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Level of Ischemia Modified Albumin in the Sera of Families with a Case of Rheumatic Fever and/or Rheumatic Heart Diseases

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Abstract

Background: Ischaemia-modified albumin (IMA), a novel biochemical marker for tissue ischaemia, was found to be associated with oxidative stress.

Aim of Study: Was to measure the level of ischemia modified albumin in sera of families with a case of rheumatic fever/rheumatic heart diseases whatever their presentation.

Patients and Methods: This study was done on 10 families of RF including 10 index cases, 10 mothers and 10 fathers and 20 siblings (total n=50) attending the RF clinic of pediatric department, All Patients had a full sheet, complete clinical examination and routine laboratory investigations (ESR, CRP, Blood group, CBC, ASOT and IMA) and ECHO. Control group, comprised 10 families including 10 mothers, 10 fathers and 30 siblings (total n=50).

Results: There was significant increase of IMA in between [cases of typical group as compared to control cases p(<0.001*) - cases of atypical group as compared to control cases p(0.001*) - cases of atypical group as compared to their siblings p(<0.001*), there was no significant increase of IMA in between (cases of typical group as compared to their siblings p(0.071) - siblings of atypical group as compared to control siblings p(0. 182)].

Conclusion: Our study concluded that serum IMA level and ASOT increased in children with ARF/RHD as well as their siblings which facilitate early detection and management of RF in siblings of a case of RF depending on positive family history and elevating anti-streptococcal antibodies and serum Ischemia modified albumin.

Key Words: *Ischemia modified albumin – Acute rheumatic fever – Rheumatic heart diseases.*

Introduction

ACUTE rheumatic fever (ARF) is a post infectious, nonsuppurative sequelae of pharyngeal infection with Group A β hemolytic Streptococcus (GABHS). Of the associated symptoms, only damage to the valve tissue within the heart, or rheumatic heart disease (RHD), can become a chronic condition leading to congestive heart failure, strokes, endocarditis, and death [1].

ARF is characterized by nonsuppurative inflammatory lesions of the joints, heart, subcutaneous tissue, and central nervous system [2]. The overall attack rate after streptococcal pharyngitis varies between 0.3-3%, but certain genetically predisposed individuals, comprising perhaps 3%-6% of the population, account for those who develop rheumatic fever [1].

Rheumatic fever manifests as various signs and symptoms that may occur alone or in various combinations: Sore throat, polyarthritis, carditis, sydenham chorea, erythema marginatum and subcutaneous nodules. Other symptoms may include fever, abdominal pain, arthralgia, malaise, and epistaxis [3].

If supported by evidence of preceding group A streptococcal infection, the presence of two major manifestations or one major and two minor manifestations indicates a high probability of ARF. Failure to fulfill the Jones criteria makes the diagnosis unlikely but not impossible. Clinical judgment is required. The World Health Organization (WHO) follows the Jones criteria for the diagnosis of ARF, but possible recurrences require only two minor criteria plus evidence of recent streptococcal infection [1].

Jones criteria include major criteria as follows: Carditis (based on clinical criteria), polyarthritis, chorea, erythema marginatum, subcutaneous nodules. Minor criteria are as follows: Arthralgia, fever, elevated ESR or CRP level and prolonged PR interval [4].

As regards diagnosis there is no single specific laboratory test can confirm the diagnosis of ARF.

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Throat culture remains the criterion standard for confirmation of group A streptococcal infection, Antibody titer tests used include anti streptolysin O titre (ASO) test. Acute-phase reactants, the erythrocyte sedimentation rate (ESR), and C-reactive protein levels (CRP) are usually elevated at the onset of ARF and serve as a minor manifestation in the Jones criteria. These tests are nonspecific, but they may be useful in monitoring disease activity [5].

Ischaemia-modified albumin is a new marker for tissue ischaemia. It has been proposed that reactive oxygen species generated during ischaemia results in ischaemia-modified albumin formation. Ischaemia-modified albumin is accepted as a marker of oxidative stress. Recently, elevated ischaemia-modified albumin levels have been reported in various clinical conditions such as ischaemia, inflammation, and oxidative stress. Furthermore, increased oxidative stress has been shown in acute rheumatic fever and heart valve disease [6].

