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Research Article

The relationship between magnesium and ambulatory blood pressure, augmentation index, pulse wave velocity, total peripheral resistance, and cardiac output in essential hypertensive patients

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Abstract

Magnesium levels have been shown to be associated with elevated blood pressure (BP), endothelial dysfunction, insulin resistance, vascular calcification, inflammation, and atherosclerosis. It was also demonstrated that patients with hypertension have increased inflammation, insulin resistance, and endothelial dysfunction. However, the relationship between magnesium, ambulatory BPs, and central hemodynamic parameters were not evaluated extensively. Serum magnesium levels, ambulatory blood pressures, augmentation index (Aix), pulse wave velocity, total peripheral resistances, and cardiac output were measured for all patients. In total, 184 essential hypertension patients were enrolled. In univariate analysis, magnesium levels were correlated with hemoglobin ($r = +0.155$; $P = .037$), albumin ($r = +0.180$; $P = .018$), pulse pressure (daytime; $r = -0.170$; $P = .021$), pulse pressure (24-hour; $r = -0.156$; $P = .035$), Aix (daytime; $r = -0.223$; $P = .002$), Aix (nighttime; $r = -0.169$; $P = .022$), and Aix (24-hour; $r = -0.247$; $P = .001$). In regression analysis, magnesium levels were independently and conversely associated with daytime Aix ($P < .0001$), nighttime Aix ($P = .019$), and 24-hour Aix ($P < .0001$). We suggest that magnesium levels were associated with Aix but not with total peripheral resistances, pulse wave velocity, cardiac output, and central BPs. The unique mechanisms related with magnesium and Aix but not shared by other central parameters needs to be determined. J Am Soc Hypertens 2014;8(1):28–35.

Keywords: Augmentation index; blood pressure; cardiac output; hypertension; magnesium; pulse wave velocity.

Introduction

Recently, lower magnesium level has been shown to be associated with elevated blood pressure (BP).[1–3] Additionally, studies have shown that magnesium may play a protective role for endothelial dysfunction, insulin resistance, vascular calcification, inflammation, and atherosclerosis.[4–7] Besides, not only serum magnesium levels per se, but increased dietary magnesium intake also confers protection against the incidence of diabetes, metabolic syndrome, hypertension, and cardiovascular disease.[8] It was demonstrated that patients with hypertension (HT) have increased inflammation,[9] insulin resistance,[10] and endothelial dysfunction,[11] all of which were related to hypomagnesemia. Additionally, not only peripheral BPs but central BPs and markers of central hemodynamics were also associated with inflammation, endothelial dysfunction, insulin resistance, and oxidative stress in hypertensive patients.[12–14] Since magnesium is related to BP, and hypomagnesemia, peripheral BP, and central hemodynamics are associated with above mentioned pathologic conditions, it could be possible that magnesium levels may be associated with various peripheral and central hemodynamic parameters. Thus by the light of aforementioned data, the current study was conducted to investigate the relationship between magnesium levels with office, ambulatory, and central BPs in essential HT patients. Additionally, the relationship between augmentation index (Aix), pulse wave velocity (PWV), total peripheral resistance (TPR), and cardiac

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output (CO) with magnesium has been also been investigated.

Methods

This cross-sectional study included essential HT patients attending the nephrology outpatient clinic of a university hospital. The study was conducted in accordance with the Declaration of Helsinki, and local ethical approval and informed consent was obtained before enrollment. The inclusion criteria for the patient enrollment were described as patients >18 years of age with essential HT who wanted to participate. The exclusion criterion for the current study was as follows:

