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Original Article

Humoral Immune Responses (IgE & IgG Classes) in Acute Myocardial Infarction and Angina Pectoris

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Abstract

Objectives: The aims of the study were to determine the pattern of changes in serum IgE and IgG levels and to evaluate their probable implications in the aetiopathogenesis of acute myocardial infarction (AMI).

Patients & Methods: Thirty one AMI patients (age: 36-80 years; sex: 27 males, 4 females) were obtained from the Coronary Care Unit (CCU) at the seventh October Hospital and Jamahiriya Hospital, Benghazi, Libya. Eleven Angina Pectoris (AP) patients (age: 48-88 years; sex 6 males, 5 females) were included as a disease control. Twenty six healthy Libyans (age 38-82 years; sex: 16 males, 10 females) were taken as Normal Control (NC). Venous blood was collected as required for routine haemotalogical tests, biochemical investigations and serum total IgE and IgG analysis at the 1st day and 7th day of attack.

Results: Serum IgE level (GM±GSD, iu/ml) was significantly elevated in AMI compared to AP and NC at the 1st day (AMI1:102.0±3.2, AP1: 39.9±1.2, NC: 36.8±1.5; ANOVA: P=0.0001) as well as at the 7th day (AMI 7: 119.8±3.7, AP 7: 37.1±1.6, NC: 36.8±1.5; ANOVA: P=0.0000). No significant differences were observed for IgE levels between AP and NC and between AMI 1 and AMI 7 (P>0.05). Elevated serum IgE level in AMI was independent of risk factors such as Hypertension (HTN), Diabetes Mellitus (DM), Smoking (Sm), history Of Previous Coronary Artery Attack (H/OP CAA), complications and streptokinase therapy (P>0.1). Serum IgG level (Mean±SD, mg/dl) was significantly declined both in AMI and AP at the 1st day as well as 7th day as compared to NC (AMI1: 1033±314, AP1: 1056±320, NC: 1258±251, ANOVA: P=0.0144; AMI7: 936±383, AP 7:1042±318, NC: 1258±251, ANOVA; P=0.0002). No significant differences were observed between IgG levels in AMI1 and AP1 (P=0.833) and in AMI7 and AP7 (P=0.307). However, the decline in IgG level at the 7th day compared with 1st day was significant in AMI (P=0.014) and insignificant in AP (P=0.859). The IgG levels at the 1st and 7th day were significantly correlated in AMI patients (r=0.764, P=0.000) and also in AP patients (r=0.658, P=0.028).

Conclusion: AMI patients with high IgE levels might be protected against complications of infarction. The probable implications and mechanisms for raised serum IgE and decreased IgG levels in AMI were discussed accordingly.

Keywords: Acute myocardial infarction; Angina pectoris; Immunoglobulin E; Immunoglobulin G

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Introduction

Acute Myocardial Infarction (AMI) is one of the major causes of death worldwide and, one way or another atherosclerosis overweighs all other aetiologies in AMI [1,2]. However, probable mechanisms such as mast cell and basophils [3], Immunoglobulin E (IgE) [4,5], complements [6], neutrophil activation [7], Immunoglobulin G (IgG) [8,9] and T-lymphocytes [10,11] have been implicated in the aetiopathogenesis of AMI. Among these changes and mechanisms, reagenic antibody (IgE) has drawn special attention. AMI is associated with a consistent pattern of change in serum IgE level with an early increase followed by sustained elevation and drop to initial level by the end of third week [4,12]. These observed characteristic behavior of IgE following AMI created the thinking that it represents a specific immunological response to antigen released from the necrotic heart muscle. IgE may cause AMI and Angina Pectoris (AP) via the well defined fundamental mechanism of allergy provoked by release of histamine [12]. Many patients with AMI have biphasic response in the level of IgG during the two weeks after the onset of chest pain. This is with minimum levels between the 4th and 7th day, followed by a peak between the 7th and 11th day after infarction [9]. An interesting characteristic of the IgG subclasses is the selective occurrence of certain antibodies in one or another class [10]. As IgG_4 subclass is able to sensitize mast cell and basophil, considerable interest was focused on the possible presence of IgG_4 antibodies in allergic patients [8,11]. Auto antibodies against cardiac tissue after AMI appeared in the 2nd and 3rd weeks post AMI. Then auto antibodies slowly decreases in titre, coinciding the period of detectable cardiac muscle autoantibody with the period of elevated levels of serum IgG [9,12]. Immunological aspects including IgE were not fully elucidated in AMI and literature survey showed that no recent studies on serum IgE and IgG statuses were done or reported AMI patients from Libya. AMI patients with high IgE levels might be protected against complications of infarction. We therefore designed and undertaken studies on serum IgE and IgG

