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Evaluation of serum Adenosine Deaminase and Uric Acid levels in patient with Acute Myocardial Infraction

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ABSTRACT

Introduction: Acute Myocardial Infarction (AMI) is one of the leading causes of mortality and morbidity globally. AMI is a term used to describe acute necrotic changes in the myocardium due to sudden deprivation of coronary blood supply (i.e., acute coronary occlusion or haemorrhage), which causes hypoxia or anoxia, forcing the cardiomyocyte metabolism to change from aerobic to anaerobic. Levels of serum Adenosine Deaminase and Uric Acid may play an important role in AMI Aim: To evaluate the levels of serum Adenosine Deaminase and Uric Acid levels among patients diagnosed with AMI. Material and Method: Fifty patients diagnosed with AMI, visiting the inpatients department (IPD) of Cardiology fulfilling the inclusion and exclusion criteria were enrolled for the study. The control group consisted of fifty age and sex matched healthy subjects. Inclusion criteria for the study was patient with history of chest pain; monitoring ECG changes- Patient with elevated ST segment and non-elevated ST segment above 25 years of age and who were interested in taking part in the study. Patients with acute or chronic renal dysfunction, history of Gout, Rheumatoid Arthritis, Hypothyroidism or Haematological Malignancy were excluded from the study. Blood samples were collected by venipuncture using standard aseptic technique and following investigation were performed, Serum Adenosine Deaminase and Uric Acid on VITROS 4600. Result: 50 patients diagnosed with AMI, and 50 age and sex matched healthy controls were included in the study. Mean levels of serum Adenosine Deaminase and Uric Acid were statistically significant among patient group when compare to healthy control. AMI frequency distribution was also done on the basis of age in the study group which showed the high incidence of the disease in between 50-70 age groups. Conclusion: Mean serum Adenosine Deaminase and serum Uric Acid levels were found to be increased in patients with AMI when compare with healthy controls.

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1. INTRODUCTION

Acute myocardial infraction (AMI) is one of the leading cause of mortality and morbidity globally[1]. AMI is a term used to describe acute necrotic changes in the myocardium due to sudden deprivation of coronary blood supply (i.e., acute coronary occlusion or hemorrhage), which causes hypoxia or anoxia, forcing the cardiomyocyte metabolism to change from aerobic to anaerobic[2].
In MI there is a decrease in coronary flow or an equivalent abrupt increase in myocardial demand for oxygen due to ischemic necrosis caused by obstruction in the coronary artery. Thrombus or atherosclerotic plaque, may obstruct the coronary flow. The atherosclerotic lesion contains macrophage and activated T-cells which secrete cytokines. This inflammatory response result in plaque rupture and thrombosis causing MI[3].

Adenosine deaminase (ADA) an enzyme involved in metabolism of purine through the salvage pathway, catalyzes the irreversible hydrolytic cleavage of (deoxy) adenosine to (deoxy) inosine and ammonia.[4] Adenosine can increase coronary artery blood flow during stress and hypoxia to balance oxygen supply and demand. The advantages of adenosine will be lost if it is rapidly metabolized by ADA. It is catalyzed to inosine which produce superoxide radicals and exaggerate ischemic injury[5].

ADA levels may be increased to compensate for the increase in adenosine levels due to MI and its product (inosine) effect in reducing the severity of inflammation. Adenosine levels are regulated by the activity of the enzyme ADA. The ADA catalyzes the deamination of adenosine to inosine contributing to the regulation of intracellular and extracellular concentrations of adenosine, and probably modulates energy metabolism. The depletion of adenosine by adenosine deaminase may lead to enhanced production of free radicals which are implicated in the pathogenesis of myocardial ischemic injury. Elevation of ADA activity in MI may be explained to be due to the damage of cells resulting in the release of intracellular components into the blood, hence elevation of its level in the blood[5].

Uric acid (urate), an organic compound of carbon, nitrogen, oxygen and hydrogen, is the final oxidation product of purine metabolism, and its elevated levels reflect increased xanthine oxidase activity. For decades it has been hypothesised that the oxidant properties of uric acid might be protective against ageing, oxidative stress and oxidative cell injury[6].

A failing heart due to acute MI may cause tissue hypoperfusion and hypoxia, which trigger xanthine oxidase activation and oxidative stress. Xanthine oxidase and oxidative stress, as reflected by uric acid levels, may form a vicious cycle that promotes severe heart failure[7,8].

