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Original Article

Study of atherosclerosis in bronchial asthma patients

Pulmonology

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ABSTRACT

Background: Bronchial asthma is commonly associated with systemic complications that contribute to its burden including cardiovascular diseases, in which atherosclerosis is the leading histopathologic ground.

Objective: To study the coexistence of atherosclerosis in bronchial asthma patients and to identify clinical characteristics and risk factors related to it in asthmatic patients.

Methodology: A case-control study that included 100 bronchial asthma patients and 100 non-asthmatic apparently healthy subjects were performed. All participants were subjected to the measurement of spirometric-indices, blood eosinophils, lipid profile, serum total IgE, and carotid duplex ultrasonography.

Results: There was a significant increase in eosinophil count, total IgE, cholesterol, low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) with a significant decrease in high-density lipoprotein (HDL) in the asthma group than in controls. Dyslipidemia was significantly more common in the asthma group than in controls (44% vs. 10% respectively, $p< 0.05$). There was a significant increase of both common carotid arteries intima-media thickness (CCAs IMT) with a significant higher frequency of atherosclerosis in asthmatic patients than in controls (26% vs. 6% respectively $p< 0.05$). In the asthma group, both CCAs IMT were positively correlated with age, BMI, asthma duration, total cholesterol (TC), triglycerides (TG), LDL, eosinophils count, and total IgE, while they were negatively correlated with the age of asthma onset, spirometric-indices, and HDL. The significant risk factors of atherosclerosis in asthmatic patients were higher LDL (OR 7.160), family history of allergic diseases (OR 5.512), poor asthma control (OR 4.318), higher eosinophils (OR 4.110), higher IgE (OR 4.066), younger age of asthma onset (OR 2.768), higher BMI (OR 2.418), lower spirometric-indices (OR >2), longer asthma duration (OR 1.704), and higher patient age (OR 1.212). On the contrary, HDL demonstrates protective effect (OR 0.071).

Conclusion: This study declares an association between asthma and atherosclerosis, in particular uncontrolled severe persistent asthma. The most impressive risk factors of atherosclerosis in asthmatics include dyslipidemia, lower spirometric-indices, higher eosinophils and total IgE levels.

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Keywords: Dyslipidemia; carotid intima-media thickness; eosinophils; total IgE.

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INTRODUCTIONS

Bronchial asthma is a chronic airway inflammatory disease characterized by severe bronchial inflammation and hyper-responsiveness. Atherosclerosis is a chronic arterial inflammatory disease. Asthma is known to have a potential systemic impact that may affect extrapulmonary tissues including atherosclerotic cardiovascular diseases (ASCVD) [1]. ASCVD is considered the "silent killer", due to the subclinical, slow but continuous progression of the disease during people's lifetime, starting asymptomatic till becoming clinically significant after the occurrence of a CVD event (i.e., transient ischemic attacks) [2]. Atherosclerosis may start during childhood and remain dormant and asymptomatic for several years before progressing in middle age and older [3].

Asthma and CVD share several risk factors including obesity, physical inactivity, smoking, psychological stress, and environmental exposure [4]. Several proposed mechanisms can explain asthma's association with CVD, e.g. shared immunopathogenesis pathways; as asthma and atherosclerosis are characterized by airway and focal intimal

accumulation of inflammatory cells respectively [5]. A spillover mechanism of chronic airway inflammation to the systemic circulation has been suggested through a 5-lipo-oxygenase enzymatic pathway that mediates airway inflammation in asthma and is also expressed in atheromatous plaques [6]. Dyslipidemia is considered one of the pro-inflammatory risk factors of asthma that can trigger the release of inflammatory mediators from the endothelium and adepokines from adipose tissue which can stimulate the development of atherosclerotic plaques [7]. Endothelial dysfunction following oxidative stress may also promote coagulation activity [6]. Medications for asthma may contribute to the incidence of CVD [8]. Therefore, the early detection of increased IMT represents the preliminary phase of atherogenesis and the basis under which lipids build up with the formation of atherosclerotic plaque is possible [3]. This study aims to study the coexistence of atherosclerosis in bronchial asthma patients and to identify clinical characteristics and risk factors related to it in asthmatic patients.

SUBJECTS AND METHODS:

This case-control study was conducted at chest diseases department, Al-Zahraa University Hospital, Al- Azhar University, Cairo, Egypt during the period from July 2022 to December 2023. It was conducted after approval by the ethical committee of faculty of medicine for girls, Al-Azhar University, Cairo, Egypt. An informed consent was gotten from all participants before included into the study.

Sample size: According to the annual flow of asthma and its prevalence in Egypt (6.8%) [9], the sample size was calculated by Epi info, Atlanta, Georgia, U.S, and it was found that the statistically representative sample was 96 (margin of error 5% and confidence level (CI) 95%). Accordingly, this study was carried out on 100 asthmatic patients and 100 non-asthmatic healthy subjects as control group:

1. **Bronchial asthma group:** included 100 stable adult asthmatic patients selected from patients attending chest diseases outpatient clinic for regular follow-up. They were diagnosed according to GINA 2022. The asthma severity was determined according to the National Asthma Education and Prevention Program Expert Panel Report 3 [10]. The level of asthma control was assessed based on GINA (2022) classification [11].
2. **Control group:** included 100 apparently healthy adults, age and sex-matched, non-obese, and non-smoker subjects selected from employees at Al-Zahraa University Hospital who have no family history of allergic diseases.

Healthy subjects and asthmatic patients younger than 18 or older than 45 years, current smokers, alcohol abusers, and obese [body mass index (BMI) ≥ 30] were ruled out from the study. Patients with other chronic lung diseases, ischemic heart diseases, vasculitis, autoimmune diseases, immunodeficiency diseases, diabetes mellitus, and hypertension were excluded from the study. Additionally, patients treated with exogenous hormones, systemic corticosteroids during the last month, lipid-lowering agents, and anti-IgE drugs were not included in the study.