responses during 1st day of attack and at 7th day after attack in Libyan patients with AMI and also in AP patients as disease control. The results of these investigations constituted the contents of the present article.

Materials & Methods

Patients & controls

A total of 31 AMI patients were obtained from Coronary Care Unit (CCU) at seventh October Hospital, Benghazi, Libya and Jamahiriya Hospital, Benghazi, Libya during the period of September to December 1998 (Sex: 27 males, 4 females; age range: 36-80 years with mean age 62 years). The diagnosis of AMI was based on clinical assessment (medical history, ECG changes) and laboratory serial evaluation of cardiac enzymes [2,13-15]. A group of 11 admitted AP cases were included as the disease control group (sex: 6 males, 5 females; age range: 48-88 years, with mean age 61 years). Their diagnosis was based on medical history and ECG changes [2,13-15]. According to the personal questionnaire, none of the AMI and AP patients were atopic, i.e., no personal or family history of asthma, eczema, allergic rhinitis, drug allergy and/or food allergy and none had any connective tissue disorder. A group of 26 randomly selected clinically healthy Libyan adults were included in the study as Normal Control (NC) subjects (sex: 16 males, 10 females: age range: 38-82 years with mean age 58 years). All NC subjects were non smokers and had no history of allergic disorders.

Blood specimens

Venous blood specimen was collected in tube with or without anticoagulant as required for routine hematological tests and biochemical investigations and special immunological assays. Blood specimens were allowed to clot at room temperature for 30 minutes and sera were separated by centrifugation at 2500 rpm for 12-15 minutes. The separated sera were aliquoted and kept frozen at -30°C until analyzed for the parameters of interest during 1st day of attack and at 7th day after attack.

IgE Assay

Serum IgE level (iu/ml) was measured by using the commercial kit (Cat.No.14551, Chemica Omnia Export Division Srl, Italy) based on the principle of an immunoenezymatic double antibody sandwich technique as described by Hoffman [16]. The intra assay and inter assay coefficients of variation were 3.5% and 4.7% respectively.

IgG Assay

Serum IgG level was quantitatively assayed by using commercially available immuno kit of BioMerieux, France (Cat.No.500) based on the principle of radial immunodiffusion as described by Mancini et al., [17].

Statistical analysis

The statistical significance of the results was evaluated by Student's t-test, Paired t- test, one way Analysis Of Variance (ANOVA), Chi-squared (χ^2) test and correlation coefficients [18]. All computing analyses were done using Statistical Package for Social Sciences (SPSS) programme. Since total IgE values followed a log normal distribution by Kolmogorov Smirnov (K-S) test, all analyses were performed on a log transformed scale.

Results

Clinical findings

Details regarding the clinical findings of AMI and AP patients are stated in table 1 and table 2 respectively.

Serum total IgE level

The Observed ranges, geometric mean±GSD and 95% CIM values at the 1st day (AMI 1) and 7th day (AMI 7) together with their ANOVA, Students t-test and Paired t-test are presented in table 3. The observed IgE elevation in AMI patients at the first day (Geometric mean: 102 iu/ml) was significant as compared with AP patients (P=0.013) and highly significant compared with NC (P=0000), while no significant deference was observed between AP and NC (P=0.556). The elevated IgE levels at the 7th day in AMI patients (Geometric mean: 120 iu/ml) was also highly significant compared with AP patients (P=0.006) as well as NC (P=0.000). Again, no significant difference between AP patients and NC subjects was observed (P=0.958). The IgE levels at the 1st and 7th day were significantly correlated (r=0.787, P=0.000), but failed to reach statistically significant difference (P=0.272).