Aim of work:

To measure the level of ischemia modified albumin in sera of families with a case of rheumatic fever/rheumatic heart diseases whatever was their presentation. Moreover, the level of ischemia modified albumin gave us an idea about the state of affection of the heart valves that helped us in treatment protocol.

Subjects and Methods

This study was carried out at the Cardiac Unit, Pediatric Department, Tanta University Hospital from June 2017 – April 2018.

This study was done on 10 families of rheumatic fever (RF) cases including 10 index cases, 10 mothers and 10 fathers and 20 siblings (total n=50) attending the RF clinic of pediatric department, Tanta University Hospitals. Control group, comprised 10 families including 10 mothers, 10 fathers and 30 siblings (total n=50).

Classification of patient and control individuals:

1- 5 families of typical presentation of rheumatic fever/rheumatic heart disease in any member including childern aged from 5-15 years.
2- 5 families of atypical presentation of rheumatic fever in any member including childern aged from 5-15 years.
3- 10 families who have no history of acute rheumatic fever nor on long acting penicillin was enrolled in the study as a control group.

Results

T&A: Comparison between Typical and Atypical group.

T&C: Comparison between Typical and Control group.

A&C: Comparison between Atypical and Control group.

Table (1): Age distribution among the studied group.

Age	Groups			ANOVA	
	Typical	Atypical	Controls	F	*p*-value
Case:					
Range	10-13	9-15	6-15	0.488	0.622
Mean ± SD	11.400± 1.140	11.600± 2.191	10.500± 2.677		
Sibling:					
Range	5-16	5-18	3-13	2.424	0.102
Mean ± SD	8.600± 4.195	10.400± 4.766	7.300± 2.618		
t-Test:					
t	1.441	0.528			
p-value	0.173	0.606			

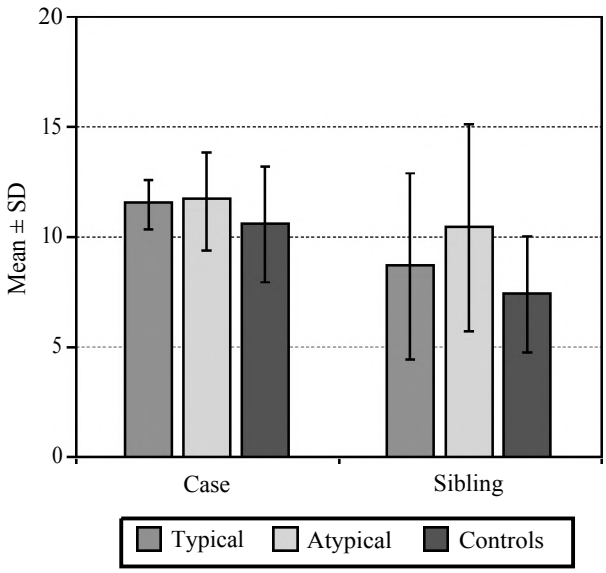

Fig. (1): Age distribution among the studied groups.

Table (1) and Fig. (1) show that there was no significance difference between studied groups as regard the age with mean 11.4 years among cases of typical group and 11.6 years among atypical group.

Table (2): Comparison of mean ESR (1 st h) among the studied groups.

