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SERUM LEVEL OF ISCHEMIA MODIFIED ALBUMIN IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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ABSTRACT : Acute myocardial chest pain is one of the most common diagnostic challenges in emergency medicine. Diagnosis principally focuses on identifying patients with acute coronary syndrome (ACS), who are at significant risk of adverse outcome and have the potential to benefit from inpatient care. The electrocardiography ECG provides a quick, cheap and simple way of identifying patients with ST segment changes, who are likely to benefit from admission and is therefore an essential tool for evaluating any patient with suspected ACS. However, some patients with chest pain and a normal or non-diagnostic ECG may also be at significant risk of adverse outcome. Biochemical cardiac markers, particularly troponins, can identify, which patients with a normal or non-diagnostic ECG are at higher risk. For selected patients, treadmill exercise testing can provide further prognostic information.

Chest pain is the initial symptom of many life-threatening disease processes. Pain may arise from any structure located in the thoracic cavity. Cardiac causes of chest pain usually have anginal symptoms. Noncardiac causes have a variety of chest pain characteristics. Diseases that require immediate attention and intervention are myocardial infarction/unstable angina, dissecting aortic aneurysm, pericarditis, pulmonary embolism, pneumothorax, pneumonia, and acute chest syndrome (Fallon and Roques, 1997). Early identification of patients with acute myocardial infarction is of prime importance due to the associated very high mortality. A number of biochemical tests like CKMB and Troponin-T/I have been introduced for early detection of the coronary syndrome (ACS). Ischemia modified albumin (IMA) has been introduced as a marker of myocardial ischemia. IMA appears to be developing into a new and very potent marker of cardiac ischemia (Chawla *et al*, 2006).

Key words : Acute myocardial chest pain, ischemia modified albumin, acute coronary syndrome.

INTRODUCTION

Ischemia modified albumin (IMA)

Exposure to ischemic myocardium modifies circulating albumin at its NH_2 terminus by different mechanisms due to hypoxia, acidosis, free-radical injury and energy-dependent membrane disruption (Sbarouni *et al*, 2008), the tissue-specific nature of the mechanism by which ischemia modifies albumin remains undetermined (Zapico-Muñiz *et al*, 2004). It has been shown that the binding affinity is altered in different pathological conditions. Initially, it was found that the avidity of cobalt for albumin is decreased in myocardial ischemia.Following these observations, similar alterations were found in other ischemic conditions such as stroke or mesenteric ischemia (Abboud *et al*, 2007). Gunduz *et al* (2008) revealing its lack of tissue specificity. In addition, the same phenomenon was also observed in nonischemic diseases with recognized oxidative stress such as ketoacidosis, endstage kidney disease (Ma *et al*, 2012; Mehmetoglu *et al* (2012) and liver disease (Jalan *et al*, 2009). Therefore, IMA became a marker of oxidative stress. In liver failure it has been shown that the normalized ischemia modified albumin ratio (IMA/total albumin [IMAR]) correlated with the severity of the disease and was significantly higher in nonsurvivors. In addition, IMAR correlated with other functional parameters assessed by EPR. Interestingly, different investigators reported its reversibility after a short period of time, suggesting that transient conformational changes in the structure of albumin can lead to this functional disturbance (Sinha *et al*, 2003).

IMA cardiospecificity has not been validated and needs an evidence base before routine clinical use. Some studies showed significant IMA increases 24–48 h after a marathon race, with exercise-promoted gastrointestinal and/or delayed skeletal muscle ischemia being evoked as possible causes of such increases. However, because IMA has shown rapid kinetics of increase (in minutes) and return to baseline no longer than 12 h after angioplastic procedures, long-duration skeletal muscle

ischemia (*i.e.*, occurring during marathons) does not appear to be the most appropriate model to investigate the effect of such ischemia on IMA values or the kinetics of IMA occurring during acute coronary syndromes (Zapico-Muñiz *et al*, 2004).