Serum total IgG level

Serum IgG levels showing the observed range, mean ±SD, 95% CIM, Student's t-test and paired t-test values at the 1st day, 7th day and the 1st & 7th day together with ANOVA are stated in table 3. At the 1st day, the decline in IgG levels in AMI patients (Mean=1033 mg/dl) were significant compared with NC (P=0.005). The decreased IgG level in AP patients (Mean=1056 mg/dl) was also significant as compared to NC (P=0.045). No significant difference between levels of IgG in AMI1 and AP1 patients was observed (P=0.833). At the 7th day, the more declined IgG level in AMI patients (Mean=936 mg/dl) was highly significant compared to NC (P=0.000) and the decreased level of IgG in AP patients (Mean=1042 mg/dl) was also significant compared to NC (P=0.034). The difference between decreased IgG levels in AMI 7 and AP 7 patients was not significant (P=0.307). The decline in serum IgG level at the 7th day in comparison with 1st day was statistically significant in AMI (t=2.61, P=0.014) and insignificant in AP (t=0.18, P=0.859). The levels of IgG at the 1st and 7th day were significantly correlated in AMI patients (r=0.764, P=0.000).

Risk factors & complications of AMI & their correlations with serum total IgE level

Correlations for IgE level and risk factors (hypertension, diabetes mellitus and smoking) and association of serum IgE level with history of previous coronary artery attack, complications and streptokinase therapy are stated in table 4. The P values of association of IgE level and these variables were also not significant. Serum IgE level in AMI patients was therefore independent to hypertension, diabetes mellitus, smoking, presence of history of previous ischemia and/or infarction attack, complications at presentation and streptokinase treatment.

Sl. No.	Age (Yrs)	Sex	Location of present AMI	Extension (size) of present AMI	Compli-cations of present AMI	H/OP Ischemea and/or Infarction	Associated problem	Sk tmt	DM	HTN	Sm
1.	80	M	Anteriosеptal	Extensive	-	+	-	+	-	-	+
2.	53	F	Anteriosеptal	-		+	-	-	+	+	-
3.	53	F	Anteriosеptal and Inferior	-		+	-	-	+	+	-
4.	68	M	Inferior	-	-	+	-	+	-	+	+
5.	56	M	Anteroposterior	-	Sinus Bradycardia	-	COPD	+	-	-	+
6.	72	F	Inferiolateral	-	-	+	-	+	-	+	-
7.	70	M	Anterior	Extensive	AEBs	-	MR, AR	+	+	-	-
8.	48	M	Anteriosеptal	Extensive	-	+	-	+	-	+	+
9.	70	M	Inferior	-	Mild LVF	-	-	+	-	+	-
10.	75	M	Inferiolateral	-	Sinus Bradycardia	-	-	-	-	+	+
11.	50	M	Inferiolateral	-		-	-	+	-	-	-
12.	66	M	Anteriosеptal	-	-	-	-	-	+	-	+
13.	64	F	True posterior	-	LVF	-	-	-	-	-	-
14.	75	M	Anteriosеptal	-	LVF	-	-	-	-	-	+
15.	58	M	Anteriosеptal	-	LBBB, LAD	-	-	+	-	+	+
16.	65	M	Inferior	-	+	-	-	-	-	-	+
17.	64	M	Inferior	-	-	-	-	-	-	+	-
18.	50	M	Anterior	Extension	-	-	-	+	-	+	+
19.	50	M	Anteriosеptal	-	-	+	-	-	+	+	+
20.	60	M	Anteriosеptal	Extension	-	-	-	-	+	-	-
21.	60	M	Anteriosеptal	Extension	-	-	-	-	+	-	-
22.	36	M	Anteriosеptal	-	RBBB, PAT	-	COPD	-	+	-	+
23.	65	M	Inferiolateral	-	LVF	+	-	-	-	+	+
24.	65	M	Inferior	-	Sinus Bradycardia, LAD, 1st degree HB	-	COPD	+	+	+	+
25.	64	M	Anterior	Extensive	VT, VF	+		-	-	+	+
26.	50	M	Anteriolateral and inferior	-	Sinus Bradycardia	+	-	-	-	+	-
27.	62	M	Anteriosеptal and apical	Extensive	LVF	-	-	-	+	+	-
28.	48	M	Anteriosеptal	-	LAD, RBBB	-	-	+	-	-	+
29.	70	M	Anterior	Extensive	-	+	COPD	+	-	-	+
30.	74	M	Inferior	-	-	+	-	-	-	-	+
31.	55	F	Anteriosеptal	-	AEB	-	-	-	+	-	-

