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The role of the heart muscle in the hemodynamic problems caused by rheumatic fever

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ABSTRACT

Purpose and Background: In many underdeveloped nations, rheumatic fever is a leading cause of acquired cardiac disease in children and young adults. Using cardiac troponin test and echocardiography, the purpose was to study the impact of myocardial involvement on hemodynamic alterations in individuals with acute rheumatic fever. The research was planned as a prospective cohort. Subjects and Procedures: Twenty-two children had cardiac issues, fourteen did not, and thirty-four had severe rheumatic fever. The primary outcome measure was the level of cardiac troponin T (cTnT), which is a marker of left ventricular function measured by echocardiography. The amount of cardiac troponin in children suffering from carditis was 0.051 ± 0.01 ng/dL, whereas in those unafflicted, it was 0.039 ± 0.02 ng/dL. We can't say that the difference is meaningful. Furthermore, when it came to left ventricular ejection and shortening fractions, there was no significant difference between the children with and without carditis. Acute rheumatic fever does not cause myocardial involvement or clinically important hemodynamic abnormalities, according to the results of the echocardiographic tests and the rise of cTnT levels.

Key words: Heart troponin test, echocardiography, myocardial involvement, and acute rheumatic fever

INTRODUCTION

In children and young people across the world, rheumatic fever is the leading cause of acquired heart disease.[...] Despite documented decreases in the incidence of acute rheumatic fever and rheumatic heart diseases in industrialized countries over the last fifty years, these non-suppurative cardiovascular sequelae of group Streptococcal pharyngitis remain a medical and public health concern in both industrialized and industrializing nations. Tragically, the casualties are mostly young people, just when they are starting to make a positive impact on the world.Rheumatic carditis, sometimes called pancarditis, is characterized by inflammation of the endocardium, myocardium, and pericardium.Although there is evidence of interstitial myocardial infiltrations and Aschoff nodules in the pathological specimens, myocyte necrosis has not been detected, even if congestive heart failure is present. The identification of myocarditis relies on the presence of myocyte necrosis.cTnT, or cardiac troponin T, is a very sensitive and specific indicator of myocardial damage [4].[5] The heart's fragile actin filaments are controlled by proteins called cardiac troponins. Using troponin T and troponin I, myocardial injury may be diagnosed very specifically and sensitively. One way to find cardiac troponins in blood samples is to use monoclonal antibodies that bind to specific regions on the troponin T or I protein. These antibodies sit on the sidelines when it comes to skeletal muscle.When it comes to acute valvular sickness, heart failure, and chronic valve disease, the mortality and morbidityratesareattheirgreatest.

that are linked to rheumatic fever.When rheumatic fever impacts the heart, it often includes the endocardium, myocardium, and pericardium to varied degrees, according to revised diagnostic criteria for rheumatic fever [7]. According to these recommendations, rheumatic myocarditis is "uncommon" when there isn't significant damage to the valves, but it "may contribute" to the development of heart failure in cases of rheumatic fever.[8] But whether or not rheumatic fever causes heart failure due to a particular main myocardial involvement is debatable and requires moreresearch.

Using echocardiography and cTnT, a particular marker of myocardial injury, this research set out to examine the function of the myocardium in the hemodynamic alterations seen in individuals suffering from acute rheumatic fever.

SUBJECTSANDAPPROACHES

The Suez Canal University Hospital was the subject of a prospective cohort research.

Whatisrequiredtobeincluded Admitted children diagnosed with acute rheumatic fever using the modified Jones criteria.[9]Criteriaorxclusion This condition is characterized by rheumatic valve lesions that can recur or are persistent, as well as congenital heart defects. In order to determine whether patients met inclusion and exclusion criteria, we took their full medical histories and performed thorough physical examinations. For the purpose of measuring ESR, C-reactive proteins, ASOT, and cTnT, blood samples were taken from every patient. Each infant had electrocardiography and echocardiography. In accordance with the established hospital practice for rheumatic fever cases, the patients underwent routine therapy. If carditis was present or not, the patients were split into two categories. Clinical criteria for carditis include an enlarged PR interval on electrocardiogram (ECG), sleeping tachycardia that is disproportionate to temperature, the discovery of a new murmur of aortic or mitral regurgitation, or the diagnosis of heart failure. At the time of diagnosis, echocardiography verified this.The tenth