Data regarding age and sex were recorded for all study participants, while asthma-related data such as age of asthma onset, asthma duration, asthma severity [10], asthma control level [11], associated other allergic diseases, family history of allergic diseases, and anti-asthma medications include inhaled corticosteroids (ICS), short-acting beta-agonist (SABA), long-acting beta-agonist (LABA) and theophylline were reported for asthma patients only.

Both asthma patients and controls were subjected to all of the following:
The BMI was calculated as weight (kg) divided by height (m ^{2}) [(weight (kg)/ height (m^2)] and spirometry was done using (MEDISOFT- HYPERAIR compact + flow meter pulmonary function testing - Belgium). The following indices were recorded: FEV_1\FVC ratio, FVC %. FEV_1% and $FEF_{25-75\%}$.

Blood eosinophil count (cells/ cmm) was measured, and peripheral eosinophilia defined as an eosinophils count ≥150/cmm [12]. Lipid profile was measured, and dyslipidemia was identified as total cholesterol (TC) ≥240 mg/dl, triglyceride (TG) ≥200 mg/dl, high-density lipoprotein (HDL) <40 mg/dl [13], and low-density lipoprotein (LDL) (≥160 mg/dl) [14]. Serum total IgE was measured, and the studied participants were categorized as either having normal serum total IgE (< 97 IU/ml) or elevated total IgE (> 97 IU/ml).

Ultrasonographic examination of both common carotid arteries (CCAs) was done using Sonoscape SSI6000 (Medical Systems, Shenzhen, China) and its 5-15 MHz vascular transducer. The examiner sat in the lateral position using his right hand and the patient's head was tilted about 45° away from the artery being examined with relaxation of the neck to avoid contractions of the sternomastoid muscle that cause inadequate sonic penetration and troublesome probe placement. Carotid vessels were followed from the clavicle using the longitudinal view as it is considered the most advantageous view for data recording [15] (figure 1).

Ultrasound examination was done twice at the same anatomical position for 10 subjects: first by the radiologist investigator and principal investigator and then by the principal investigator one day apart as a pilot test. Estimation of inter and intra-observer agreement was evaluated by statistical analysis of the intra-rater reliability of measurements. Inter and intra-rater reliability test was done by calculating inter or intra-class correlation coefficient (ICC) respectively using a one-way random-effect model and evaluation of absolute agreement. CI was calculated at 95% for reliability coefficients (estimated ICC=0.90, with

95% CI, 0.75–0.98). The measurements of the pilot test were not included in the sample of this study.

The B-mode imaging was used for measuring CCAs IMT and for localizing plaque in the arterial wall. The optimal B-mode imaging angle was adjusted at 90° to the surface of the artery for ideal US reflection [16]. The IMT was measured at the far wall of the artery as it reflects the true wall thickness than the near-wall, in a segment without a focal lesion over a 10-mm segment located approximately 5 mm proximal to the carotid bulb [17]. Two radiant interfaces were seen along the artery wall, the interface between the blood and intima is represented by the upper radiant interface, and the interface between the media layer and adventitia layer is expressed by the lower radiant interface. The distance between the upper and lower bright lines represents IMT [18] (figure 1). Subjects with an IMT <0.8mm were considered to have no atherosclerosis and those with an IMT ≥ 0.8 mm were considered to have atherosclerosis [19].

For Spectral Doppler examination; the sample volume cursor was located in the central stream of the vessel. The angle of the Doppler beam to the long axis of the vessel was as close as possible to 60 [20] (figure 1). Spectral waveforms were obtained from both CCAs at proximal, mid and distal segments, peak systolic velocity (PSV) and end-diastolic velocity (EDV) of each segment were measured and mean value of these three was taken out [21]. Additionally, CCAs resistive index was calculated [resistive index = PSV-EDV / PSV] [22].