MATERIAL AND METHOD:

Fifty patients diagnosed with AMI, visiting the inpatients department (IPD) of Cardiology fulfilling the inclusion and exclusion criteria were enrolled for the study. The control group consisted of fifty age and sex matched healthy subjects. Inclusion criteria for the study was patient with history of chest pain; monitoring ECG changes- Patient with elevated ST segment and non-elevated ST segment above 25 years of age and who were interested in taking part in the study. Patients with acute or chronic renal dysfunction, history of Gout, Rheumatoid Arthritis, Hypothyroidism or Haematological Malignancy were excluded from the study. Blood samples were collected by venipuncture using standard aseptic technique and following investigation were performed, Serum Adenosine Deaminase and Uric Acid on VITROS 4600.

RESULTS:

The mean value of Serum ADA of cases is 34.37 ± 7.15 U/L and of healthy controls is 12.1 ± 4.92 U/L with p-value is 0.000, mean value of Serum Uric Acid in AMI cases is 7.15 ± 1.43 mg/dl and in health controls is 4.5 ± 1.01 mg/dl with p-value is 0.000. Frequency distribution was also done on the bases of age in the study group which showed the high incidence of the disease in the age group of between 50-70 years.

Table 1: Comparison of SERUM URIC ACID between Control and Case Group

Groups	Serum Uric Acid(mg/dL)	P-value
Controls (n=50)	4.58 ± 1.01	
Case (n-50)	7.15 ± 1.43	0.000

Table 2: Comparison of Serum ADA between Control and Case Group

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Groups	Serum ADA(U/L)	P-value
Controls (n=50)	12.15 ± 4.92	0.000
Case (n-50)	34.37 ± 7.15	

Table 3: Frequency of AMI in different ages in Case group

Age (years)	Frequency
≤40	2
>40-≤50	7
>50-≤60	18
>60-≤70	17
>70	6

DISCUSSION:

AMI results in irreversible damage to the heart muscle due to a lack of oxygen. This may further lead to impairment in diastolic and systolic function and make the patient prone to arrhythmias[9]. Acute Myocardial Infarction (AMI) is one of the leading causes of increasing mortality and morbidity globally. Ischaemic heart disease (IHD), particularly acute myocardial infarction is the leading cause of death across the world accounting for 12.7 % of global mortality[10].

In the present study mean serum ADA and Uric Acid levels among AMI patients were statistically significant. Mean serum ADA and Uric Acid levels were high among patients group as compared to control group.

ADA levels may be increased to compensate for the increase in adenosine levels due to MI and its product (inosine) effect in reducing the severity of inflammation. Adenosine levels are regulated by the activity of the enzyme ADA[5]. **Priya S Hari *et al.*, 2015** reported that there was rise in serum ADA level in patients with AMI. They also suggested that ADA can serve as inflammatory marker which is poorly studied with respect to AMI[11]. **Wadood A Shatha *et al.*, 2014** concluded that ADA levels may be increased to compensate for the increase in adenosine levels due to MI and its product (inosine) effect in reducing the severity of inflammation. Also the observations of the present study provide evidence for T lymphocyte activation and proliferation in MI patients and suggest ADA as one of the markers to elucidate the pathogenesis of MI. They support the suggestion that ADA act as an inflammatory molecule in acute MI. The study suggests the inclusion of another easily measurable and cost effective marker ADA along with other biomarkers for better management and for the development of new treatment strategies[12]. **Cabarcas-Bonfante R *et al.*, 2009** suggested in their finding that serum ADA level was found to be elevated as a consequence of myocardial and/or pulmonary hypoxia in patients diagnosed with AMI[13].

Xanthine oxidase, the rate limiting enzyme for synthesis of uric acid, has been found localised in endothelial cells and smooth muscle cells of arteries. The resultant uric acid results in free radial injury to the vessel wall and contributes to development of degenerative vascular disease as well as worsening of acute thrombosis[14]. Uric acid promotes the development of atherosclerosis. The high level of uric acid causes oxidation of LDL-C and the peroxidation of lipid, formation of oxygen radicals in inflammatory reaction, increases platelet aggregation and the formation of uric acid crystals in the arterial wall which damages the tunica intima of arteries and promotes coronary thrombosis[15]. **Biswas K *et al.*, 2016** reported that serum uric acid level in association with Killip class after acute myocardial infarction is a good predictor of severity of heart failure after myocardial infarction[16].

CONCLUSION:

The serum ADA and Uric Acid were measured between AMI and controls. Our present study showed that significantly higher value of serum ADA and Uric Acid. Serum Uric Acid level after AMI is a good predictor of severity of heart failure and ADA can serve as inflammatory marker in patients with AMI. Thus, serum ADA and Uric Acid can be considered as a cheap and effective prognostic indicator in patients with AMI.

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