Table 1: Clinical data of AMI patients.

M: Male; **F:** Female; **AMI:** Acute Myocardial Infarction; **H/OP:** History Of Previous; **Sk tmt:** streptokinase treatment; **DM:** Diabetes Mellitus; **HTN:** Hypertensions; **Sm:** Smoking; **LVF:** Left Ventricular Failure; **COPD:** Chronic Obstructive Pulmonary Disease; **MR:** Mitral Regurgitation; **AR:** Aortic Regurgitation; **RHD:** Rheumatic Heart Disease; **AEBs:** Atrial Ectopic Beats; **LBBB:** Left Bundle Branch Block ; **1st degree HB:** 1st degree Heart Block; **LAD:** Left Axis Deviation; **RBBB:** Right Bundle Branch Block; **PAT:** Paroxysmal Atrial Tachycardia; **VT:** Ventricular Tachycardia; **VF:** Ventricular Fibrillation

Discussion

The present study showed that AMI was associated with elevation of serum IgE during first week of infarction. In our AMI patients the raised IgE levels at the 1st day and at the 7th day were not significantly different which was consistent with previous studies [4,19]. In some patients the elevated IgE levels were reached within few hours post cardiac attack and continued at approximate levels on the 7th day. In four patients there were a dramatic variability between 1st and 7th day IgE levels; the higher values were found in three of them at the 7th day and in one case at the 1st day. In two AMI patients no IgE rise were achieved at the end of 1st day, whereas at the 7th day a considerable elevation had been found. In one AMI patient who showed elevated IgE levels at the 1st and 7th day, 3 weeks later presented as AP case with backtracked normal IgE. These observations together with those of Szczeklik et al., on post AMI IgE pattern revealed that the raised serum IgE was post AMI response. This was unlikely a pre infarction basal level as suggested by Komraz et al., based on their findings of consistent IgE elevation at the first, third and seventh day post infarction [4,19]. Similar patterns for serum IgE in acute coronary syndromes were reported also by other investigators [3,20,21].

In our study, serum IgE level was independent to hypertension, diabetes mellitus and smoking and no associations were found between IgE and history of previous myocardial ischemia and/or infarction

SI. No.	Age (Yrs)	Sex	Complication of Present Angina	H/OP MI	DM	HTN	Sm
1	49	F	-	+	+	-	-
2	54	M	-	-	+	+	-
3	48	M	-	+	-	-	Ex smoker
4	50	F	LBBB	+	+	+	-
5	50	F	Sinus Bradycardia, IVF, AEBs	-		+	-
6	70	M	LVF	-		+	-
7	88	M	SVT	+	+	+	-
9	80	F	Mild LVF	+		+	-
10	67	M	LVF	+		+	-
11	62	M	-	+	+	-	-

Table 2: Clinical data of AP patients.