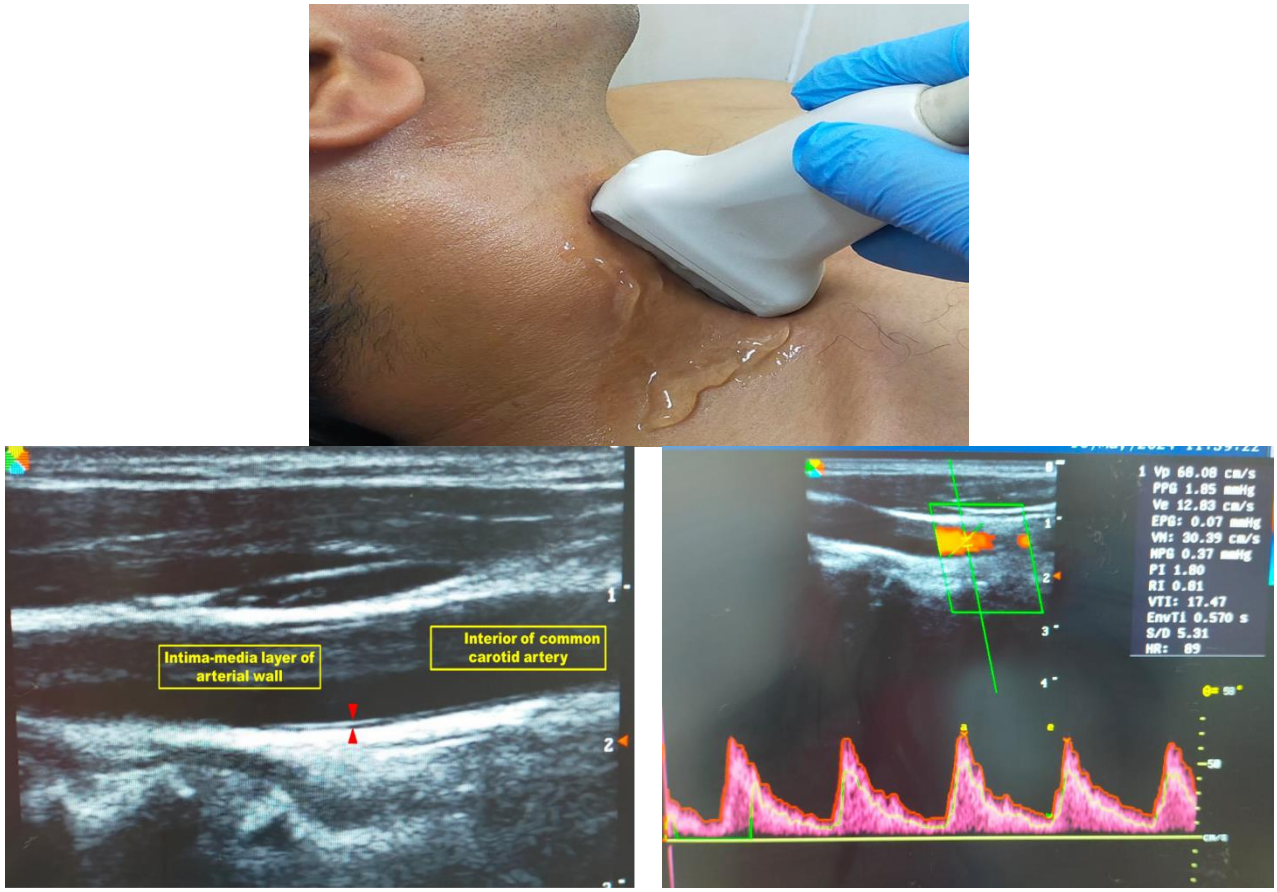


Figure (1): Ultrasonographic longitudinal view of common carotid artery

The upper image is the probe Position for examination of CCA by ultrasonography. The lower left image is the US B-mode longitudinal view of CCA: the upper arrowhead is the CCA intima, the lower arrowhead is the CCA media, and the distance between the 2 arrowheads represents CCA IMT. The lower left image is the Doppler US spectral waveform of CCA

Statistical analysis

The data was statistically analyzed by the Statistical Package for Social Science (SPSS) program version 17.0 (SPSS Inc., Chicago, USA). Shapiro-Wilk test was used for testing normality of quantitative variables. As the studied quantitative data were non-parametric it was expressed as median with interquartile range (IQR) and as percentages for nominal data. Mann Whitney (MW**)** was used for comparison of quantitative variables between two groups. The Chi-square (X^2) test was used for the comparison of qualitative variables between the studied groups. Linear correlation coefficient test (r) was used for the detection of a correlation between two quantitative variables in the asthma group. Multivariate logistic regression analysis was used to identify the most relevant risk factors for atherosclerosis among asthmatic patients. The strength of relevance between the risk factors and the outcome was determined according

to the value of the odd ratio, and significance according to the Wald Chi-square test. For all used tests the statistical significance was considered at a probability value (p-value) < 0.05 (with a confidence limit of 95%).

RESULTS

The features of the studied participants are illustrated in Table (1). Moreover, 51% of asthmatic patients had moderate asthma, 43% had partially controlled asthma, 53% had a family history of allergic diseases, and 76% had associated other allergic diseases. There was significant increase of CCAs IMT bilaterally in asthmatic patients than in control subjects (p<0.001), with no detectable plaques in either group. Atherosclerosis was more common in asthmatic patients than non-asthmatic control subjects (26% vs. 6%, respectively, p< 0.001) (table 2).

Table (1): Comparison of demographic data and spirometric-indices between bronchial asthma group and control group

Item		Asthma group n = 100	Control group n = 100	Stat. test	p-value
Sex	Males	18 (18%)	16 (16%)	0.14$^{\Delta}$	0.707
	Female	82 (82%)	84 (84%)		
Age (yrs.)	Median (IQR)	40 (34-43)	39 (32-43)	469$^{\#}$	0.458
BMI (kg/m^2)	Median (IQR)	27.4 (25.5-29)	26.7 (24.5-28.7)	428$^{\#}$	0.082
FEV_1/FVC	Median (IQR)	72 (69.2-80.4)	84 (82-87.9)	186$^{\#}$	0.002*
FVC%	Median (IQR)	80 (75.25-85)	85.5 (83.1-88)	226$^{\#}$	0.011*
FEV_1%	Median (IQR)	69.7(63.07-80.9)	84 (81.6-86)	970$^{\#}$	0.001*
FEF_{25-75}%	Median (IQR)	68 (66.6-71.3)	75 (72.6-77.8)	114$^{\#}$	0.005*
Asthma duration (yrs.)	Median (IQR)	30 (25.9-32.2)			
Asthma severity	Intermittent	16 (16%)			
	Mild	17 (17%)			
	Moderate	51 (51%)			
	Severe	16 (16%)			
Asthma control level	Controlled	32(32%)			
	Partially controlled	43(43%)			
	Uncontrolled	25(25%)			
Family history of allergic diseases		53 (53%)			
Associated other allergic diseases		76 (76%)			

IQR: Interquartile range, BMI: Body mass index, FEV_1%: Forced expiratory volume in first-second percent predicted, FVC%: Forced vital capacity percent predicted, FEF25-75%: Forced expiratory flow 25-75% percent predicted, IgE :Immunoglobulin E, Δ: Chi-square test, #: Mann Whitney U test, *: Significant p-value (< 0.05),

Table (2): Comparison of common carotid artery B mode and Doppler ultrasound indices between bronchial asthma group and control group

CCAs ultrasound indices		Asthma group n = 100	Control group n = 100	Stat. test	p-value
Right CCA IMT (mm)	Median (IQR)	0.67 (0.56-0.81)	0.54 (0.48 - 0.6)	221$^{\#}$	0.001*
Left CCA IMT (mm)	Median (IQR)	0.68 (0.59 - 0.8)	0.55 (0.49 - 0.6)	205$^{\#}$	0.001*
Right CCA PSV (cm/s)	Median (IQR)	50 (45 - 57)	49 (44 - 54.75)	439$^{\#}$	0.137
Left CCA PSV (cm/s)	Median (IQR)	49 (42 - 66.75)	50 (44.8 -58.15)	481$^{\#}$	0.658
Right CCA EDV (cm/s)	Median (IQR)	23.4 (19 - 27)	23 (18 - 26.5)	499$^{\#}$	0.980
Left CCA EDV (cm/s)	Median (IQR)	22 (18 - 28.7)	23 (19.5 - 26.3)	496$^{\#}$	0.931
Right CCA resistive index	Median (IQR)	0.56 (0.52-0.61)	0.55 (0.47 - 0.6)	419$^{\#}$	0.048*
Left CCA resistive index	Median (IQR)	0.57 (0.5 - 0.61)	0.55 (0.48 - 0.6)	459$^{\#}$	0.322
Plaques	No. (%)	0(0%)	0(0%)	-	-
Atherosclerosis	No. (%)	26 (26%)	6 (6%)	14.8$^{\Delta}$	0.001*