A number of candidate biomarkers have been proposed for the detection of cardiac ischemia; however, only Ischemia IMA has been released for clinical use. IMA is a good discriminator between ischemic and non-ischemic patients. Changes in IMA concentration have shown to occur during coronary angioplasty-induced ischemia. Clinical studies indicate that IMA appears to offer on admission an early test which can be combined with electrocardiographic findings and cardiac troponin measurements for the early exclusion of acute coronary syndrome. IMA is an independent predictor of short and long term adverse outcomes in patients with acute chest pain. However, this test is relatively new and uncertainties remain. Elevations of IMA occur in conditions other than chest pain, thus questioning its specificity. The mechanism of IMA formation and the precise entity being measured are not fully known. Nevertheless, IMA measurement remains the only current clinical biomarker which may be used for the diagnosis of patients suspected of cardiac ischemia (Gaze, 2009)

Aim of the study

To evaluate the serum level of IMA in patients with acute myocardial chest pain as a diagnostic marker in those patients.

MATERIALS AND METHODS

This is a case control study. Approval & permission to perform the study was obtained from College of Medicine, Tikrit University, Salah-Adin Health Directorate Office in the Samarra general hospital. This study was carried out on the adults males and females population their age were 40-80 years, who were attending the intensive care unit in Samarra general hospital in the period from Jun 2018 to May 2019 in Samarra city.

Patients

A total of 131 patients were fit to the inclusion criteria of this study, only 43 patients were agree to continue as a study group while the rest were escaped.

Control

Normal control group: which including 47 person, who were normal according to their physiological examination & ECG.

Inclusion criteria

1. Adult.
2. Both gender.
3. Complain of acute myocardial chest pain
4. Aged from 40-80years.

Exclusion criteria

Each patient with any of the following condition was excluded from this study.

1. Hypothyroidism and hyperthyroidism.
2. Epilepsy
3. Lactating & pregnant.
4. Chronic drug use.
5. Chronic NSAID use.
6. Chronic steroid drug use.

Blood sampling

Blood samples were obtained from the patients and control. All subjects were invited to a quiet room. Sterile disposable syringes (G21 needle) and plain plastic tubes. Blood samples of 5 mils were taken from antecubital vein puncture. The blood sample obtained from each subject was transferred into plain tube for separation of serum. Then blood in the plain tubes was then allowed to clot at room temperature (25^0C) for 1 hour .After that centrifugation was done at (3000) rpm for 10 minutes to separate the serum. The serum of each patient and control was divided and stored in to 6 small tubes and stored at -20^0C until the time of analysis to avoid thawing and refreezing. Thawing of the samples was allowed to take place at 25^0C before conducting the assay.

Method

The ELISA kit that used for IMA was manufactured SunLong Biotech Co., LTD (CHINA). The test procedure and protocol was adopted for the recommendation of the kit manufacturer, which was given in details in the kit's insert.

RESULTS

Ischemia Modified Albumin (IMA)

The Mean IMA of Patient (47.36), Std.D (10.44) & The Mean of Control (45.83), Std.D (10.13). The relationship between patients and the controls is none significant (P-Value > 0.05) as in Table 1.

Table 1 : IMA Patients and the controls.

Parameter	Group Statistics	No.	Mean	Std.D	P-Value
IMA	Patient	42	47.36	10.44	P = 0.485
	Control	47	45.83	10.13	