H/OP MI: History of previous Myocardial Infarction; **DM:** Diabetes Mellitus; **HTN:** Hypertension; **Sm:** Smoking; **LBBB:** Left Bundle Branch Block; **LVF:** Left Ventricular Failure; **SVT:** Supraventricular Tachycardia; **LAD:** Left Axis Deviation; **IVF:** Intraventricular Fibrillation; **AEBs:** Atrial Ectopic Beats

Statistical Analysis	IgE (iu/ml)				IgG (mg/dl)			
	AMI (n=31)	AP (n=11)	NC (n=26)	ANOVA: F(P)	AMI (n=31)	AP (n=11)	NC (n=26)	ANOVA: F(P)
1st day analysis								
Obs range	23-1075	25-55	15-80		710 -2010	630-1056	810- 000	
GM±GSD	102±3.2	39.9±1.2	36.8±1.5	11.577	-	-	-	
				0				4.527
Mean±SD	-	-	-		1033±314	1056±320	1258±251	-0.014
95% CIM	65.9-156.3	34.5-46.3	31.1-43.2		918-1148	841- 1272	1157- 1359	
7th day analysis								
Obs range	20-1071	20-85	15-80		630 -1800	600-1780	810-2000	
GM±GSD	120 ± 3.7	37.1± 1.6	36.8± 1.5	13.203	-	-	-	
Mean±SD	-	-	-	0	936±383	1042±318	1258±251	9.668
95% CIM	74.3- 193.2	27.5-50.2	31.1-43.2		832-1040	828-1255	1157-1359	0
Student's t-test					**Student's t-test**			
Groups	**t value**	**df**	**P**		**t value**	**df**	**P**	
1st day analysis								
AMI vs NC	4.17	55	0		- 2.95	55	0.005	
AMI vs AP	2.58	40	0.013		- 0.21	40	0.833	
AP vs NC	0.59	35	0.556		2.06	35	0.045	
7th day analysis								
AMI vs NC	4.41	55	0		-4.51	55	0	
AMI vs AP	2.91	40	0.006		-1.03	40	0.307	
AP vs NC	0.058	35	0.958		2.21	35	0.034	

Table 3: Serum levels of IgE (iu/ml) and IgG (mg/dl) in AMI and AP patients at the 1st and 7th day and control subjects and their statistical analysis.

AMI: Acute Myocardial Infarction Patients; **AP:** Angina Pectoris Patients; **NC:** Healthy Control Subjects; **SD:** Standard Deviation; **GM:** Geometric Mean; **GSD:** Geometric Standard Deviation; **95% CMI:** 95% Confidence Interval For Mean/Geometric Mean; **DF:** Degree of Freedom; **ANOVA:** One-Way Analysis Variance; **P:** Probability; **P≤0.05:** Significant; **P>0.05:** Not significant

attack and between IgE and AMI complications at presentation week (Table 4). These were in contrary to the report that IgE was associated with hypertension, diabetes mellitus and smoking [19]. We could not find any association between serum total IgE level and streptokinase therapy (Table 4) and no anaphylactic reaction had been registered among our streptokinase treated AMI patients [22]. Recently, IgE response was described in patients underwent coronary bypass surgery that did or did not develop perioperative AMI [23]. The behavior of IgE was characteristic: it started to rise after surgical operation, continued to increase to reach a peak by fifth day and then gradually declined.

The present study demonstrated that AMI and AP patients had decreased serum IgG levels at first day post coronary event. One week later serum IgG became markedly depressed in AMI cases, while in AP patients it was sustained approximately to the initial values. In a previous study, a biphasic pattern in the level of serum IgG after AMI was reported, with minimum levels at the end of first week followed by an elevation to normal values during the second week [9]. The similar

Variable	Distribution of AMI Patients			Chi-squared (χ^a) Tests
	Group A	Group B	Total	
HTN	11	8	19	χ^2=0.197, df= 1 P> 0.10
No HTN	6	6	12	
	Total 17	14	31	
DM	7	12	19	χ^2=0.0916, df= 1, P=0.5
No DM	5	7	12	
	Total 12	19	31	
Sm	9	4	13	χ^2=1.0466, df= 2, P> 0.10
N Sm	6	6	12	
Ex Sm	4	2	6	
	Total 19	12	31	
H/OP CAA	8	4	12	χ^2=0.206, df- 1, P>0.10
No H/OP CAA	11	8	19	
	Total 19	12	31	
Comp	10	9	19	χ^2=0.0916, df- 1, P= 0.5
No Comp	9	3	12	
	Total 19	12	31	
Sk tmt	10	3	13	χ^2=1.0466, df- 2, P> 0.10
	9	9	18	
No Sk tmt	Total 19	12	31	

Table 4: Chi-square (χ^2) test for distribution of AMI patients serum IgE level at the 1st day and conventional risk factors and complications and streptokinase treatment in 31 AMI patients.