Patients who had suffered from acute coronary syndrome, myocardial infarction, angina pectoris, or coronary revascularization procedure (coronary stent replacement and coronary artery bypass graft surgery; three patients), stroke (one patient), or peripheral revascularization within last 3 months (one patient), patients with Alzheimer disease and dementia (two patients), patients with heart failure (two patients), patients with hypo- (two patients), and hyperthyroidism (one patient), patients with renal artery stenosis (one patient), patient with adrenal mass (one patient), patients with rhythm problems and ST–T changes (six patients), patients with type 1 diabetes mellitus (two patients), patient with chronic liver disease (two patients), and patients who did not want to participate (15 patients) were excluded. Coronary artery disease was defined as presence of previous myocardial infarction, angina pectoris, or coronary revascularization procedure. Patients with secondary hypertension, rhythm problems, type 1 diabetes mellitus, hepatic, autoimmune, or malignant disease were also excluded. On 12-lead electrocardiogram, all included patients had normal sinus rhythm, no conduction disturbances, and no ST-T changes. Patients with known coronary artery disease who were free of the above-mentioned cardiovascular events and who did not undergo any revascularization procedures within the last 3 months were included. Our study did not include any shift-worker patients. All attending patients underwent the following procedures: anamnesis, office BP measurement, physical examination, routine biochemistry analysis, 24-hour ambulatory BP monitoring, determination of CO, determination of central BPs, determination of PWV, determination of TPR, and determination of Aix. To determine renal function and 24-hour urinary protein and albumin excretion, 24-hour urine specimens were collected. During the anamnesis procedure, demographic and clinical characteristics were recorded by an interview.

Office Blood Pressure Measurement

Office BP measurements were recorded by Omron MZ model (Omron Health Care, Mukou City, Kyoto, Japan) sphygmomanometer. Blood pressures were measured according to European Society of Hypertension guidelines.[15]

Ambulatory Blood Pressure Measurement

Ambulatory BP measurement was performed by Mobil-O-Graph Arteriograph (IE.M. GmbH, Stolberg, Germany) device, which has been validated.[16] The device was set to obtain BP readings at 30-minute intervals during the day (07:00 AM–10:00 PM) and at 60-minute intervals during the night (10:00 PM–07:00 AM). Each ambulatory BP monitoring dataset was first automatically scanned to remove artifactual readings according to preselected editing criteria. All subjects were instructed to rest or sleep between 10:00 PM and 7:00 AM (nighttime) and to continue their usual activities between 7:00 AM and 10:00 PM (daytime).

Measurement of Central Blood Pressures, Augmentation Index, Cardiac Output, Total Peripheral Resistance and Pulse Wave Velocity

By using Mobil-O-Graph arteriograph device with an ARC solver method (Austrian Institute of Technology, Vienna), pulse wave forms from brachial artery were recorded during 24 hours. This method, which oscillometrically captures the pulse wave form from the brachial artery by an upper-arm cuff, has been validated before.[17] The recordings were carried out at diastolic pressure level for approximately 10 seconds using a conventional BP cuff and a high fidelity pressure sensor (MPX5050, Freescale Inc, Tempe, AZ). The sensor is connected to a 12 bit A/D converter by means of an active analogue band bass filter (<0, >25 Hz). After digitalization, the signal processing was performed using a three-level algorithm. In a first step, the single pressure waves were verified for their plausibility by testing the position of minima and the corresponding wavelengths. During the second stage, all single pressure waves were compared with each other to recognize artifacts. Thereafter, an aortic pulse wave is generated by the means of a generalized transfer function. The idea behind a transfer function is the modification of a certain frequency range within the acquired pulse signal to get the aortic pressure wave.[17] The first positive zero crossing of the fourth-order time derivative of the generated aortic pulse wave represents the desired inflection point. In the last step, the coherence of the measured parameters was verified. Therefore, the inflection point of each single pulse wave was compared with the mean inflection point. The recording time of the oscillometric signal at diastolic level allows the derivation of central hemodynamic parameters, such as central BPs, Aix@75 (Aix adjusted for heart rate 75), and CO as well as TPR from the pulse wave form by means of a transfer function. For the calculation of CO, the patients were measured automatically using software