International Journal of Enhanced Research in Science, Technology & Engineering
ISSN: 2319-7463, Vol. 7 Issue 10, October -2018, Impact Factor: 4.059

Relation of allergic diseases and morbidity rate of acute myocardial infarction: A case-controlled study

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ABSTRACT

The aims of the study were to explore the probable implications of total IgE& allergic disorders in the aetiopathogenesis of acute myocardial infarction (AMI). It was conducted as a case-controlled study on 100 patients with acutemyocardialinfarction (AMI) admitted to the cardiac care unit at Ibn-Sina Teaching Hospital in Mosul city. Another 100 age and genders matched healthy subjects of attendance to the hospital for other reason were recruited as control group. The diagnosis of allergic diseases was based on clinical findings and estimation of total serum IgE level. The mean (SE) of total IgE in AMI patients were 89.50 (5.89) IU/ml and 60.62 (6.82) IU/ml in control group. The difference was statistically significant (p = 0.001). Total IgE level was <20 IU/ml (an allergic disease is not probable) in 21 % of AMI patients, between 20-100 IU/ml (allergic disease is questionable) in 36% of patients, and >100 IU/ml (allergic disease is very probable) in 43% of them. Result shows that 28% of AMI patients have different types of allergic diseases while 8% of control group report these disorders and the difference was statistically significant (p = 0.01). In both study groups, bronchial asthma was the most common allergic disease, then allergic dermatitis and rhinitis. In conclusion, the observed higher initial serum IgE concentration in patients with ischemic heart diseases may serve as evidence contribution to pathogenesis of myocardial ischemia.

HOW TO CITE THIS ARTICLE

Salah Hamad Izba, Haitham B. Fathi, Samet Alyas Qasim, "Relation of allergic diseases and morbidity rate of acute myocardial infarction: A case-controlled study", International Journal of Enhanced Research in Science, Technology & Engineering, ISSN: 2319-7463, Vol. 7 Issue 10, October-2018.

INTRODUCTION

Coronary artery disease (CAD) and its end result, acute myocardial infarction (AMI) continue to be a significant cause of mortality and morbidity worldwide[1]. The role of immunologic mechanisms in the development of atherosclerosis and its thrombotic complications (collectively named "atherothrombosis") has attracted considerable interest[2]. Recently, many immune factors both cellular and humoral factors have been implicated in the aetiopathogenesis of AMI. Mast cells have been implicated in the pathogenesis of coronary heart disease[3]. They can be activated by immunoglobulin (Ig) E-mediated mechanisms to release powerful mediators affecting local blood flow. Immunoglobulin E (IgE) has drawn special attention as it showa consistent pattern of changesduring acute infarction[4]. IgE is also a mediator of allergy, can affect platelets and arterial smooth muscle leading to both platelet activation and arterial spasm[5].Most of these findings were a product of cross-sectional studies that doesn't clarify the temporal relation of elevated IgE and incident AMI. This raise inquiry (is the elevation of serum total IgE is a cause or result of cardiac damage process? Furthermore, what isthe role of history of various allergic histories on predicting risk of myocardial infarction?.We need to support these cross-sectional evidences by more powerful longitudinal studies to clarify this association. This knowledge gape prompts us to conduct this more powerful longitudinal design (retrospective case-control study) aiming to:

1. To explore the relation of different allergic diatheses and acute myocardial infarction.
2. To estimate the difference in total IgEbetween patients with acute MI and healthy subjects.
3. To compare the frequencies of allergic diseases between patients with acute MI and healthy subjects.

PATIENTS AND METHODS

The study was conducted as a case-controlled study. Ethical approval was obtained from regional research of committee of Ninevah directorate of health. It was carried out at Cardiac Care Unite (CCU) at Ibn-Sina teaching hospital during a period extends from28 to August 31, 2007. A random sample of 100 patients with acute myocardial infarction range in age from 30-70 year was asked to participate in the study as case group. A patient with a history of worm infestation was excluded from the study. Another sample of 100 age and gender matched healthy subjects who attend to the hospital for other reason was asked to participate in the study as control group. All participants were interviewed by researchers using structured interview. The data collected include presence and type of allergic diseases. A 5ml of blood was taking from each participant to estimate total serum IgE level. The quantitative direct determination of serum total lgE was assessed by the use of ELISA (ELX 800 universal microplate reader I USA and its commercial kit. The total IgE was subdivided into: allergic disease is not probable (IgE<20 IU/ml); allergic disease is questionable (IgE 20-100 IUml), and allergic disease is very probable (IgE> 100 IU/ml).