ESR 1	Groups			ANOVA		TUKEY'S Test		
	Typical	Atypical	Controls	F	*p*-value	T&A	T&C	A&C
Case:								
Range	20-70	30-40	3-30	16.75	<0.001 *	0.582	<0.001 *	0.002*
Mean ± SD	43.00±18.57	36.0±4.18	11.50±8.16					
Sibling:								
Range	10-40	10-30	3-25	7.154	0.002*	0.041*	0.002*	0.240
Mean ± SD	24.00±12.42	17.0±6.32	11.55±7.08					
t-Test:								
t	2.376	6.032						
p-value	0.034*	<0.001 *						

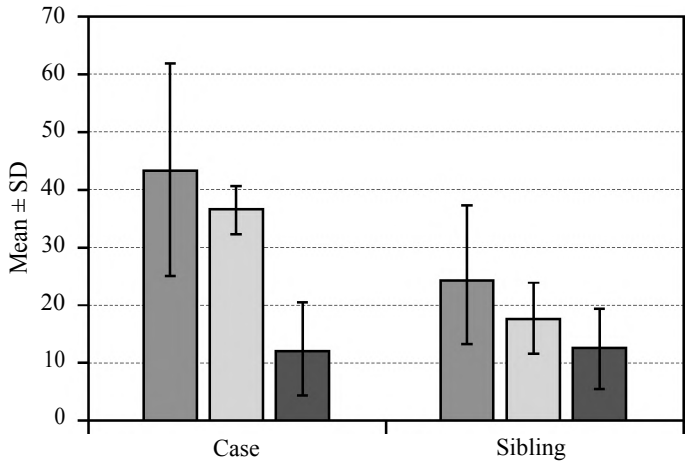

Fig. (2): Comparison of Mean ESR (1 st h) among the studied groups.

Table (2) and Fig. (2) show that there was significant increase of mean ESR (1 st h) in between [cases of typical group as compared to control cases p(<0.001*) - cases of atypical group as compared to control cases p(0.002*) - cases of typical group as compared to their siblings p(0.034*) - cases of atypical group as compared to their siblings p(<0.001*) - siblings of typical group as compared to control siblings p(0.002*) -siblings of typical group as compared to siblings of atypical group p(0.041 *)], there was no significant increase of mean ESR (1 st h) in between [cases of typical group as compared to cases of atypical group p(0.582)-siblings of atypical group as compared to control siblings p(0.240)].

Table (3): Comparison of IMA level among the studied groups.

IMA (pg/ml)	Groups			ANOVA		TUKEY'S Test		
	Typical	Atypical	Controls	F	*p*-value	T&A	T&C	A&C
Case:								
Range	215-432	190-220	12-150	32.894	<0.001 *	0.03 1 *	<0.001 *	0.001*
Mean ± SD	292.40±82.13	202.00±13.038	73.400±43.002					
Sibling:								
Range	80-323	70-130	12-132	20.874	<0.001 *	0.001 *	<0.001 *	0.182
Mean ± SD	196.20±92.43	99.200±19.71	61.500±37.801					
t-Test:								
t	1.965	10.470						
p-value	0.071	<0.001 *						

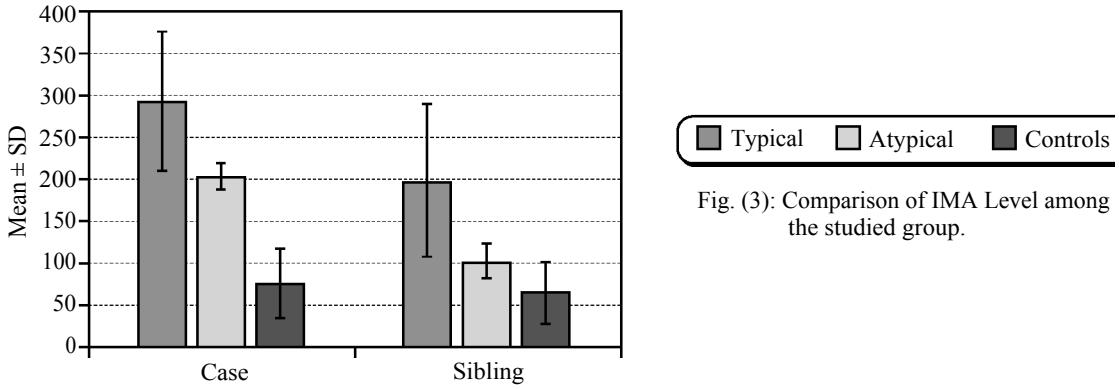

Fig. (3): Comparison of IMA Level among the studied group.

