Abstract:

Dyslipidemia is an established risk factor of acute myocardial infarction (AMI), but measurement of macro metals like magnesium can be helpful in the prevention and better management of AMI. The aim of this study was to estimate serum magnesium in AMI. This is a case control type of study carried out in the Department of Biochemistry, Dhaka Medical College, Dhaka during the period of January 2015 to December 2015 with a total number of 100 study subjects. Acute myocardial infarction patients were selected as case (50) from coronary care unit (CCU), Department of Cardiology, Dhaka Medical College Hospital. Normal healthy individuals were selected as control (50) from the attendants of patients, relatives and doctors. Serum level of magnesium were assessed for both case and control groups. The mean values of the variable were compared between them by statistical analysis using SPSS version 16. For all the statistical analysis P<0.05 was considered as significant. The mean values of serum magnesium were 1.63±0.27mg/dl in cases. The mean values of serum magnesium were 2.35±0.28 mg/dl in control group. Significant differences were found in mean values between case and control groups and differences were highly significant (p<0.001). In AMI, serum magnesium level was found to be lower in this study. Serum magnesium is an important trace element that act as cofactor in many biochemical reactions. Decrease level of this important trace element may contribute to pathogenesis of AMI. So with other biochemical risk parameters, routine assessment of serum magnesium level is advocated, which might be helpful for prevention and better management of AMI.

Key words: Myocardial infarction, Serum magnesium level.

Introduction:

Coronary artery disease (CAD) is an important medical and public health issue because it is common and leading cause of death throughout the world. Bangladesh has been experiencing epidemiological transition from communicable disease to non-communicable disease (NCD). The overall mortality rate has decreased significantly over the last couple of decades. But death due to chronic diseases, specially the 'fatal four' i.e. cardiovascular disease (CVD), cancer, chronic respiratory disease and diabetes, are increasing in an alarming rate1.

Atherosclerotic coronary artery disease causing myocardial ischemia may manifest itself either as acute myocardial infarction (AMI), unstable angina or effort angina. The World Health Organization (WHO) estimated that 12.2% of worldwide deaths were from ischemic heart disease in 2004. People of South Asia have a 1.5-fold greater susceptibility to MI than the general population2. The exact prevalence of MI in Bangladesh is not known. Recent data indicates the prevalence between 1.85% and 3.4% in rural and 19.6% in an urban sample of working professionals3. Traditional risk factors of MI are helpful in diagnosis; specific clinical markers would be valuable in identifying the persons who are at risk3. Many prospective studies have implicated metal ions in the genesis of myocardial infarction. Therefore, attention is being focused on metals as risk factors for AMI. Magnesium is the fourth most common cation in the body and the second most common intracellular cation after potassium. It has a fundamental role as a cofactor in more than 300 enzymatic reactions involving energy metabolism and nucleic acid synthesis4. Magnesium modulates ion transport by pumps, carrier and channels. It intervenes in the action of serum calcium and sodium/potassium ATPase (NA⁺/K⁺ATPase).
Serving as a cofactor in the enzyme system, it influences sodium and potassium flux across the cell membrane. Magnesium blocks outward movement of potassium through potassium channels in cardiac cells. Decrease in magnesium causes outward movement of potassium, including depolarization and thereby causing cardiac arrhythmias.

The total body magnesium content of an average adult is 25 gram or 1000 mmol. Approximately 99% of total body magnesium is intracellular or bone deposited, with only 1% present in plasma. Magnesium is ionized or complex to filterable for glomerular filtration, while 20% is protein-bound. Normal plasma magnesium concentration is 1.6-2.6 mg/dl.

Magnesium deficiency may result in failure to inhibit entry of calcium into myocardial cells, failure to extrude calcium from cells, formation of crystals in mitochondria and failure of sarcoplasmic reticulum to sequester excess calcium. In clinical and population studies where magnesium tissue level can be measured, evidence of hypomagnesemia is commonly found. Hypomagnesemia occurs in about 65% of intensive care unit patients and in 11% of the general population. The clinical manifestations of hypomagnesemia include neuromuscular hyperactivity, psychiatric disturbances, calcium/potassium abnormalities and cardiac effects. Other clinical data support a relationship between magnesium and cardiovascular function. Deficiency in magnesium has been shown to cause an increase in cardiac arrhythmias. Lower level of magnesium was found in the heart muscle of persons who died suddenly from ischemic heart disease compared to people who died from other causes. Therefore, magnesium therapy has been tested as a treatment for people with a known or suspected myocardial infarction. This study was designed to investigate the serum level of magnesium in AMI patients.

