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SERUM IgE AND BETA 2 MICROGLOBULIN LEVELS AFTER MYOCARDIAL INFARCTION

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ABSTRACT

Serum IgE and beta 2 microglobulin levels were determined in 31 patients suffering from acute myocardial infarction and 30 patients with other forms of ischemic heart disease.

The levels of these parameters were studied on the first, third and seventh day after the onset of disease. The immunological method used for the determinations was ELISA

Patients with myocardial infarction showed an elevated level of both parameters. The peak value of IgE was observed on the seventh day ($P<0.05$) but that of beta 2 microglobulin on the third day ($P<0.05$) after the onset of myocardial infarction.

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INTRODUCTION

Recent research has uncovered considerable indirect evidence for the involvement of the immune system in different aspects of ischemic heart disease.[1] Recently IgE elevation after myocardial infarction which is followed by a return to normal after about 3 weeks has been studied[2,3,4] but the role of this elevation is controversial. Szczeklik[2] considers a protective role for IgE in the development or subsequent course of myocardial infarction while Criqui[3] and Michael[4] believe that elevated IgE levels increase the risk of cardiovascular disease. Increase of beta 2 microglobulin levels after myocardial infarction has been reported,[5] but the effect of this increase remains to be elucidated.

MATERIAL AND METHODS

We studied 61 patients with coronary heart disease who were admitted to the cardiac care unit of Imam Khomeini Hospital (Tehran). They were divided into two groups; the first consisted of 31 patients (24 men and 7 women, average age: 57.9 years) with acute myocardial infarction (MI), and the second, 30 patients (25 men and 5 women, average age: 56.1 years) with other forms of ischemic heart disease (non-MI). The diagnosis of acute myocardial infarction was based on clinical signs (chest pain), changes in the electrocardiogram and serial elevation of serum enzymes.

Stool examination for parasitic infections was performed and atopic patients were distinguished
tionnaires concerning signs and symptoms of allergic disease in the patients or their families.

Serum IgE and beta 2 microglobulin were determined on days 1,3 and 7 after the onset of disease. The immunological method used for determination of both IgE and beta 2 microglobulin was enzyme-linked immunosorbent assay; sandwich method for IgE (Enzygnost IgE monoclonal, Behring Co.) and competitive method for β 2 microglobulin (Enzygnost β 2M, Behring Co.).

The normal values of IgE and β 2M (for comparison of the results) were determined simultaneously in 30 normal subjects in the same conditions.

RESULTS

Mean and standard deviation of IgE and beta 2 microglobulin in the two groups (MI and non-MI) are

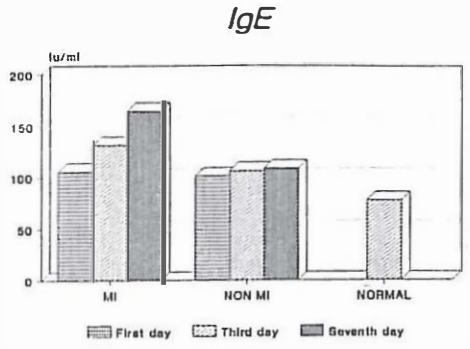

Fig. 1. Histogram of IgE levels on three different days after the onset of disease in MI, non-MI and normal individuals.

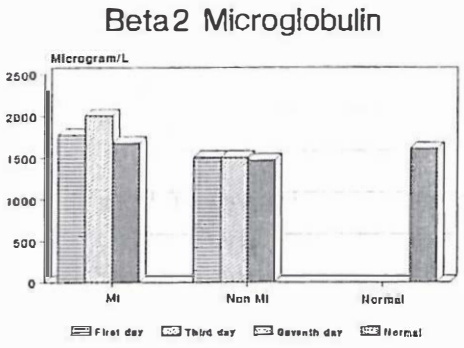

Fig. 2. Histogram of B2M on three different days after the onset of disease in MI, non-MI and normal individuals.

summarized in Tables I and II. Normal IgE and β 2M levels of 30 persons were 77.63±61 IU/mL and 1600±250μg/L, respectively. The histograms of these findings are presented in Figs. 1 and 2.

IgE levels increased significantly after myocardial infarction ($P<0.05$, paired T test) by the 7th day, while no evidence of IgE elevation was observed in group 2. Consideration of the normal IgE level (77.63±61 IU/mL) and mean IgE level of both groups on the first day reveals that they are not in the confidence interval of normal IgE levels. In other words, the mean IgE level of both groups at the onset of disease are higher than normal values.

β 2M levels increased after myocardial infarction (group 1) and by day 3 reached its peak level ($P<0.05$, paired T test). There was no increase in β 2M at different stages of disease in group 2 (non-MI).

No difference between normal and group 2 (non-MI) beta 2 microglobulin levels was observed, whereas there was a significant difference ($P<0.05$, paired T test) between group I (MI) levels and normal values.

DISCUSSION

IgE holds a unique position among immunoglobulins;

Table I. Mean and standard deviation of IgE levels (IU/mL) on three different days after the onset of MI.

Day / Patient Group	First	Third	Seventh
MI	105.4±104.7 n=31	131±124 n=31	164±148 n=29
Non-MI	101±98 n=30	106±100 n=30	108±96 n=16

Table II. Mean and standard deviation of beta 2 microglobulin (μg/L) on three different days after the onset of MI.

Day / Patient Group	First	Third	Seventh
MI	1774±522 n=31	2006±693 n=31	1670±561 n=31
Non-MI	1496±495 n=30	1500±404 n=30	1463±405 n=19

it is normally present in extremely small amounts, and it's serum concentration may increase as high as several hundred fold in specific conditions such as allergic states, parasitic infections, cancer,[6] skin diseases,[7] and generalized immunodeficiency.[8]

Recently IgE elevation after myocardial infarction has been investigated. The data in this study confirm the increase of IgE after myocardial infarction and also revealed that the mean IgE level in both groups (MI and non-MI) was higher than normal.

IgE can induce the release of mast cell mediators such as PAF, histamine, PGD_2 and leukotrienes C_4 and D_4. These mediators can induce platelet activation, increase platelet aggregation and cause coronary spasm. Thus there is a possible link between IgE and cardiovascular disease, although Szczeklik[2] believes in a protective role for it.

Elevation of IgE after myocardial infarction can be a response of the immune system to myocardial antigens. These elevations mediate an inflammatory response in the myocardium (IgE-mediated inflammation).[9] The effect of this inflammation on the prognosis of MI requires further investigation.

Beta 2 microglobulin increases only after myocardial infarction. Beta 2 microglobulin not only presents as a structural protein of HLA class I, but also acts as an

inflammatory mediator[10] in specific neutrophil granules and acts as a chemotactic factor for lymphocytes.[11] Therefore elevation of serum beta 2 microglobulin after myocardial infarction has two origins: HLA class 1 of the infarcted myocardial cells and the neutrophils anticipated in the inflammatory process due to infarction. Thus the increase of beta 2 microglobulin can be considered as an index of infarction size and intensity of the inflammatory response.

There are reports concerning the anticoagulant effect of beta 2 microglobulin.[11] These reports suggest that it may have a role in reducing post-MI complications.

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