IQR: Interquartile range, CCA: Common carotid artery, IMT: Intima-media thickness, PSV: Peak systolic velocity, EDV: End diastolic velocity, Δ: Chi-square test., #: Mann Whitney U test, *: Significant p-value (< 0.05),

Table (3): Comparison of lipid profile indices, eosinophil count, and serum total immunoglobulin E between bronchial asthma group and control group

Lipid profile indices		Asthma group n = 100	Control group n = 100	Stat. test	p-value
TC (mg/dl)	Median (IQR)	162 (137.4-220.7)	136 (113.2-168.3)	299#	0.001*
TG (mg/dl)	Median (IQR)	128.5 (100.6-176.5)	121 (99.2-167.9)	468#	0.338
HDL (mg/dl)	Median (IQR)	41.2 (37-47)	47 (40.6-50)	335#	0.001*
LDL (mg/dl)	Median (IQR)	95.6 (70-120)	88.2 (56.9-102.1)	356#	0.001*
Non-HDL (mg/dl)	Median (IQR)	99 (83.6-123)	97.3 (79.2-108.7)	442#	0.158
VLDL (mg/dl)	Median (IQR)	27.8 (20.05-32.3)	24 (19.9-26.9)	393#	0.010*
Eosinophils (cmm)	Median (IQR)	154(100-215.7)	59.5(40-81.9)	135#	0.001*
Total IgE (IU/ml)	Median (IQR)	247.1(91-363.3)	51.4(32.1-83.4)	159#	0.001*
Dyslipidemia	No. (%)	44 (44%)	10 (10%)	29.3 Δ	0.001*
Eosinophilia	No. (%)	59 (59%)	4 (4%)	70.1 Δ	0.001*
Elevated IgE	No. (%)	74(74%)	6(6%)	96.3 Δ	0.001*

IQR: Interquartile range, TC: Total cholesterol, TG: Triglyceride, HDL: High=density lipoprotein, LDL: Low-density lipoprotein, Non-HDL: Non-high density lipoprotein, VLDL: Very-low density lipoprotein, Δ: Chi-square test. #: Mann Whitney U test, *: Significant p-value (< 0.05).

Table (4): Correlation of both common carotid artery intima-media thickness with other variables in bronchial asthma group

Item	Right CCA IMT n = 100		Left CCA IMT n = 100	
	r	p-value	r	p-value
Age (yrs.)	0.689	0.001*	0.663	0.001*
BMI (kg/m^2)	0.782	0.001*	0.75	0.001*
Age of asthma onset (yrs.)	-0.321	0.003*	-0.289	0.004
Asthma duration (yrs.)	0.748	0.001*	0.705	0.001*
ICS dose (mcg)	0.137	0.200	0.113	0.292
FEV_1/FVC ratio	-0.68	0.002*	-0.65	0.002*
FVC%	-0.74	0.001*	-0.721	0.001*
FEV_1%	-0.81	0.001*	-0.772	0.001*
FEF_{25-75}%%	-0.602	0.002*	-0.566	0.003*
TC (mg/dl)	0.846	0.001*	0.824	0.001*
TG (mg/dl)	0.836	0.001*	0.829	0.001*
HDL (mg/dl)	-0.676	0.002*	-0.691	0.002*
LDL (mg/dl)	0.826	0.001*	0.803	0.001*
Non-HDL (mg/dl)	0.216	0.031*	0.195	0.052
VLDL (mg/dl)	0.297	0.003*	0.194	0.053
Eosinophils (cmm)	0.779	0.002*	0.774	0.001*
Total IgE (IU/ml)	0.884	0.001*	0.85	0.001*

BMI: Body mass index, ICS: Inhaled corticosteroids, FEV_1%: Forced expiratory volume in first-second percent predicted, FVC%: Forced vital capacity percent predicted, FEF25-75%: Forced expiratory flow 25-75% percent predicted, TC: Total cholesterol, TG: Triglyceride, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, Non-HDL: Non-high density lipoprotein, VLDL: Very low-density lipoprotein, IgE: Immunoglobulin E, CCA IMT: Common carotid artery intima-media thickness, r: Pearson correlation coefficient, *: Significant p-value (< 0.05)

There was significant increase of TC, LDL, and VLDL, with significant decrease of HDL in asthma group than control group (p< 0.001 each). Both eosinophil count and total IgE were significantly higher in asthmatic participants than in healthy subjects (p< 0.001 each). Accordingly, dyslipidemia, eosinophilia, and elevated total IgE were more common in asthmatic patients (44%, 59%, and 74% respectively) than in healthy subjects (10%, 4%, and 6% respectively) (p< 0.001 each) (table 3).

