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RESEARCH ARTICLE

MOLYBDENUM AS A BIOMARKER OF MYOCARDIAL INFARCTION

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ABSTRACT

Objective: to determine the difference in serum Molybdenum and Malondialdehyde levels among Myocardial Infarction (MI) patients and healthy control subjects.
Methods: 60 MI patients and 60 healthy volunteers as control were included in this study. Serum levels of Molybdenum was determined using Electro thermal atomic absorption spectroscopy (ETAAS), whereas Malondialdehyde (MDA) level was measured using UV-VIS spectroscopy.
Results: highly significant differences in serum Mo and MDA levels were observed between the (MI) patients and healthy control (P<0.0005)
Conclusion: our study found that the highly increasing of Mo concentration in (MI) patients sera maybe used as a marker for Myocardial Infarction depending on its relationship with xanthine oxidase, which recently considered as a biomarker for Myocardial Infarction

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INTRODUCTION

Myocardial Infarction (MI) is an object of coronary heart disease, which defined as a part of acute coronary syndrome (Joseph *et al.*, 2000). All the trace elements are harmful to our body beyond a certain level, In some studies it was found that trace elements may play a vital role, resulting in either harmful or beneficial effects by damaging or protecting vessel wall and altering lipid profile, these trace elements acts as a contributing factors for the growing burden of MI. Molybdenum is an essential element for humans, as well as for animals and plants (Schwarz *et al*., 2013). At least 50 molybdenum-containing enzymes were known by 2002, mostly in bacteria, and the number is increasing with every year; (Enemar *et al*., 2004)(Mendel *et al.,* 2006). Mammalian Mo-dependent enzymes are known, all of them harboring a pterin-based molybdenum cofactor (Moco) in their active site, In humans and other mammals, molybdenum is a key constituent of at least three important enzymes: sulfite oxidase, xanthine oxidase and aldehyde oxidase (Kisker *et al*., 1997) (Mendel and Ralf, 2009). In some animals, and in humans, the oxidation of xanthine to uric acid, a process of purinecatabolism, is catalyzed by xanthine oxidase, a molybdenum-containing enzyme. The activity of xanthine oxidase is directly proportional to the amount of molybdenum in the body. Molybdenum concentration also affects protein synthesis, metabolism, and growth. (Mitchell and Phillip, 2003).

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Figure 1 show the chemical structure of xanthine oxidase when combined with Molybdenum as a co-factor. Trace amounts of molybdenum are found in a wide variety of foods, and human exposure to molybdenum may occur via the diet, drinking water and occupational exposure from mining operations and industrial uses. The human body contains about 0.07 mg of molybdenum per kilogram of body weight (Holleman *et al*., 2001).with higher concentrations in the liver and kidneys and in lower in the vertebrae (Considine *et al*., 2005). Molybdenum is also present within human tooth enamel and may help prevent its decay (Curzon *et al*., 1971). Human deficiencies of molybdenum have not been linked toinadequate dietary intake (Nielsen, 1999) (Turnlund and Friberg, 2007) (Considine *et al*., 2005). Reports of molybdenum deficiency in humans have been limited to genetic defects that interfere with the molybdenum cofactor's ability to activate molybdoenzymes and to one case of feeding molybdenum-free total parenteral nutrition (Considine *et al*., 2005).

The toxicity of molybdenum compounds in humans has been observed to be low, and in general, soluble molybdenum compounds (e.g., sodium molybdatedihydrate) are more toxic than insoluble compounds (Vyskocil and 1999). Oxygen free radical generation has been shown to be an important mechanism of cellular injury in ischemic myocardium (Pandey *et al*., 2000). Several mechanisms have been proposed to be involved in the generation of oxygen free radicals but xanthine oxidase has been shown to be a major source of free radical generation under ischemic conditions (Xia *et al*., 1996). Oxy free radicals produced by the action of xanthine oxidase, in

turn, oxidize cellular proteins and membranes resulting in myocardial cellular injury (Gorman and Zweier, 1990). Malondialdehyde (MDA), a naturally occurring end product of membrane lipid peroxidation, is one of the most frequently used biomarker for free radical mediated damage (Nielsen *et al*., 1997).

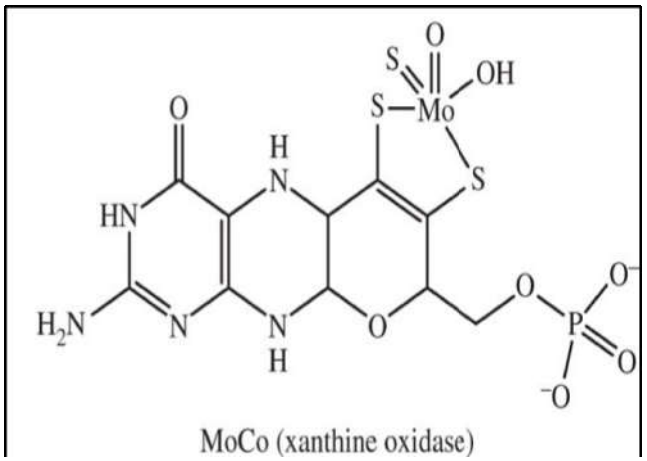

Figure 1. Molybdenum as co-factor with Xanthine oxidase

MATERIALS AND METHODS

All chemicals used in this study with highly purified material and no farther purification done.