Table (3) and Fig. (3) show that there was significant increase of IMA in between [cases of typical group as compared to control cases p(<0.001 *) - cases of atypical group as compared to control cases p(0.001 *) - cases of atypical group as compared to their siblings p(<0.001 *) - cases of typical group as compared to cases of atypical group p(0.031 *) - in siblings of typical group as compared to control siblings p(<0.001 *) - siblings of typical group as compared to siblings of atypical group p(0.001*)], there was no significant increase of IMA in between (cases of typical group as compared to their siblings p(0.071) - siblings of atypical group as compared to control siblings p(0.182)].

Table (4): Blood group distribution among the studied groups.

Blood group	Groups								Chi-Square	
	Typical		Atypical		Controls		Total			
	N	%	N	%	N	%	N	%	X^2	p-value
Case:										
A+	3	60.00	2	40.00	2	20.00	7	35.00	3.571	0.734
B+	1	20.00	2	40.00	3	30.00	6	30.00		
O+	0	0.00	0	0.00	1	10.00	1	5.00		
AB+	1	20.00	1	20.00	4	40.00	6	30.00		
Sibling:										
A+	4	40.00	4	40.00	5	25.00	13	32.50	2.769	0.837
B+	0	0.00	0	0.00	2	10.00	2	5.00		
O+	3	30.00	3	30.00	6	30.00	12	30.00		
AB+	3	30.00	3	30.00	7	35.00	13	32.50		
Chi-Square:										
X^2	3.911		5.625							
p-value	0.271		0.131							

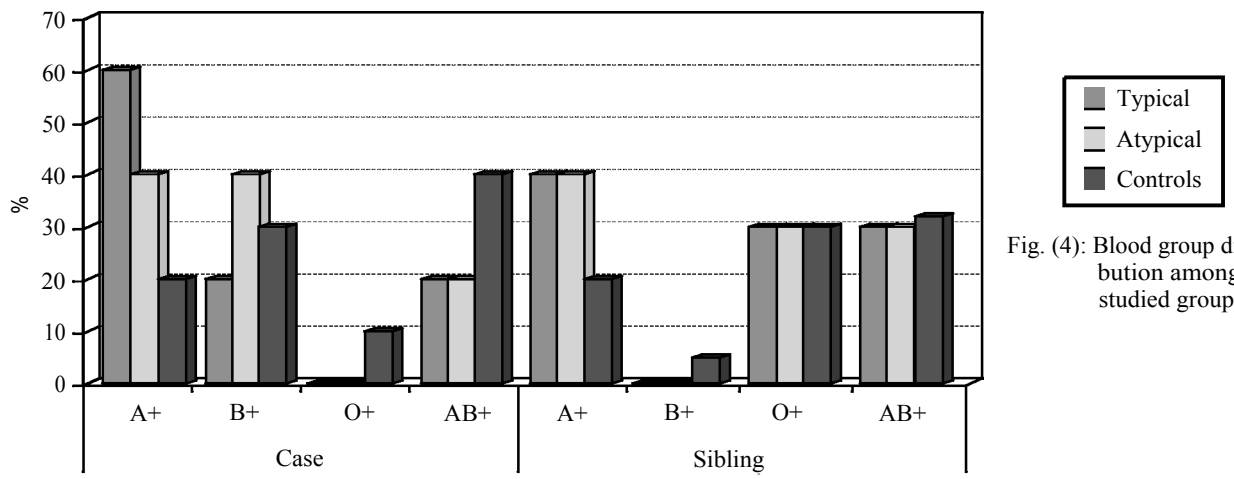

Fig. (4): Blood group distribution among the studied groups.

Table (4) and Fig. (4) show that A+ blood group was more common in typical cases while A+ and B+ were more common in studied atypical cases.

Table (5): Comparison of CRP results among the studied groups.