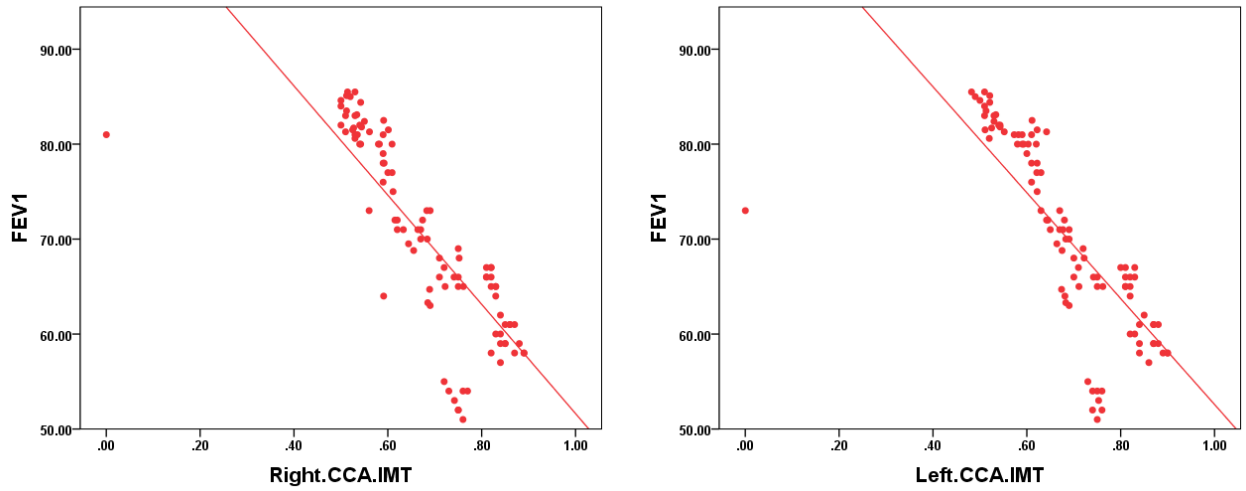
Table (4) demonstrates that in the asthma group, both CCAs IMT were positively correlated with the patient's age, BMI, asthma duration, TC, TG, LDL, eosinophils count, and total IgE (p < 0.05) (figure 2). On the other hand, they were negatively correlated with age of asthma onset, FEV_1/FVC ratio, FVC %, FEV_1% (figure 2), FEF_{25-75}%, and HDL IgE (P < 0.05). Additionally, the right CCA IMT was positively correlated with non-HDL and VLDL (p <0.031 and <0.003, respectively).

The significant risk factors of atherosclerosis in asthmatic patients in descending orders were; higher LDL (OR 7.160), family history of allergic diseases (OR 5.512), poor asthma control level (OR 4.318), higher eosinophils (OR 4.110), higher total IgE (OR 4.066), younger age of asthma onset (OR 2.768), higher BMI (OR 2.418), lower FVC% (OR 2.220), lower FEV_1% (OR 2.169), and low FEV_1/FVC ratio (OR 2.129), longer asthma duration (OR 1.704), and higher patient age (OR 1.212). On the other hand, HDL demonstrates a protective effect (OR 0.071) (table 5).

Table (5): Multivariate logistic regression analysis for risk factors of atherosclerosis in bronchial asthma group

Item	B	SE	p-value	OR	95% CI	
Age (yrs.)	0.192	0.062	0.002*	1.212	1.072	1.37
BMI (kg/m^2)	0.883	0.214	0.001*	2.418	1.59	3.677
Age of asthma onset (yrs.)	-0.570	0.138	0.002*	2.768	1.35	2.31
Asthma duration (yrs.)	0.351	0.078	0.002*	1.704	0.60	0.81
Asthma control level	-1.463	0.379	0.001*	4.318	2.053	9.082
Family history of allergic diseases	1.707	0.550	0.002*	5.512	1.875	16.205
Associated other allergic diseases	0.372	0.564	0.510	0.451	0.480	4.385
ICS use	-1.081	0.580	0.062	0.339	0.109	1.057
ICS dose (mcg)	0.001	0.001	0.657	0.001	0.998	1.003
SABA use	0.41	0.088	2.02	0.9	4.53	0.70
LABA use	0.634	0.677	0.349	0.530	0.141	1.998
Theophylline use	0.865	0.484	0.074	0.375	0.919	6.137
FEV_1/FVC ratio	- 0.121	0.038	0.001*	2.129	1.048	1.216
FVC%	- 0.198	0.052	0.001*	2.220	1.101	1.351
FEV_1%	- 0.156	0.036	0.001*	2.169	1.089	1.254
FEF_{25-75}%	0.136	0.076	0.073	2.146	0.987	1.330
Eosinophils (cmm)	10.411	19.10	0.036*	4.110	6.110	2.6
Total IgE (IU/ml)	1.403	47.14	0.002*	4.066	7.711	5.5
HDL (mg/dl)	2.647	0.984	0.007*	0.071	0.01	0.488
LDL (mg/dl)	11.808	218.92	0.006*	7.160	2.106	88.32
TC (mg/dl)	0.560	0.320	0.081	0.571	0.305	1.071
TG (mg/dl)	4.987	144.05	0.972	0.007	2.72	16.23
Non-HDL (mg/dl)	0.012	0.007	0.067	0.988	0.975	1.001
VLDL (mg/dl)	0.025	0.025	0.303	0.975	0.929	1.023

BMI: Body mass index, ICS: Inhaled corticosteroids, SABA: Short-acting beta-agonist, LABA: Long acting beta agonist, FEV_1%: Forced expiratory volume percent predicted, IgE: Immunoglobulin E, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, TC: Total cholesterol, TG: Triglyceride, Non-HDL: Non-high density lipoprotein, VLDL: Very low-density lipoprotein, B: Regression coefficient, SE: Standard error, OR: Odds ratio, CI: Confidence interval, *: Significant p-value (< 0.05).