All the data have been processed by the use of statistical package SPSS Ver 18 (SPSS Inc., Chicago, Ill). Descriptive statistical methods were used to summarize and tabulate data. For proportional data (presence of allergic disease and class of total IgE), the significance of their association with AMI were assessed by the use of $\chi 2$ test. For continuous variables, the significances of differences in mean level between the cases and controls were estimated by means of two independent samples Student *t*-test. Because of the strongly skewed IgE distribution, logarithmic transformations of the values (log IgE) were used in the analyses, and the results are given as medians. P-value of <0.05 was considered statistical significant.

RESULT

The age of AMI patients range from 33 to 79 years with mean (SD) of 62.00 (12.35) years. Males form 74% of cases while females form26% of them with male to female of 3:1. The male patients were on average 6 years younger than females (mean age of males were 60.27 years while in in females were 66.92 years), but the difference was statistically not significant (p = 0.2). The age range of control group were from 30 to 80 years with mean (SD) of 59.8 (13.11) years. They are consisting of 70 males & 30 females. The differences between patients and control group regarding age & gender were statistically not significant (p-value equal 0.4, and 0.5 respectively).

The mean (SE) of total IgE in AMI patients were 89.50 (5.89) IU/ml while mean (SE) of total IgE in control healthy group were 60.62 (6.82) IU/ml and the difference was statistically significant (p = 0.001).

Subdividing total IgE into classes of allergyin AMI patients group revealed the following: IgE< 20 IU/ml (an allergic disease is not probable) was found in 21% of patients; thirty-six percent of patients have total IgE between 20 and 100 IU/ml (allergic disease is questionable); and the remaining 43% of patients have a total IgE exceed 100 IU/ml (allergic disease is very probable).Further classification of total IgE according to gender of patients is shown in (Table 1). All differences of classes of allergy between both genders of AMI patients were statistically not significant.

Table 1: Comparison of total IgE classes according to gender of acute myocardial infarction (AMI) patients

Total IgE classes*	Gender of patients with AMI			P-value
	Males N=74 No. (%)	Females N=26 No. (%)	Total N=100 No. (%)	
<20 IU/ml	13 (17.6)	8 (30.8)	21 (21%)	0.1
20-100 IU/ml	29 (39.2)	7 (26.9)	36 (36%)	0.3
>100 IU/ml	32 (43.2)	11 (42.3%)	43 (43%)	0.9

*Total IgE<20 IU/ml (allergic disease is not probable); Total IgE 20-100 IUml (allergic disease is questionable); IgE> 100 IU/ml (allergic disease is very probable)

Comparison of classes of total IgE between AMI patients and healthy control groups is shown in (Table 2). The results revealed the following: class of allergy is not probable (<20 IU/ml) is significantly more than two times higher in healthy

group(p = 0.0001); no significant difference of class of allergy is questionable (total IgE 20-100 IU/ml) between both groups (p = 0.1); lastly, class allergy is very probable (total IgE>100 IU/ml) is twice more common in AMI patients group than in healthy group (P = 0.002).

Table 2: Comparison of total IgE classes between acute myocardial infarction (AMI) patients and healthy control groups

Total IgE classes	AMI patients No. (%)	Healthy control No. (%)	P-value
<20 IU/ml	21 (21%)	55 (55%)	0.0001
20-100 IU/ml	36 (36%)	26 (26%)	0.1
>100 IU/ml	43 (43%)	19 (19%)	0.002

*Total IgE<20 IU/ml (allergic disease is not probable); Total IgE 20-100 IUml (allergic disease is questionable); IgE> 100 IU/ml (allergic disease is very probable)

(Table 3) revealed that twenty-eight AMI patients reported different types of allergic diseases while 8 participant of control group report to have allergic disorders and the difference was statistically significant (p = 0.01). In both study groups, bronchial asthma was the most common allergic disease, then allergic dermatitis and rhinitis.