Materials and Methods:

This case control study was conducted in the Department of Biochemistry, Dhaka Medical College, Dhaka, Bangladesh. The duration of the study was from January 2015 to December 2015.

The study comprises of 50 cases of AMI admitted in CCU and 50 normal healthy individuals (attendants of patients, relatives & doctors) as control using purposive sampling technique. Diagnosed cases of AMI of both sexes who were admitted in the hospital within 72 hours of symptoms and age 40-65 years were included as case. Control was healthy adult of both sexes with age range 40-65 years. Patient with history of IHD, congenital cyanotic heart disease, CKD, DM, COPD, malignancy, history of medication (like lipid lowering drug, magnesium supplement, zinc supplement, antiplatelet drug), any acute inflammatory conditions (like RTI, UTI), pregnancy and lactation were excluded from the study.

With all aseptic precaution 8ml of venous blood was drawn from anterior cubital vein in a disposable plastic syringe and immediately transferred to a dry clean test tube which was allowed to clot. Then serum was separated after centrifuging at 3000 rpm for 10 minutes and was collected in ependrop tube, labeled appropriately. Serum magnesium was estimated by colorimetric method at the Department of Biochemistry, Dhaka Medical College. Normal level of serum Magnesium is 1.6-2.6 mg/dl, the cut of value for the study was 2 mg/dl.

A preformed questionnaire sheet was used to record information. Informed written consent was taken from the participants. Initial evaluations of the study population were done by taking history (Demographic, family H/O hypertension, diabetes, obesity and relevant drug history). Pulse, blood pressure, height, weight and BMI were measured and laboratory findings of serum magnesium were recorded in the preformed data collection sheet. Cardiac marker (Troponin-I) level was collected from patient's record book. Statistical analysis were performed with SPSS, version 16. All data were processed and presented as mean and standard deviation. Difference of mean among the case and control groups were compared by unpaired 't' test, chi-square test and Fisher exact test and determination of correlation between variables were done by Pearson's correlation coefficient test. For all statistical analysis p<0.05 was considered as significant.

Results:

Table-I shows the sex distribution in case and control group. The sexes are equally distributed between case and control (P>0.05). It also shows age group for 100 sample size. The minimum age was 40 years and maximum age was 65 years with the mean age 53.30±6.44 years. The age group are equally distributed between case and control (p>0.05).

| Table I: Distribution of patients according to demographic profile |
|--------------------------------|-----------|----------|-----------------|-----------------|
| Characteristics | Case | Control | Test statistics | P value |
| Sex | Female | 14 | 16 | $X^2=0.19$ | 0.414 | df=1 |
| | Male | 36 | 34 | | | |
| Total | 50 | 50 | | | |
| Age group (years) | 40-50 | 17 | 21 | Mean age =53.30±6.44 year | |
| | 50-60 | 26 | 23 | (40-65) | 0.711 | |
| | 60-70 | 7 | 6 | $X^2=0.682$ | |
| Total | 50 | 50 | | | df=2 |

*Chi square test was done at the level of 95% confidence interval.*
Table-II shows that Mean±SD of S. Mg levels were 1.63±0.27 and 2.34±0.28 in case and control respectively. The level of S. Mg is significantly low in case group in comparison with control (p <0.001).

Table-II: Level of S. Magnesium between case and Control

| Parameter | Case(mean±SD) | Control(mean±SD) | t    | p-value |
|-----------|--------------|-----------------|------|---------|
| S. Mg(mg/dl) | 1.63±0.27    | 2.34±0.28       | 12.874 | <0.001  |
|            | (1.21-2.33)  | (1.80-2.96)     |      |         |

*Unpaired t test was done with 95% confidence interval. The ranges are shown within parenthesis.

Figure-I shows among the case 46 persons having serum magnesium level <2mg/dl (92%) and 4 persons having serum magnesium level >2md/dl (8%).

Table-III shows decreased serum magnesium level has very strong association (p value <0.001) with AMI patient.