Significant = P-Value < 0.05
None significant = P- Value > 0.05

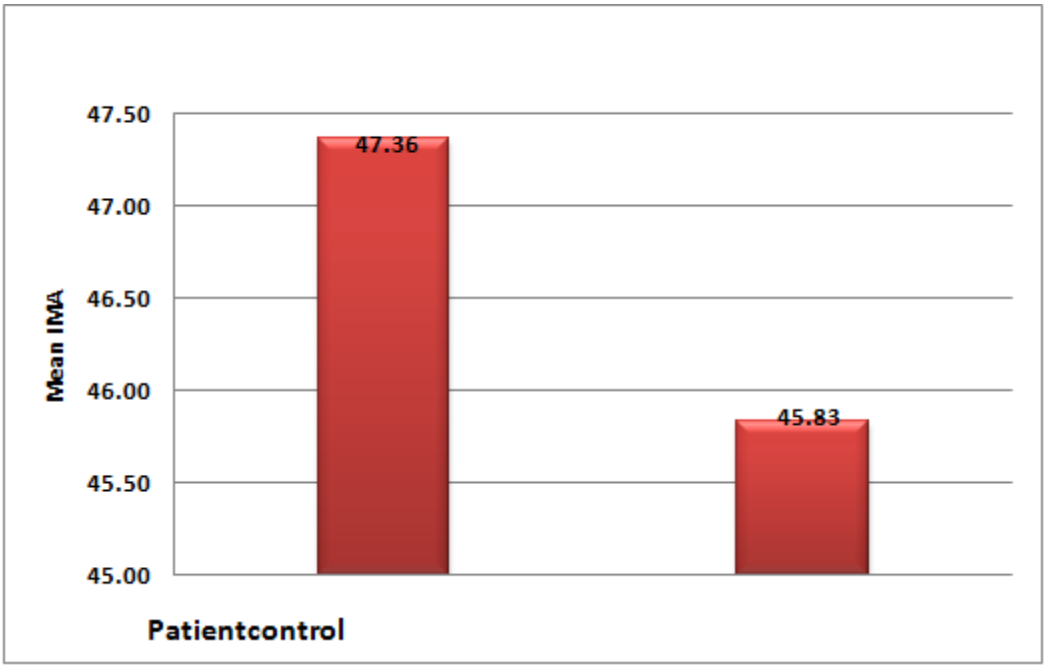

Fig. 1 : shows that there is no an influence of difference on IMA of mean patient and controls.

Comparison between Patient and Control IMA shown in Fig. 1.

DISCUSSION

Ischemia modified albumin cardio-specificity has not been validated and needs an evidence base before routine clinical use. Some studies showed significant increases in serum level of IMA, 24 – 48 h after a marathon race, with exercise-promoted gastrointestinal and/or delayed skeletal muscle ischemia being evoked as possible causes of such increases. However, because IMA has shown rapid kinetics of increase (in minutes) and return to baseline no longer than 12 h after angioplastic procedures, long-duration skeletal muscle ischemia (***i.e.***, occurring during marathons) does not appear to be the most appropriate model to investigate the effect of such ischemia on IMA values or the kinetics of IMA occurring during acute coronary syndromes (Zapico-Muñiz ***et al***, 2004).

Ischemia modified albumin is an independent predictor of short and long term adverse outcomes in patients with acute chest pain. However, this test is relatively new and uncertainties remain. Elevations of IMA occur in conditions other than chest pain, thus questioning its specificity. The mechanism of IMA formation and the precise entity being measured are not fully known. Nevertheless, IMA measurement remains the only current clinical biomarker which may be used for the diagnosis of patients suspected of cardiac ischemia (Gaze, 2009).

The result of this study showed that the serum IMA level was non-significant different between patients and the controls, this finding is in agreement with Andrew Worster ***et al***, who study a group of 189 patients, 24 had a serious cardiac outcome within 72 hours after their arrival at the emergency department, these data suggest that in patients presenting with chest pain who have not yet experienced a serious cardiac event, IMA is a poor predictor of serious cardiac outcomes in the short term. While the results is disagreement with, who studied 131 patients (mean age 58.5 years; 95 male) presenting to the emergency department with symptomssuggestive of ACS but with normal or non-diagnostic ECGs. Cardiac troponinT (cTnT) and IMA were measured within 3 h of last chest pain episode. Based on hospital diagnostic test results, patients were classified as having ACS or non-ischemic chest pain (NICP), by two independent cardiologists unaware of IMA results (Oran and Oran, 2017).

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