Table 3: Comparison of frequency and types of allergy according to gender of acute myocardial infarction (AMI) patients

Total IgE classes*	AMI patients No. (%)	Healthy control No. (%)	P-value
Bronchial asthma	10 (10%)	2 (2%)	
Allergic dermatitis	6 (6%)	3 (3%)	
Allergic rhinitis	4 (6%)	2 (2%)	0.01
Allergic conjunctivitis	3 (2%)	-	
Drug allergy	3 (3%)	1 (1%)	
Foot allergy	2 (1%)	-	
Total	28 (28%)	8 (8%)	

DISCUSSION

The analysis of this series of patients revealed that (AMI) was more common in patients older than 50 years and males. The male to female was 3:1. This is in agreement with the conclusion of recent study of Moshkietal in which out of 200 patients, 33% of them were females and 67% were males[6]. A similar trend was reported by Yang etal, large 10-year nationwide-based analysis of Taipei health data base[7]. The male patients were on average 6 years younger than females (mean age of males were 60.27 years while in in females were 66.92 years). This is consistent with result reported by Roger[8].

The mean (SE) of total IgE in AMI patients were 89.50 (5.89) IU/ml while mean (SE) of total IgE in control healthy group were 60.62 (6.82) IU/ml and the difference was statistically significant (p = 0.001). This result provides new support to previous epidemiological studies that show that immunoglobulin E (IgE) levels were higher in subjects with acute coronary events[9-12]. Langer etalconfirmed the association between IgE and coronary heart disease (CHD) by using cross-sectional design and considered as a predictor for future nonfatal myocardial infarction[9]. In the prospective study of Erdogan et al., found high initial IgE level not only in patients with (AMI), but even in unstable angina pectoris (UAP), and stable angina pectoris (SAP).

The IgE level was significantly higher in CHD patients than its level in healthy controls group (p = 0.002)[10].Kounis measured inflammatory mediators like histamine in blood or urine in both allergic episodes and acute coronary syndromes. He found high concentration of this metabolite in both condition[11]. Based on these finding, he suggests a common pathway between them and called it allergic angina and allergic myocardial infarction (Kounis syndrome). Furthermore, he recommends using drugs to stabilize mast cell membrane as novel therapeutic modalities to prevent acute coronary and cerebrovascular events. Recently, Unal et al investigate the relationship between the level of IgE and endothelial functions

of the coronary arteries measured by coronary flow reserve (CFR) using transthoracic Doppler echocardiography[12]. They CFR were significantly lower in the patients with high IgE levels. They conclude that it seems to support the role of IgE in the vascular pathology of atherosclerosis.

From the current study and above mentioned studies we can propose the following cascade of mechanisms that explain the consequence of elevation of IgEon development of coronary heart disease. At the beginning, mast cell stimulates by high IgE lead to formation of cholesterol-containing foam cells in the arterial wall. Degranulation of mast cell led to release histamine which causes vasoconstriction of coronary artery. Lastly, inflammation triggered by IgEleads to plaque destabilization, rupture, and ultimately coronary thrombosis.

Bronchial asthma was the most commonly reported allergic disease in patients with AMI. Ten percent of the current series of AMI patients have history of bronchial asthma and in 6% of the control group. This close to the figure reported by Bang etal, in their large population-based study[13]. They found of the 543 MI cases, (15%) had a history of asthma prior to index date of MI whereas (10%) of control group had such a history. They conclude that active asthma is an unrecognized risk factor for MI. This risk can be partiallyexplained as complication of Beta-adrenoceptor agonists (β -agonists), which is widely used for obstructive lung disease [14]. Bangetal recalculate odd ration (OR) after adjusting for treatment yield adjusted odds ratio: 1.68; 95% CI: 1.06–2.66. This odd ratio is lower than OR reported in the current study (5.0). Painless is one of characteristic of AMI in patients with concurrent bronchial asthma, this Chappell postulation [15]. He found that of a total of 35 instances of painless myocardial infarction encountered in a District General Hospital over a 15-year period, ten occurred in seven patients admitted with acute exacerbations of asthma.

In the current study, atopic dermatitis (AD) was the second most common allergic disease in patients with AMI. Six percent of them have a concomitant AD, while 3% of control group has a history of AD. This finding is in agreement with recent large population-based study with long follow-up[16]. which reported an increased prevalence of asymptomatic coronary artery disease among individuals with AD. The cumulative incidence of MI was 0.6% for patients with AD and 0.4% for their matched controls.

The allergic rhinitis was reported by 4% of patients while 2% of control group declare complaining of such a disease. This result was inconsistent with finding of Cransetal who reported that patients with physician-diagnosed allergic rhinitis (N = 110, 207 in matched cohort) had significantly lower risk for myocardial infarction (HR, 0.63; 95% CI, 0.59-0.67; P < .001)[17].

In conclusion, the observed higher initial serum IgE concentration in patients with ischemic heart diseases may serve as evidence contribution to pathogenesis of myocardial ischemia.

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