Table-III: Association of Serum Magnesium with cases.

| S. Mg(mg/dl) | Case (MI) | Control (Normal) | Total | Test statistics | P-value |
|--------------|----------|------------------|-------|-----------------|---------|
| <2           | 46       | 8                | 54    | X^2=58.13       | <0.001  |
| >2           | 4        | 42               | 46    | df=1            |         |
| Total        | 50       | 50               | 100   | Fisher’s exact  | test=0.00007 |

In the present study the sexes were equally distributed between case and control, there was no significant difference found in gender between case and control group. There was no significant difference found in age of the case and control, age of the respondent were equally distributed. The study reports have inverse association between serum magnesium levels and acute myocardial infarction. The mean values of serum magnesium in case were 1.63±0.27mg/dl. In control group the mean values of serum magnesium were 2.35±0.28mg/dl. Thus, significant difference was found in mean values between case and control groups. Some previous studies showed similar significant decrease in serum magnesium level of MI patient when compared with the corresponding control and it was clear that magnesium level of the MI groups was significantly lower than that of control groups (p<0.02-14).

Conclusion:

Serum magnesium is an important trace element act as cofactor in many biochemical reactions. Decrease level of this important trace element may contribute to pathogenesis of AMI. So with other biochemical risk parameters, routine assessment of serum magnesium level is advocated, which might be helpful for prevention and better management of AMI. Study with large sample will be helpful to evaluate the definite role of serum magnesium in acute myocardial infarction.

References:

1. Islam AM, Majumder A. Coronary artery disease in Bangladesh: A review. Indian heart journal 2013; 65(4):424-35.
2. Barakat K, Wells Z, Randhany S, Mills P, Timmis A. Bangladeshi patients present with non-classic features of acute myocardial infarction and are treated less aggressively in east London, UK. Heart 2003; 89(3):270-9.
3. Ibrahim AK, Serum copper, zinc, and magnesium in acute myocardial infarction in Ramadi municipality. Al-Anbar Medical Journal 2009; 7(1):130-7.
4. Fawcett W, Haxby E, Male D. Magnesium: physiology and pharmacology. British journal of anaesthesia 1999; 83(2):302-20.
5. Esen F, Telci L. Magnesium in ICU: sine qua non. In: Vincent J-L, ed. Yearbook of Intensive Care and Emergency Medicine 2008 Springer-Verlag Berlin Heidelberg 2008. p. 491-501.
6. Burtis CA, Ashwood ER, Bruns DE. Tietz textbook of clinical chemistry and molecular diagnostics-e-book: Elsevier Health Sciences; 2012.
7. Lal L, Murmu H. Serum Magnesium in Patients with Acute Myocardial infarction. International journal of scientific study 2016; 4(3):167-9.
8. Frost FJ. Studies of minerals and cardiac health in selected populations. Nutrients in Drinking Water. WHO 2005;101.
9. Parikka H, Tuovinen L, Naukkarinen V, Tjeltvedt J, Pohjola-Sintonen S, Heikkilä J, et al. Decreases by magnesium of QT dispersion and ventricular arrhythmias in patients with acute myocardial infarction. European heart journal. 1999; 20(2):111-20.
10. Oirim O, Ochui Y. The role of calcium and magnesium in the development of atherosclerosis. Experimental and clinical pharmacology. Annals of the New York Academy of Sciences. 1990; 598:444-57.
11. Hruby A, O’Donnell CJ, Jacques PF, Meigs JB, Hoffmann U, McKeeown NM. Magnesium intake is inversely associated with coronary artery calcification: the Framingham Heart Study. JACC: Cardiovascular Imaging. 2014; 7(1):59-69.
12. Metwalli O, Al-Okbi S, Motawi T, El-Ahmady O, Abdoul-Hafeez S, El-Said E. Study of serum metals and lipids profile in patients with acute myocardial infarction. J Islam Acad Sci. 1998; 11(1-5-12.
13. Akila A, Anandraj J, Karthikaivan S, Serum Magnesium Levels in Acute Myocardial Infarction. IOSR-Journal of Dental and Medical science (IOSR-JDMS) 2017; 16(5):35-40.
14. Chakraborty P, Hoque M, Paul U, Husain F. Serum magnesium status among acute myocardial infarction patients in Bangladesh. Mymensingh medical journal: MMJ. 2014; 23(1):41-5.