CRP	Groups								Chi-Square	
	Typical		Atypical		Controls		Total			
	N	%	N	%	N	%	N	%	X2	*p*-value
Case:										
Negative	0	0.00	0	0.00	10	100.00	10	50.00	20.000	<0.001*
Positive	5	100.00	5	100.00	0	0.00	10	50.00		
Sibling:										
Negative	5	50.00	5	50.00	18	90.00	28	70.00	7.619	0.022*
Positive	5	50.00	5	50.00	2	10.00	12	30.00		
Chi-Square:										
X2	5.232		5.232							
p-value	0.022*		0.022*							

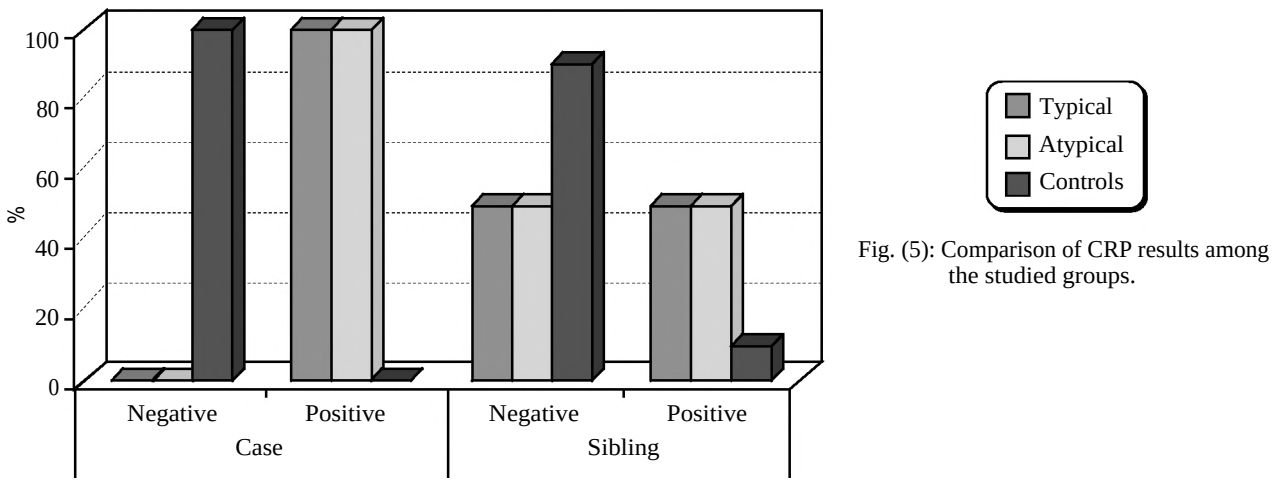

Fig. (5): Comparison of CRP results among the studied groups.

Table (5) and Fig. (5) show that there was significant increase of CRP level in patients as compared to controls in the two studied groups with (p=< 0.001*), and there was significant increase in patients as compared to their siblings in the two studied groups with (p=0.022*) also there was significant increase in patient siblings as compared to control siblings with (p=0.022*) in the two studied groups.

Table (6): Clinical data of siblings of families.

CRP	Sibling of families	
	N	%
Organic lesion	7	35.00
Atypical	3	15.00
Normal	10	50.00
Total	20	100.00

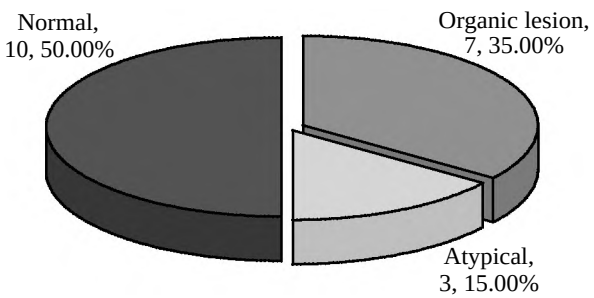

Fig. (6): Clinical data of siblings of families.

Table (6) and Fig. (6) show that 10 siblings were normal with percentage 50%, 7 siblings had atypical presentation with percentage 35% and 3 had organic lesions with percentage 15%.