TestforcardiacroponinT Every kid had their blood sampled. The blood was separated by centrifugation and then refrigerated at -20 °C until the batch analysis could be done. The researchers used an immunoassay called the Elecsys Troponin T STAT from Roche Diagnostics GmbH in Mannheim, Germany, to conduct the biochemical study. As a standard range for cTnT, this electrochemiluminescent sandwich enzyme-linked immunosorbent test can detect concentrations as low as 0.01 ng/mLEvaluationusingechocardiography A 3.5-5 MHz transducer from Hewlett-Packard's Sonos 1800 series was used for the operation. All of the investigations made use of the following views: parasternal long and short axis, apical four chamber, subxiphoid four chamber, and suprasternal. The valvular regurgitation severity was assessed using a qualitative grading system ranging from 0 to 4. A score of 0 indicates no regurgitation, a 1 indicates mild to moderate regurgitation, a 3 indicates moderate to severe regurgitation, and a 4 indicatessevereregurgitation[10]. Dataanalysisusingstatistics We used SPSS, a statistical software, to evaluate the data. The features of the research group were laid out using descriptive statistics. The Student's t-test was used for comparing continuous data. We used a two-tailed c2 test to compare the categorical data. $P < 0.05$ was used to accept statisticalsignificance. Concernswiththics With the blessing of the ethics committee, the participants' families providedwrittenpermission.

Theoutcome Nineteen (56%) of the thirty-four children with ARF were girls and fifteen (44%) were boys. The average age among the participants in the research was 11.3 ± 2.65 years.

Fifteen children (59%) in the research group had symptoms of carditis, whereas fourteen (41% of the total) had ARF butnosuchsymptoms. Table 1 displays the demographics of the participants in the research. This table shows that when looking at age, sex, CRP, and ESR, there is no statistically significant difference between the two groups of children with and without carditis. In the group of patients with carditis, the PR interval on the electrocardiogram was longer (0.17 ± 0.03 s) than in the group without carditis (0.10 ± 0.05 s). There was a statistically significant difference. Furthermore, individuals with carditis had a much greater resting pulse rate.

Table 1: Clinical and biological characteristics of the study groups

	ARF with carditis, n = 20 (59%)	ARF without carditis, n = 14 (41%)	*P*
Age (years)	11.5 ± 4.6	10.9 ± 5.3	0.374
Sex			
Female	12	8	0.083
Male	8	6	
CRP (mg/dL)	6.5 ± 5.2	7.1 ± 4.9	0.153
ESR (mm/h)	69 ± 21.3	58 ± 26.8	0.215
PR (s)	0.17 ± 0.03	0.10 ± 0.05	0.021*
Heart rate (b/min)	109.3 ± 15.7	89.6 ± 19.4	0.013*

**Significant difference.*

Table 2: Distribution of valvular lesions among children with carditis

	Grade 1 (trivial to mild)	Grade 2 (moderate)	Grade 3 (moderate to severe)	Grade 4 (severe)
MR (n)	4	7	4	0
AR (n)	1	1	0	0
MR + AR (n)	0	3	0	0

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MR: Mitral regurgitation; AR: Aortic regurgitation; N: Number of children.(109.3 ± 15.7 beats/min), compared to those withoutcarditis (89.6 ± 19.4 beats/min).

The distribution of valvular lesions in children with carditis

Table 3: Cardiac troponin T level in the study groups

	ARF with carditis, *n* = 20 (59%)	ARF without carditis, *n* = 14 (41%)	*P*
Troponin T(ng/dL)	0.051 ± 0.01	0.039 ± 0.02	0.089

is shown in Table 2. Fifteen children had isolated mitralregurgitation (four children grade 1, seven children grade 2, and four children grade 3). Another three patients had both mitral and aortic regurgitation (grade 2). Isolatedaortic regurgitation was present in two children (one child rade 1, and one child grade 2). Among the 20 childrenwith carditis, heart failure was present in five children withmitral regurgitation.

Table 4: Functional echocardiography data

	ARF with carditis *n* = 20 (59%)	ARF without carditis *n* = 14 (41%)	*P*
Ejection fraction (%)	59.2 ± 7.3	65.6 ± 6.4	0.087
Fractional shortening (%)	31.8 ± 6.2	37.5 ± 6.4	0.216

Table 3 shows the cTnT level in both groups of children with ARF. The mean level of cTnT in patients with carditisis 0.051 ± 0.01 ng/mL, while the mean level of cTnT in patients without carditis is 0.039 ± 0.02 ng/mL. The difference is not significant ($P > 0.05$).