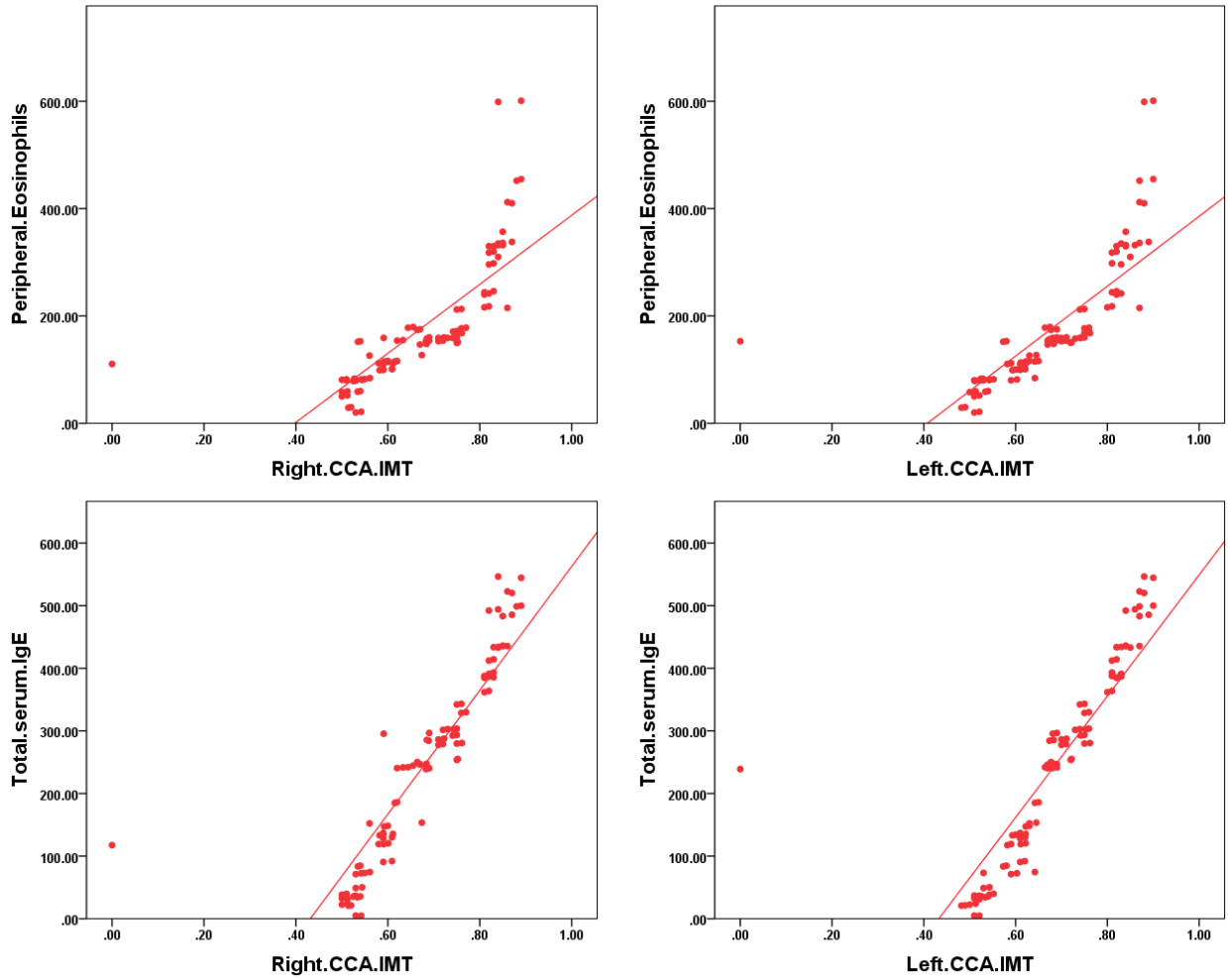


Figure (2): Correlation of both common carotid arteries initima-media thickness with forced expiratory volume in furst-second %, eosinophils count, and serum total immunoglobulin E in bronchial asthma

DISCUSSION

Owing to asthma prevalence and the critical coexistence of CVD, understanding this relationship between asthma and CVD would be illuminative to the clinicians managing asthmatic patients and have important implications for disease management and targeted treatments [23]. Therefore, this work aims to study atherosclerosis in asthmatic patients and to identify clinical characteristics and risk factors related to it in these patients.

Since the purpose of this study was the identification of early atherosclerotic changes in asthmatic patients, this study was conducted on young and middle-aged adults, non-obese, and non-smokers in whom the risk of atherosclerosis was very low. After fully adjusting for potential confounders for atherosclerosis, asthmatic patients had significantly higher atherosclerosis prevalence than healthy controls (26% vs. 6% respectively). These results have ascertained bronchial asthma itself as an etiological risk factor for atherosclerosis. Although it is still largely unknown which potential biological mechanisms of asthma are involved in subclinical atherosclerosis in young asthmatics, chronic systemic inflammation has been claimed to have a pivotal function in the development of asthma and endothelial dysfunction which is an influential parameter in the development of subclinical atherosclerosis. Endothelial dysfunction has been remarked as an indicator that may suggest early abnormal vascular function and structure and is typically present in asthmatic adults confirming the association of the development of endothelial dysfunction with asthma [24]. Similar results were reported in previous studies [25-26]. Additionally, further epidemiological studies have concluded that asthma is associated with an increased risk of coronary artery disease by up to about 30% [27-28], On the contrary, Podgórski [29], and Otsuki et al. [30] studies revealed that the frequency of atherosclerotic carotid arteries lesions was significantly lower in asthmatic patients than in healthy participants. This difference could be explained by different inclusion criteria of studied participants, as Otsuki et al. [30] included those with other risk factors of atherosclerosis such as smoking, diabetes mellitus, and hypertension.

Although there were no atheromatous plaques in any studied participants, asthmatic patients had significantly increased right CCA resistive index than controls. These findings

indicate that the studied asthmatic patients have early atherosclerotic changes that if left untreated will progress to more advanced stages with the affection of cardiovascular, cerebrovascular, and peripheral arteries [29]. These results are matched with Tattersall et al., [25], but they differ from this study in that visible plaques weren't detected and this could be ascribed to the younger age group of participants. The results of current study differ from Podgórski [29] as he concluded that patients with bronchial asthma had a decreased risk of atherosclerosis than controls.

This present study showed that in the asthma group; both CCA IMT were negatively correlated with all spirometric indices. Moreover, the lower FVC%, FEV_1%, and FEV_1/FVC ratio were risk factors for atherosclerosis. These findings suggest a strong association between asthma severity and a significant risk of subclinical atherosclerosis that could be explained by that severe asthma has an enhanced underlying airway inflammation with more systemic spillover that subsequently leads to atherosclerosis. Additionally, severe asthma is frequently associated with hypoxemia alters vessels' structure and function and increases the risk of atherosclerosis. Corlateanu et al. [31] concluded that the higher prevalence of atherosclerosis in patients with either uncontrolled or severe asthma could be attributed to the high prevalence of comorbidities encountered with uncontrolled severe asthma, more flare-ups with more frequent inflammation, adverse reactions of extra medications, and the remodeling process linked to pulmonary function decline. However, the studied asthmatic patients have no comorbid diseases suggesting that the severity of airway inflammation could be the main etiological factor for atherosclerosis in asthmatic patients. Declined lung function has been correlated with increased CVD mortality among asthmatic patients [1].