Discussion

Acute rheumatic fever is an autoimmune inflammatory process that develops as a sequelae of streptococcal infection. ARF has extremely variable manifestations and remains a clinical syndrome for which no specific diagnostic test exists. Persons who have experienced an episode of ARF are predisposed to recurrences following subsequent GAS infections. The most significant complication of ARF is RHD, which usually occurs after repeated bouts of acute illness [7].

To avoid over diagnosis in low-incidence populations and to avoid under diagnosis in high-risk populations, variability in applying diagnostic criteria in low-risk compared with high-risk populations is reasonable [8].

ARF is diagnosed in the presence of two major, or one major and two minor manifestations and must be accompanied by supporting evidence of antecedent GAS infection in the form of positive throat culture or elevated or rising anti-streptolysin titer [9]. Moreover, Shoheib concluded that, ARF may present with three major signs [10].

The aim of this work was to estimate the value of measuring serum ischemia modified albumin in the sera of children of families with a case of RF/RHD.

The present study was done on 10 families of RF attending the RF clinic of pediatric department, Tanta University Hospitals. Control Group was 10 families free from RF or any systemic diseases.

In the present study, the age of patients ranged from 5-15 years old with mean value of 10 years. Most studies attribute this age of incidence to the repeated sore throat and streptococcal infection in school children [11].

Previous reports have indicated an association between ABO blood group and RF, but the results differ from one study to another. In the present work, when comparing the results of ABO blood group of rheumatic patients to that of control, a definite increase in A+ (50%) and B+ (30%), at expense of AB+ and O+. Group these results are similar to previous study of Shoheib et al., which concluded that both A+ and B+ blood groups were more common in patients than in controls [12].

Our patients showed statistically significant differance between the studied groups compared to control as regard TLC and CRP as they are considered acute-phase reactants which are usually elevated at the onset of ARF and serve as a minor manifestation in the Jones criteria. These tests are nonspecific, but they may be useful in monitoring disease activity) [13].

We found in this work 100% of rheumatic cases were positive for CRP with significant increase compared with control in both typical and atypical groups (p-value <0.001*). We found also 50% of rheumatic patient siblings were positive for CRP compared with their control (p-value=0.022*).

Similar results were obtained by Rayamajhi et al. in a prospective, cross sectional study over 2 years on 36 children under 14 years with RF and found that elevated CRP in 78% of patients and this was confirmed by Echocardiography [14].

Our results showed that ESR was significantly elevated in rheumatic patients and sibling groups in both typical and atypical groups compared with the corresponding control. These results may be explained on the basis that elevation of ESR is most commonly associated with infection and extreme elevation of ESR in children may be used as sickness index, but not as screen tool for disease [15].

RF was the most common disease that causes elevation of ESR. Idress and AL Qahtani reported that children measured and adults in areas where RF is common, ESR >60mm/h may favor the diagnosis of RF [16].

Our results showed that there was positive correlation between ASOT and ESR and CRP. In contrast to Ben-Chetrit et al., in a study showed that there was no correlation between ASO serum levels, ESR and CRP [17].

As regard IMA, our study compared patients with controls in both typical and atypical groups and found that there was statistically high significant elevation of IMA in the patient groups as compared to control group in both typical group and atypical group with mean value (292.400pg/ml) (202.000pg/ml) respectively, with (p-value <0.001*) (p-value=0.001 *) respectively, there was significant increase in patients of atypical group as compared to their siblings with (p-value <0.001 *), there was significant increase in siblings of typical group and control siblings (p-value <0.001*), also there was significant increase in siblings of typical group and siblings of atypical group (p-value 0.001*).

These results were quite similar to the results of KarataŞ et al., who showed that in a previous study, the serum ischaemia-modified albumin levels of both acute rheumatic carditis and isolated arthri-

tis groups were found to be significantly higher when compared with controls **[18]**.

