | p value |
|---|---|---|---|
| Male / Female | 7 / 5 | 13 / 15 | |
| Age (years) | 51 ± 9 | 53 ± 8 | .25 |
| Hypertension Duration (years) | 7 ± 2 | 8 ± 3 | .29 |
| Systolic blood pressure (mmHg) | 166 ± 7 | 163 ± 5 | .11 |
| Diastolic blood pressure (mmHg) | 107 ± 6 | 105 ± 7 | .20 |
| Body Mass Index | 24.7 ± 2.4 | 25.3 ± 2.0 | .38 |
| Sodium excretion (mmol / day) | 357 ± 168 | 268 ± 126 | .04 |
| Potassium excretion (mmol / day) | 90 ± 90 | 64 ± 35 | .10 |
| Sodium / Potassium ratio | 4.8 ± 1.3 | 4.8 ± 1.5 | .49 |

values are mean ± standard deviation

cardiogram. This is consistent with other human studies which have demonstrated a stronger association between sodium intake and left ventricular dimensions by echocardiography than with other measured parameters, including systolic blood pressure, diastolic blood pressure, and body mass index [9, 11].

Animal studies have demonstrated an absence of LVH in spontaneously hypertensive rats deprived of sodium from an early age [12]. Similar effects in two kidney, one clip renal hypertensive rats are seen [13, 14].

As yet, the strongest evidence from animal studies which indicates that sodium intake may be important in the development of LVH was recently reported by Frohlich, et al. WKY rats on a high (4%) sodium intake had a higher left ventricular mass than those on a low or normal sodium intake, despite no difference in mean blood pressure between the groups [15].

Studies in human adolescents demonstrate that normotensive adolescents with hypertensive parents have greater left ventricular dimensions than do normotensive adolescents with normotensive parents [16]. Further, young patients with borderline hypertension have an increase in left ventricular mass [17]. These studies strongly argue against a hemodynamic mechanism as the only cause of LVH and suggest a mechanism beginning early in life.

The link between dietary sodium intake and hypertension has long been debated. Most researchers conclude that the INTERSALT study [18] clearly established the relationship between daily urinary sodium excretion and blood pressure. Although the link between hypertension and sodium is established, the pathogenetic mechanism for this remains unresolved.

The pathogenetic mechanisms of dietary sodium causing LVH also are not clear. Reactivity of the sympathetic nervous system has been demonstrated to be a determinant of LVH [19]. Excess dietary sodium intake may be a primary factor in stimulation of the sympathetic nervous system leading to LVH.

If one assumes that sodium is causative or permissive in the development of LVH in those genetically predisposed, then onset is likely in childhood or adolescence. Studies demonstrating LVH in the very young support this hypothesis [16, 17].

Studies designed to demonstrate the relationship between dietary sodium intake in adolescence and adult left ventricular dimension should give further insight, and plans for these studies are underway.

We conclude that among all examined clinical factors, dietary sodium intake, as measured by 24

hour urine sodium excretion, is the only factor related to any electrocardiographically determined LVH in a statistically significant fashion. Further studies are needed to determine if restriction of dietary sodium from an early age might prevent LVH in patients with essential hypertension.

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