This study revealed significantly increased TC, LDL, and VLDL with significantly decreased HDL, with a significantly higher prevalence of dyslipidemia in asthmatic patients than control subjects (44% vs.10% respectively). Both CCAs IMT were positively correlated with TC, TG, and LDL, and the right CCA IMT was positively correlated with non-HDL and VLDL. Additionally, both CCAs IMT were negatively correlated with HDL (table 4). These findings confirm the anticipated role of dyslipidemia in the pathogenesis of atherosclerosis mediated by the underlying inflammatory reaction of asthma [32]. Moreover, Feingold and Grunfeld [33] explained the mechanism of the development of dyslipidemia in asthmatic subjects; they reported that multiple cytokines can increase serum TG and VLDL levels via an increase in adipose tissue lipolysis. This would result in increased hepatic VLDL production and secretion and a decline in the clearance of TG-rich lipoproteins by a decrease in lipoprotein lipase. The increase in TG-rich lipoproteins, in turn, leads to an increase in the interchange of TG from TG-rich VLDL to LDL so increasing the TG content of LDL. This gives rise to the increased formulation of small dense LDL that is believed to be more pro-atherogenic. Also, elevations in TG-rich lipoproteins can lead to the enrichment of HDL with TGs with resultant acceleration of HDL clearance. Ko et al. [34] study emphasized the relation between asthma and dyslipidemia. Another study highlighted the role of dyslipidemia in atherosclerosis development [30]. The current study revealed that higher LDL was the strongest risk factor for atherosclerosis in asthmatic patients (OR 7.160) and HDL had a significant atheroprotective role (OR 0.071). These findings indicate that the increased LDL might hurt endothelial functions in asthmatic patients. In contrast, HDL might have beneficial effects in patients with bronchial asthma as it has anti-inflammatory effects and its decrease could predispose asthmatic patients to atherosclerosis [35].

In the present study, asthmatic patients had significantly higher eosinophil count with a significantly higher frequency of eosinophilia than control subjects. Moreover, both CCAs IMT were positively correlated with eosinophil count. The eosinophil count was a significant risk factor for atherosclerosis in asthmatic patients (OR 4.110). These findings confirm the role of eosinophils in atherosclerosis. Marx et al. [36] reported that eosinophils increase endothelial von Willebrand factor expression and cause platelet adhesion, then eosinophils activate the attached platelets, and both eosinophils and platelets can contribute to atherosclerotic plaque formation and thrombosis. Current results are in concordance with Tuleta et al. [1] study who report a close association between eosinophils and atherosclerosis. On the contrary, Gao et al. [37] concluded that atherosclerosis was associated with lower eosinophil; this difference may be due to the exclusion of patients with allergic diseases including asthma from their study.

The current study revealed that the total IgE levels and frequency of elevated IgE were significantly higher in asthmatic patients than in controls. Both CCAs IMT were positively correlated with total IgE. Additionally, higher total IgE was one of the strongest risk factors for atherosclerosis in asthma (OR 4.066). These results imply that IgE plays a pivotal function in both asthma and atherosclerosis through allergic interaction with eosinophils in inflammatory allergic pathways. In favor of this theory; most studied asthmatic patients had a family history of allergic diseases (53%) and a history of associated allergic diseases (76%). IgE activates several inflammatory cells that contribute to atherogenesis particularly mast cells, also macrophages, eosinophils, monocytes, dendritic cells, and vascular endothelial cells by binding with high affinity to FcεR1expressed on these cells [39]. Similar results regarding the role of allergy in atherosclerosis had been disclosed in a previous study [39].

In this study, both CCAs IMT were negatively correlated with age of asthma onset and positively correlated with asthma duration. Longer asthma duration and poor asthma control were significant risk factors for atherosclerosis in asthmatic patients (OR 4.318 and 1.704, respectively). These

findings suggest that both longer asthma duration and uncontrolled asthma were always associated with persistent underlying inflammatory process, remodeling, and chronic airway obstruction lead to decreased pulmonary function with subsequent development of atherosclerosis. Our results are compatible with Wee et al. [23] and Tattersall et al. [25].

The current study revealed that older patient age is an anticipating liability factor for atherosclerosis in asthmatic patients (OR1.212) and both CCAs IMT were positively correlated with age. The possible explanation for this finding is that besides asthma-related systemic inflammation, it is well known that increasing age is always associated with low-grade systemic inflammation with premature cellular senescence. Both chronic inflammation and cellular senescence predispose to atherosclerosis. Tyrrell and Goldstein [40] postulated that with aging, hematopoietic stem cells lose their regenerative capacity due to epigenetic dysregulation despite an increase in their absolute number. Additionally, aging cells have other features including an elevated tendency to undergo senescence, increased nucleic acid damage, and marked telomere shortening and dysfunction. All of these characteristics can be identified in cells from atherosclerotic plaques [41]. This result is matched with Otsuki et al. [30] who demonstrated that atherosclerosis is linked to age in asthmatic patients. Hence the study participants were ≤45 years of age; the detected atherosclerosis in asthmatic patients indicates that the atherosclerotic changes start either in childhood or early adulthood, which strongly suggests that atherosclerosis is not just confined to the elderly.

This study demonstrates that higher BMI is a risk factor for atherosclerosis in asthmatic patients (OR 2.418) and both CCAs IMT were positively correlated with BMI. These findings indicate that obesity-induced dyslipidemia and obesity-induced low-grade systemic inflammation could have a potential role in increased tendency to athero-sclerosis in asthmatic patients. Pakhare and Anjankar [42] documented that obesity raises the risk of CVD through associated risk factors like elevated levels of insulin, dyslipidemia, and high blood pressure. Moreover, obesity is not only based on a higher BMI definition of obesity, but also normal weight obesity which is abdominal obesity in the absence of overall obesity is considered a risk factor of cardiovascular disease [43]. Our results are matched with Sturesson et al. study [44].