Also, these results were supported by the findings of Toker et al., who reported that IMA levels increased in patients with ARF at the time of diagnosis are positively associated with increasing level of inflammation. Also, serum IMA levels could be used as a follow-up marker just like acute inflammatory reactants for evaluating the efficacy of treatment in ARF **[19]**.

The pathogenesis of rheumatic valvular disease has been responsible for fibrosis that occurs at the end of the inflammatory process. Furthermore, the oxidative stress increases in inflammatory disease. In addition, previous studies have shown that oxidative stress has been found to increase in acute rheumatic fever and chronic rheumatic heart disease and it is also thought to have a role in the pathogenesis of rheumatic carditis **[20,21]**.

Ischaemia-modified albumin is considered to be a biochemical marker for ischaemic conditions [22].

Initially, ischaemia-modified albumin was shown to be the most promising biomarker for early detection of cardiac ischaemia **[22]**. However, in recent studies, it has been shown to increase in several chronic diseases such as diabetes mellitus, chronic liver disease, end-stage renal failure, obesity, hypercholesterolaemia, and advanced cancer [23].Therefore, increased ischaemia-modified albumin does not only show cardiac ischaemia. In addition, chronic hypoxia, inflammation, and increased oxidative stress are considered to change the structure of albumin, and eventually elevation of the ischaemia modified albumin level occurs [24].On the other hand, despite the fact that pathologic evidence of myocardial inflammation has been shown in acute rheumatic carditis, the fact that the level of cardiac troponin-I is not elevated in acute rheumatic carditis suggests that there is minimal or no myocytic necrosis in this setting [25].

The generation of reactive oxygen species can transiently modify the N-terminal region of albumin and produce an increase in the concentration of ischaemia modified albumin **[26]**.

More recently, increased oxidative stress in patients with acute rheumatic fever compared with healthy controls has been reported **[18]**.

Our results show that there was positive significant correlation between IMA level and the CRP, ASOT, ESR (1 st h and 2 nd) of patients.

These results were supported by the findings of Kaefer et al., who reported that there was positive correlation between ischaemia-modified albumin level and high sensitive C-reactive protein concentration in patients with type-2 diabetes mellitus and hypercholesterolaemia has been reported in several studies **[27]**.

These results were supported by the findings of Karatas¸ et al., who reported that there was positive correlation between acute phase reactants and serum ischaemia-modified albumin levels **[18]**.

And also, these results were supported by the findings of Toker et al., who reported that there was positive correlation between acute phase reactants and serum ischaemia-modified albumin levels **[19]**.

Our results showed that there was significant increase of IMA level in patients with blood group A+ with mean value 265.400pg/ml.

Our results also revealed that there was positive significant correlation between IMA level and the age of the patients due to increase the rate of URTI in agreement with **[28]**.

We found by screening of families siblings of typical group that five siblings from ten had organic heart diseases, (two of five had active rheumatic heart diseases and other three siblings had chronic heart diseases). There was no significant increase of IMA in siblings who had chronic rheumatic heart dieases but there was significant increase of IMA in siblings who had active rheumatic disease with mean (196.2pg/ml).

In atypical group, we found that two siblings had organic heart diseases and two had typical rheumatic arthritis from ten siblings, and there was no significant increase of IMA and acute phase reactants in these siblings as they had chronic rheumatic disease not active disease.

These results were supported by Karatas et al., **[18]** who reported that serum ischaemia-modified albumin and acute phase reactant levels of the patients with chronic rheumatic heart disease have been detected to be normal, although higher serum ischaemia-modified albumin and acute phase reactants levels have been demonstrated in patients with acute rheumatic fever.

The benefit of our study is "Early detection of RF in siblings of a case of RF with early management and prevention of RHD depending on positive family history of RF and elevating serum IMA".

Conclusion:

Our study concluded that serum IMA level and ASOT increased in children with ARF/RHD as well as their siblings which facilitate early detection and management of RF in siblings of a case of RF depending on positive family history and elevating anti-streptococcal antibodies and serum Ischemia modified albumin.