Table 4 shows the echocardiographic measurements in thetwo groups of children with ARF. We can see from thistable In individuals suffering from carditis, the left ventricular ejection fraction measures 59.2 ± 7.3%, whereas in people free of carditis, it is 65.6 ± 6.4%. Statistically, the change is not noteworthy ($P > 0.05$). The fractional shortening was 31.8 ± 6.2% in the group of children with carditis and 37.5 ± 6.4% in the group without carditis, although there was no significant difference between the two groups when it came to this measure ($P > 0.05$).

Talkingout

The importance of myocarditis in children with acute rheumatic carditis is still debatable, even if there is histologic evidence of inflammation in the heart muscle.the eleventh Though rare, rheumatic myocarditis may have had a role in the development of heart failure in cases of rheumatic fever, according to the criteria for the diagnosis of the disease. This would be the case in cases where there was no significant damage to the valves.[8]

There were no changes in biochemical parameters or echocardiographic data that would indicate substantial hemodynamic consequences owing to myocardial involvement in this investigation. The level of C-reactive protein did not vary significantly between individuals with and without rheumatic heart disease. Also, even among individuals suffering from heart failure, every single one had cTnT levels that were well within the normal range. As a sensitive and specific sign of even preclinical myocardial damage, cardiac troponin T has emerged as one of the new "gold indicators" of ischemic myocardial injury. Myocarditis and other forms of nonischemic myocardial damagearealsomonitoredanddiagnosedwithitshelp.[12]

Furthermore, there was no statistically significant difference in left ventricular ejection or shortening fractions between the groups of children with and without this condition, according to the echocardiographic investigation.

the presence or absence of congestive heart failure in cases with rheumatic carditis. In children with and without carditis, our findings are consistent with those of Tavli et al.,[13] who demonstrated that cTnI levels are not raised

during acute rheumatic fever. Furthermore, in their investigation of pediatric rheumatic fever, they could not find any echocardiographic indicators of cardiac involvement. In addition to not finding any notable change in function owing to myocarditis using echocardiography, Alehan et al.[14] showed that serum cTnT concentration did not rise beyond normal limits in rheumatic carditis with or without heart failure. Despite the presence of congestive heart failure, Kamblock et al.[15] found no cTnI elevations or echocardiographic abnormalities in 95 children with ARF, indicating that rheumatic fever does not cause significant hemodynamic changes owing to myocardial involvement. Severe valve regurgitation, and never myocarditis, was the underlying cause of congestive heart failure. Williams et al. [4] also found no increase in serum cTnI in individuals with acute rheumatic fever, supporting their findings. Children with acute rheumatic carditis do not have high cardiac troponin I levels in their blood, which suggests that thereislimitedmyocytenecrosisinthiscase,accordingtotheirstatement.

Significant ischemic myocyte damage is cast into doubt by the low cTnT levels, which are particularly noticeable in cases with active carditis.[16] Even if there is a lot of inflammation in ARF, the myocardial necrosis is not noticeable.[17] The most important component of ARF, which is mostly a connective tissue disease, is endocarditis.[18] in Patients with ARF carditis may have normal cTnT concentrations because of this. The histological characteristic of rheumatic fever, known as Aschoff nodules, may be seen in the ventricular myocardium and left atrial appendages of individuals with rheumatic fever and rheumatic heart disease.The year 19 In the early stages of congestive heart failure, the appearance of Aschoff nodules in the ventricular myocardium doesnotalwaysindicatetheirinvolvementinthedisease'sdevelopment.[8]

The results show that even in individuals with severe congestive heart failure and carditis, acute rheumatic fever did not cause a rise in cTnT levels that would indicate clinically significant myocardial involvement. We also did not find any statistically significant differences in echocardiographic parameters between the carditis and non-carditis groups of patients. These results provide credence to the idea that involvement of the myocardium during acute rheumatic fever does not contribute to the onset of heart failure in rheumatic carditis patients or have any noticeable hemodynamic repercussions. The severity of valve abnormalities, not cardiac dysfunction, is the primarydeterminantofheartfailureseverity.

takingpartNevertheless, a rather small sample size is one of the study's drawbacks. To demonstrate that myocardial involvement during acute rheumatic fever does not have a significant role in the development of heart failure in individualswithrheumaticfever,moreresearchareecessary.

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