Sample collection

About (5 ml) of blood was drawn from forearm vein of patients suffering from myocardial infarction. The blood samples were allowed to clot for about (10 minutes) at room temperature and then centrifuged in (402 X g) for (15 minutes).

The serum was stored in deep freeze and became ready to use in digestion procedures for determining Mo and in measuring the Malondialdehyde (MDA).

Determination of Molybdenum

Molybdenum determined by using Electrothermal atomic absorption spectroscopy (ETAAS), after the digestion of the sera according to the method of (Weatherby and Feruson, 2004).

Measurement of Malondialdehyde (MDA)

Lipid peroxidation was estimated by the method of by measuring the levels of MDA (Burtis and Ashwood, 1999). Free MDA , as an indicator of lipid peroxidation, was measured spectrophotometrically as thiobarbituric acid reactive substances (TBASR) after precipitating the protein with trichloroacetic acid (TCA), forming an MDA-TBA2 adduct (pink color) that absorbs strongly at (532 nm) (21).

Statistical Analysis

Data were analyzed using SPSS software (Version 19) and the values were expressed as mean and standard deviation. Pearson's correlation analysis was carried out.

All comparison were 2-tailed, and p value of <0.05 or <0.01 were considered significant.

RESULTS

As shown in the table, Molybdenum concentration increases by 120% in the sera of patients with myocardial infarction when compared to healthy controls (p<0.0005). Malondialdehyde (MDA) levels are also statistically very significantly increased in the sera of patients with myocardial infarction. There is about 260 % increase in MDA levels in the blood of patients when compared to healthy controls (p<0.0005).

Table. Molybdenum and MDA levels in the sera of healthy controls and patients with myocardial infarction

Case	Molybdenum (ppb)	MDA(mmol/L)
Healthy control n=60	2.082±1.111	1.902±0.847
Patients with myocardial infarction n= 60	6.701±1.719	6.87±2.354
P- value	P<0.0005	P<0.0005

Values reported as mean±SD; n= no. of cases.
P<0.0005 highly significant

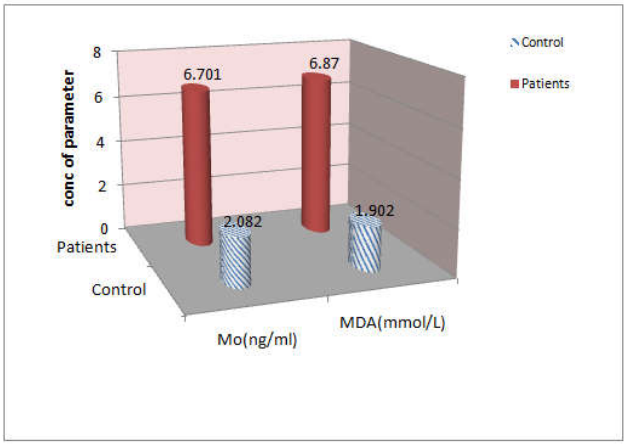

Figure 2. Molybdenum and MDA levels in the sera of healthy controls and patients with myocardial infarction

DISCUSSION

Our results showed highly significant increase in the concentration of both Molybdenum and MDA in the sera of patients with myocardial infarction (MI) compared to healthy controls. Oxidative stress or lipid peroxidation, which can represented by MDA measurement, is associated with an increased free radical burden. In this study, serum MDA levels were significantly higher in MI patients than healthy control that's agree with the study of (Haseeb, *et al*. 2013) (Haseeb Ahmad *et al*., 2013). These results suggest higher free radical metabolism in MI patients and therefore indicate some extent of tissue damage due to oxidative stress, also the prolong oxidative stress due to impaired balance between prooxidant and antioxidant mechanism may lead to lipid peroxidation and tissue damage. In addition to the elevated of MDA levels in MI patients, also we observed a highly significant increase in Molybdenum concentration compared with healthy control, which is an essential trace element, this element essential for xanthine oxidase activity, which is consideredas a marker of myocardial infarction according to a study done by (Rashmi, *et al*., 2007).

(Rashmi Raghuvanshi *et al*., 2007) they found a highly significant increase in xanthine oxidase activity in MI patients, their study indicated that myocardial ischemia has a definite correlation with xanthine oxidase activity and thus the measurement of xanthine oxidase activity may be used as a biochemical marker of myocardial infarction. Since the Molybdenum is an essential for xanthine oxidase activity, where this enzyme increase significantly in this pathogenesis, and depending in our results which shows a highly significant increase in Molybdenum concentration , so it is possible to use the Molybdenum as an indicator or biomarker for myocardial infarction.

Conclusion

Molybdenum is an essential for Xanthine Oxidase activity, this enzyme increased significantly in MI status, Our results shows a highly significant increases in Molybdenum concentration in MI sera, so Molybdenum can considered as an ideal biomarker for the diagnosis of MI

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