Recommendations:

Early detection of RF in siblings of a case of RF with early management and prevention of RHD depending on positive family history and elevating anti-streptococcal antibodies and serum ischemia modified albumin.

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نسبة الزلال المعدل نتيجة نقص التروية لدى أطفال العائلات التى لديها حالة مصابة بالحمى الروماتيزمية الحادة / أمراض القلب الروماتيزمية

تعد الحمى الروماتيزمية مرضاً يحدث بعد الاصابة بالبكتريا السبحية العقدية ويؤثر على الأطفال بالدرجة الأولى. وتشمل أعراضه حمى، ألم مفصلى، والغثيان والقيء. ويمكن أن يتسبب بحدوث تأثيرات طويلة الأمد فى الجلد والمفاصل والقلب والدماغ. تمثل الحمى الروماتيزمية وأمراض القلب الروماتيزمية السبب الرئيسى للوفاة والإعاقة بين أطفال لمدارس فى البلدان النامية. ومع ذلك، فإنه مرض من الأمراض المتكررة التى عادة ما يمكن منعها. وبعد الزلال المعدل نتيجة نقص التروية دلالة جديدة على نقص تروية الأنسجة، وقد تبين أن تكوين أنواع الأكسجين التفاعلية نتيجة نقص التروية أدى إلى تكوين الزلال المعدل نتيجة نقص التروية. يزيد فى نقص الأكسجين المزمن والإلتهابات والأكسدة وهذه الأكسدة تغير من تركيب الزلال وفى النهاية زيادة الزلال المعدل نتيجة نقص التروية.

المرضى وطرق البحث: لقد تم تنفيذ هذه الدراسة فى وحدة أمراض القلب بقسم طب الأطفال فى مستشفى جامعة طنطا. وتم إدراج ٢٠ عائلة فى الدراسة التى تم تقسيمها إلى ثلاث مجموعات بينهما ترابط فى العمر والجنس.

المجموعة الأولى: شملت العائلات التى لديها حالة بها علامة نمطية من الحمى الروماتيزمية الحادة أو أمراض القلب الزوماتيزمية وشملت الأطفال من سن ٥ إلى ١٥ سنة.

المجموعة الثانية: شملت العائلات التى لديها حالة بها علامة غير نمطية من الحمى الروماتيزمية الحادة أو أمراض القلب الزوماتيزمية وشملت الأطفال من سن ٥ إلى ١٥ سنة.

المجموعة الثالثة: شملت العائلات التى ليس لها أى تاريخ مرضى من الحمى الروماتيزمية أو بنسللين طويل المفعول قيدوا كمجموعة التحكم.

هذه الدراسة تمت فى عام واحد الهدف من هذا العمل هو قياس مستوى الزلال المعدل نتيجة نقص التروبة لدى العائلات التى لديها حالة مصابة بالحمى الروماتيزمية الحادة / أمراض القلب الروماتيزمية أى كان الصورة التى يظهر بها.

النتائج: إرتفاع نسبة الزلال المعدل نتيجة نقص التروية ومعيار الإستربتوليسين O لدى أطفال الذين يعانون من الحمى الروماتيزمية أو أمراض القلب الروماتيزمية وكذلك بعض أشقائهم، مما يسهل الكشف المبكر وعلاج الحمى الروماتيزمية فى أشقاء الحالات التى تعانى الحمى الروماتيزمية أعتماداً على التاريخ العائلى الإيجابى وإرتفاع مستوى الأجسام المضادة للاستروبتوليسين O وارتفاع نسبة الزلال المعدل نتيجة نقص التروية.

التوصيات: الكشف المبكر عن الحمى الروماتيزمية فى بعض أشقاء الحالة التى تعانى من الحمى الروماتيزمية مع العلاج المبكر والوقاية من أمراض القلب الروماتيزمية إعتمادا على تاريخ عائلى إيجابى وإرتفاع الأجسام المضادة للاستروبتوليسين O وإرتفاع نسبة الزلال المعدل نتيجة نقص التروبة.