A very interesting and important finding of the current study is that the most commonly used asthma medications (ICS, LABA, SABA) have no atherogenic or athero-protective role in asthmatic patients. Accordingly, it can be used it freely, but it must be taken into consideration the other cardiovascular effects of beta-agonist such as tachycardia and arrhythmia.

The main strength of this study is the control of confounders that may predispose asthmatic patients to atherosclerosis; therefore, the results highlight the atherogenic role of asthma. Although this may bias conclusions derived for the whole asthma population. However, this study encountered some limitations, by being an observational study; therefore, the described associations do not confirm causation. Another important concern of our research is the assessment of asthma severity, control, and medications reported only in the preceding six months, not during the entire course of the disease. Additionally, the endotypes of asthma were not evaluated. Finally, it was a hospital-based study, conducted at a single center, thus, the studied population might not be representative of all asthma populations.

CONCLUSION

This study declares proof for a conjunction between asthma and atherosclerosis, in particular uncontrolled severe persistent asthma. So annual screening using carotid duplex US and lipid profile analysis is recommended in susceptible asthmatic patients. Additionally, the data implicate that the utmost impressive risk factors for atherosclerosis in asthmatic patients are dyslipidemia, lower pulmonary function, higher eosinophil count, and higher total IgE. Moreover, the association between atherosclerosis and early-onset asthma, family history of allergic diseases, elevated eosinophils, and increased total IgE suggest that asthma and atherosclerosis may share allergic pathogenic pathways. Future studies assessing the relationship between different asthma endotypes, and atherosclerosis are recommended.

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الملخص العربي

دراسة تصلب الشرايين في مرضى الربو الشعبي

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ملخص البحث

الخلفية : عادة ما يكون الربو الشعبي مصحوبا بمضاعفات تشمل مختلف أجهزة الجسم و التي تساهم في ما يسببه الربو الشعبي من عبء .هذه المضاعفات تشمل أمراض القلب و الأوعية الدموية و التي يعتبر تصلب الشرايين هوالسبب المؤدي لحدوثها على مستوى التشريح المرضي.

الهدف: دراسة تصلب الشرايين في مرضى الربو الشعبي و معرفة الخصائص السريرية و عوامل الخطورة المساعدة لتواجده في مرضى الربو الشعبي.

الطرق: اشتملت دراسة الحالات والشواهد هذه على 100 مريضا بالربو الشعبي و 100شخصا سليما. خضع جميع المشاركين لعمل وظائف التنفس، قياس عدد خلايا الحمضات في الدم ،قياس مستوى الدهون بالدم، قياس الجلوبيولين المناعي هـ الكلي في مصل الدم و موجات فوق صوتية على الشريان السباتي.

النتائج: كان عدد خلايا الحمضات، قياس الجلوبيولين المناعي هـ الكلي، مستوى الكوليسترول، البروتين الدهني منخفض الكثافة و البروتين الدهني منخفض الكثافة للغاية أعلى احصائيا، بينما كان البروتين الدهني عالي الكثافة منخفض احصائيا في مرضى الربوالشعبي مقارنة بالأشخاص الأصحاء. و بالتالي كانت نسبة عسر شحميات الدم أعلى احصائيا في مرضى الربوالشعبي مقارنة بالمشاركين الأصحاء (44% مقارنة ب10%على التوالي). كان قياس سمك الوسطانية - الباطنية للشريان السباتي على كلا الجانبين اكبر احصائيا و بالتالي كان معدل تصلب الشرايين أعلى احصائيا في مرضى الربو الشعبي مقارنة الأصحاء (26% مقارنة ب 6%على التوالي). في مرضى الربو الشعبي تناسب قياس سمك الوسطانية -الباطنية للشريان السباتي على الجانبين إيجابيا مع السن، مؤشر كتلة الجسم، مدة الربو الشعبي، الكوليستيرول الكلي، الدهون الثلاثية، البروتين الدهني منخفض الكثافة، عدد خلايا الحمضات و قياس الجلوبيولين المناعي هـ الكلي. بينما تناسب سلبيا مع عمر المريض عند حدوث اول نوبة للربو، معدل وظائف التنفس و البروتين الدهني عالي الكثافة. كانت أكثر عوامل الخطورة ذات الدلالة الإحصائية لتصلب الشرايين في مرضى الربو الشعبي تتضمن: إرتفاع البروتين الدهني منخفض الكثافة (نسبة احتمال 7,160)، وجود تاريخ عائلي لأمراض الحساسية (نسبة احتمال 5,512) ،عدم التحكم بالربو الشعبي (ن نسبة احتمال 4,318)، زيادة عدد خلايا الحمضات (نسبة احتمال 4,110)، ارتفاع الجلوبيولين المناعي هـ الكلي (نسبة احتمال 4,066)، بداية الربو الشعبي في سن مبكر(نسبة احتمال 2,768) ، زيادة مؤشر كتلة الجسم (نسبة احتمال 2,418)، إنخفاض معدل وظائف التنفس (نسبة احتمال >2)، طول مدة الربو الشعبي(نسبة احتمال 1,704) و كبر سن المريض (نسبة احتمال 1,212)، و على العكس من ذلك كان للبروتين الدهني عالي الكثافة أثر وقائي (نسبة الاحتمالات 0,071).

الاستنتاجات: توضح هذه الدراسة ارتباط الربو الشعبي بتصلب الشرايين و تحديدا الربو الشعبي متواصل الشدة، الغير متحكم فيه. إضافة إلى ذلك فإن أكثر عوامل الخطورة المؤثرة في حدوث تصلب الشرايين في مرضى الربو الشعبي تشمل عسر دهون الدم، انخفاض معدل وظائف التنفس، زيادة عدد خلايا الحمضات، و ارتفاع قياس الجلوبيولين المناعي هـ الكلي.

الكلمات المفتاحية: عسر شحميات الدم ، سماكة الوسطانية - الباطنة للشريان السباتي ، خلايا الحمضات ، الجلوبيولين المناعي هـ الكلي.

الباحث الرئيسي

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