Status of serum magnesium in Sudanese patients with cardiac syndrome X

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Abstract

Background:Cardiac syndrome X (angina chest pain, positive stress - ECG, and normal coronary angiogram) has serious medical complications. Magnesium may be an important factor associated with the disease. The present study aimed to examine the association between serum magnesium and cardiac syndrome X (CSX) in Sudanese patients.

Materials and methods:A total of 50 patients with CSX and their matching control were enrolled in this study, 4 ml of venous blood were collected and placed in plain containers, centrifuged and

used for determination of serum magnesium. Magnesium quantified by using a calmagite method. Data were entered to the computer and analysed by using SPSS.

Results:Median (75th -25th quartile) of the serum magnesium showed no significant difference between the patients with CSX and their well-matched control [2.4(1.9-2.8) vs. 2.5 (1.9-3.2) mg/dl;*P*= 0.150]. Direct correlation was observed between serum magnesium and urea [r= 0.214;*P*=0.032].

Conclusion serum magnesium was not associated with cardiac syndrome X in this setting.

Keywords: cardiac syndrome X , Magnesium, Sudan, chest pain

Introduction

Cardiac syndrome X (CSX) is defined as typical angina chest pain, a positive response to stress testing and normal coronary arteriography (CAG) [1]. CSX is associated with increased frequency of myocardial infarction (MI), stroke, congestive cardiac failure, and death. Subsequent functional disability secondary to the syndrome was also reported. Patients with CSX represented 10 – 20% of those undergoing coronary angiography. The aetiology of CSX remains mysterious. Suggested mechanisms include abnormal subendocardial perfusion, smooth muscle cell hypertrophy, Autonomic imbalance, and imbalance between the endothelial-derived nitric oxide (NO) (vasodilator) and Endothelin-1 (ET-1) (vasoconstrictor) [2-5]. Other suggested mechanisms include Coronary endothelial dysfunction, myocellular ischemia, abnormal cardiac sensitivity, abnormal pain perception or pain threshold [6-10]. Others also suggested the potential role of sodium-hydrogen exchange and high levels of C-reactive protein as markers of coronary vascular dysfunction [11, 12].

Magnesium is essential intracellular cations that should be tightly regulated [13]. Magnesium act as a co-factor for over 600 enzymes including major enzymes in protein synthesis and energy dealing enzymes [14]. Disturbance in magnesium levels was associated with many cardiovascular diseases such as ventricular tachycardia, coronary artery calcification, hypertension, atherosclerosis, coronary spasm and sudden cardiac arrest [15-17]. However, little is known about magnesium level in cardiac syndrome X patients, although Guo et al observed a decrease in intracellular magnesium levels in patients with CSX, but this disappears when comparing the extracellular magnesium [18]. The present study aimed to examine the association between serum magnesium and CSX in Sudanese patients. Such basic science research of paramount importance, as it provides an evidence for any possible intervention and a better understanding of the disease nature.

Materials & methods

Fifty patients with CSX and their well matching control residing in Khartoum State, Sudan were involved in the present study after signing a written informed consent. The study was carried out during the period from February 2011- June 2012 in Al Shaab Hospital. Inpatients patients were asked to participate in the study. After providing the signed informed consent, demographic, social, and clinical data were collected from all participants using a pre-structured interview questionnaire directly from the patients. The study received Ethical approval from a local institutional committee at Al-Neelain University.

Blood sampling and magnesium quantification

Four ml of venous blood were taken and collected in a plain tube and allowed to clot at room temperature and then centrifuged at 4.000 rpm to obtain serum. The clear serum was taken immediately for analysis. Magnesium was measured by calmagite method. Briefly, magnesium reacts with calmagite (blue dye) in an alkaline media to produce a reddish complex. Protein interference can be avoided by adding 9-ethylene-oxide adduct. Hypomagnesaemia were considered if serum magnesium less than 1.6 mg/dl. Lipid profile, random blood glucose, and serum creatinine were measured by using standard procedures.

Statistical analysis

Data were entered into the computer and analysed using SPSS for windows version 18.0 software. Student t-test was used to compare means between the cases and controls in case of normally distributed data, while Mann-Whitney U test used in case of abnormally distributed data. The chisquare test used for comparing categorical variables. A *P*-value less than 0.05 was considered significant.

Results

Both groups were well matched in their basic characteristics, age, sex and their biochemical parameters (table 1). No significant difference were observed between the median (25th-75th quartile) of serum magnesium in cases and control group [2.4(1.9-2.8) vs. 2.5 (1.9-3.2) mg/dl;*P*= 0.150], respectively. In correlation analysis, only serum magnesium significantly directly correlated with the blood urea [r=0.214; *P*=0.032], no other correlations were observed, table 2.

Table 1: Socio-demographic and biochemical data in cases with cardiac syndrome X and their controls.

Variables	Cases N=50	Controls N=50	*P*-value
Age, years	44.9 (8.0)	40.3 (8.4)	0.06
Sex			1.000
Male	20 (40)	20 (40)	
Female	30 (60)	30 (60)	
Magnesium mg/dl	2.4(1.9-2.8)	2.5 (1.9-3.2)	0.150
RBG, mg/dl	129 (39.5)	141.3(34.5)	0.100
Cholesterol, mg/dl	182 (18.1)	180 (20.3)	0.941
LDL, mg/dl	136.7 (14.6)	136.6 (16.4)	0.971
HDL, mg/dl	49.7 (7.1)	46.6 (11.3)	0.114
TAG, mg/dl	128 .7 (5)	128.1 (14)	0.712
Urea, mg/dl	32.3 (10.7)	32.6(8.8)	0.976
Creatinine, mg/dl	1.17 (0.49)	1.21(.13)	1.000

Data expressed as mean (SD) or N(%) as applicable.

Table 2: correlation between age, blood urea, creatinine, and random blood glucose and serum magnesium in patients with cardiac syndrome X

Variables	Magnesium		Urea		Creatinine		Random blood glucose	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*
Age	0.079	0.437	0.101	0.315	-0.057	0.570	0.099	0.329
Magnesium			0.214	0.032*	-0.121	0.232	-0.070	0.492
Urea					-0.053	0.599	-0.041	0.688
Creatinine							0.079	0.435

Discussion

In this study, no association was found between serum magnesium and cardiac syndrome X. This result is similar to that of Guo et al [18]. However, such comparison should be taken cautiously, as both cases and controls have a lower normal range of magnesium. Perhaps, this may reflect the low dietary intake of magnesium in Sudanese culture or increased urinary losses. Magnesium plays a pivotal role in normal physiological action in the cardiac myocytes. It acts as a major regulator of the cations and anions trafficking in and out of the cell [19]. Hence, hypomagnesaemia has been linked with various arrhythmias, which may lead to sudden cardiac arrest [15]. Likewise, hypomagnesaemia observed in patients with plaque formation, calcification of soft tissues, atherosclerosis and coronary spasm. But, the exact mechanism remains unknown [20]. On the other hand, increase dietary magnesium intake has a significant inverse relation with the development of cardiovascular diseases [21]. Moreover, experimental studies showed that diet rich in magnesium decrease the risk of cardiovascular disease by 15%

[21].Recently, magnesium reported as a predictor of the coronary artery disease morbidity and mortality [22].

This study has several limitations; firstly, the source of low magnesium was not determined, as quantification for dietary and urinary magnesium were not done. Secondly, full blood count was not done to enable better interpretation of the result. A further study addressing these issues with larger sample size is needed.

Conclusion & recommendations

Serum magnesium has no role in the cardiac syndrome X. Further study is needed.

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