Group A: AMI patients with high IgE level (i.e., ≥ 80.0 IU/ml); **Group B:** AMI patients with normal IgE level (i.e..,<80.0 IU/ml); **HTN:** Hypertension; **DM:** Diabetes Mellitus; **Sm:** Smoking; **Ex sm:** Ex-smoking; **N Sm:** Non smoking; **H/OP CAA:** History Of Previous Coronary Artery Attack; **Comp:** Complications; **Sk tmt:** Streptokinase treatment; **P≤ 0.05:** Significant; **p>0.05:** Not significant

IgG pattern had been demonstrated also after major surgical operations [24]. IgG diffusion into the injured myocardium could be among the factors responsible for decreased serum IgG levels post AMI; it was observed that both albumin and IgG diffuse and become firmly bound to cardiac muscle fibres within one hour after ligation of coronary artery in dog [25]. This IgG diffusion is most likely a specific immune response, as had been previously reported a significant immune response against actin and myosin via IgG and IgM classes in patients suffering from various cardiovascular diseases including AMI [26]. As well, a significant correlation between precardiac injury and peak post cardiac antimyosin and anti actin autoantibody levels were demonstrated suggesting that preinjury sensitization to these auto antigens plays an important role in evoking post cardiac injury immune response [26].

Further, recent result showed that even unprimed B cells from spleen, liver or bone marrow could be induced to high levels of IgE production *in vitro*, [27,28]. It was documented that anti IL-6 antibody strongly inhibits IgE production by hydrocortisone and IL-4 [29]. Endogenous IL-6 level increased dramatically in patients who underwent major surgery, peaking at 24 hours post surgical trauma and returning to initial level on ninth post operative day. This was suggestive that IL-6 has the critical role for induction of IgE by hydrocortisone and IL-4 [23]. Accordingly it could be hypothesized that the stimulation of hypothalamus pituitary adrenal axis and/or release of glucocorticoids which interact with newly synthesized IL-6 could explain the characteristic IgE response to tissue injury [23]. The potential significance of still another role of IgE, i.e. IgE involvement in post inflammatory mechanism, needs careful evaluation. Influence of corticosteroid on serum IgG was reported earlier [30]. The decrease in serum total IgG by corticosteroid might by mainly due to inhibition of the synthesis of this immunoglobulin rather than promoting antibody destruction [30]. The proposed influence of corticosteroid on serum levels of IgE and IgG in ischemic heart disease may be concentration dependent. However, AP patients showed a sustained decline in IgG at the 1st week of ischemic attack, while no IgE alteration was observed and in addition, around 40% of AMI cases also showed no change in IgE.

In conclusion, the elevated serum total IgE level in AMI patients was independent to hypertension, diabetes mellitus and smoking and complications at presentation. The IgE increment in AMI patients was possibly post AMI polychonal upregulation and not allergen specific. Thus steps of mast cell degranulation via allergen specific IgE were not fulfilled and hence anaphylaxis via IgE as the aetiopathogenesis of AMI was unlikely. We observed that AMI and AP were associated with a depression in serum IgG levels during the 1st week of coronary artery attack. Diffusion of IgG in the injured myocardium may be the major factor responsible for decreased serum level of it. However, the role of IgE should be more carefully considered and analyzed. AMI patients with high IgE levels might be protected against complications of infarction. Other immunological responses such as IgG, complements, cytokines and cellular immunity and particularly, studies on IgG subclasses are warranted to know their role in the aetiopathogenesis of AMI.

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