SCIENTIFIC LETTER

Role of serum cardiac troponin T in the diagnosis of acute rheumatic fever and rheumatic carditis

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The incidence of acute rheumatic fever (ARF) has declined in industrialised countries since the 1950s. In developing countries, it remains an endemic disease of school aged children and is a major cause of cardiovascular morbidity and mortality. Cardiac troponin T (cTnT) is a highly sensitive and specific marker of cardiomyocyte damage.[1] In this study, we attempted to assess the role of serum cTnT values in the documentation of cardiac damage in ARF patients with and without carditis.

PATIENTS AND METHOD

Forty six consecutive patients (29 males) with ARF diagnosed (according to the modified Jones' criteria) within two years, were prospectively studied. A new murmur of aortic or mitral regurgitation was considered as clinical evidence of carditis. This was confirmed by echocardiography at the time of the diagnosis. We used the previously established Doppler echocardiographic guidelines to define pathological mitral and aortic insufficiency.[2] We also measured left ventricular systolic and diastolic diameters and fractional shortening in parasternal long axis position with M mode echocardiography. Serum cTnT concentrations were determined by using the third generation Elecsys Troponin T STAT immunoassay (Roche Diagnostics Mannheim, Germany), standardised with human recombinant cTnT. The lower detection limit of the assay is 0.01 ng/ml and the normal range for cTnT is 0.01 to 0.1 ng/ml. Serum creatine kinase isoenzyme MB (CK-MB) activity level was measured by routine laboratory assays, which have an upper reference limit of 5 ng/ml. Troponin T and CK-MB assessments are not standard components of the evaluation of suspected ARF at our institution. Data are compared between groups using independent sample *t* test. Probability values of $p < 0.05$ were considered significant.

RESULTS

The mean (SD) age of the study group was 10.92 (2.42) years (range 7–16 years; median 11 years). The major findings of Jones' criteria were chorea and carditis in two patients (4.3%), carditis alone in nine patients (19.6%), arthritis with carditis in 23 patients (50%), and arthritis alone in 12 patients (26%).

Nineteen patients with carditis had mitral regurgitation, one patient had aortic regurgitation, and 14 patients had both mitral and aortic regurgitations. Among 33 patients with mitral regurgitation, one had severe regurgitation, seven patients had moderate regurgitation, and the remaining had mild regurgitation. Aortic regurgitation was diagnosed in 15 patients, of which 10 were mild and five were moderate regurgitations. All patients, except two, had normal left ventricular fractional shortening on echocardiography. The fractional shortenings of the left ventricle in these two patients were 27% and 24%. The latter patient also had mild pericardial effusion as a result of pericarditis accompanying moderate mitral regurgitation, while the former one had moderate aortic and mitral regurgitation.

Table 1 The echocardiographic measurements of serum troponin and creatine phosphokinase isoenzyme MB concentrations in patients with acute rheumatic fever

	No carditis	Carditis	p Value
Ejection fraction (%)	73.6 (6.4)	70.8 (6.1)	>0.05
Fractional shortening (%)	41.5 (6.7)	40.0 (5.3)	>0.05
Heart rate (beats/min)	92.0 (17.4)	86.5 (13.2)	>0.05
CK-MB (ng/dl)	1.39 (1.22)	1.46 (1.12)	>0.05
Troponin T (ng/dl)	0.0 (0.0)	0.038 (0.22)*	>0.05

CK-MB, creatine phosphokinase isoenzyme MB
*Only one patient had a detectable troponin T value (1.30 ng/dl).

The mean (SD) CK-MB was 1.44 (1.14) ng/ml (range 0.26–5.16 ng/ml) and mean cTnT concentration was 0.028 (0.19) ng/ml (range 0.0–1.30 ng/ml) in the study group. The differences of CK-MB and cTnT values were insignificant between patients with and without carditis ($p > 0.05$) (table 1). Only one patient had an abnormal CK-MB value of 5.16 ng/ml. This patient had a typical mitral regurgitation murmur with moderate regurgitation detected on echocardiography. The left ventricular systolic function was normal. Another patient with mild mitral regurgitation and normal left ventricular systolic function had a cTnT value of 1.3 ng/ml, which was above the normal limit. The troponin concentrations were undetectable in all the other patients.

DISCUSSION

This study, with the largest patient population reported to date, demonstrates that there is no significant elevation of cTnT in ARF patients with or without carditis. According to the guidelines of National Academy of Clinical Biochemistry and International Federation of Clinical Chemistry, cTnT is considered as one of the new ''gold markers'' of ischaemic myocardial injury. It is also used in the diagnosis and monitoring of non-ischaemic myocardial injury, like myocarditis, cardiac contusion, chemotherapy, cardiac catheterisation, or radiofrequency ablation and has been detected as a sensitive and specific marker of even subclinical myocardial injury.[1 3] This study used the third generation cTnT assay, which is the most sensitive and specific assay currently available with a low detection threshold of 0.01 ng/ml. Despite this, only one ARF patient had elevated cTnT values and only one patient had elevated values of CK-MB. The differences in CK-MB and troponin concentrations among patients with or without carditis were not significant ($p > 0.05$). Gupta and colleagues[4] have measured serial cardiac troponin I in the sera of ARF patients. They found a minimal degree of elevation in cardiac troponin I above

Abbreviations: ARF, acute rheumatic fever; cTnT, cardiac troponin T; CK-MB, creatine kinase isoenzyme MB

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normal values in 18% of the patients with carditis. Similar to our study, among their patients with ARF, there was no significant difference in peak values between those who had carditis and those who did not.[4]

It is well known that elevation of cardiac troponin is greater in ischaemic injury (such as myocardial infarction), but it is less in non-ischaemic injury (such as myocarditis).[1 3] The low cTnT values, especially in the presence of active carditis, disputes significant ischaemic myocyte injury.[4] Myocardial necrosis is not prominent despite intensive inflammation in ARF. This is supported by lack of myocardial necrosis observed in biopsy specimens of patients with ARF carditis.[5] ARF is mainly a disease of connective tissue and endocarditis is the prominent factor. This may explain the normal concentrations of cTnT found in patients with ARF carditis. The mild elevation of troponin found in one of our patients may be the result of an inflammatory process with minimal damage to myocardial cells. None of our patients with carditis had heart failure, which probably indicates only a slight cardiac involvement, and may also explain the low serum concentrations of cTnT. Our results are in agreement with the study of Williams and colleagues,[6] who did not observe elevation of troponin values in patients with carditis. Furthermore, more than one third of their patients had symptoms of congestive heart failure.

In conclusion, serum cTnT concentration does not increase above normal limits in rheumatic carditis, probably because of the less destructive nature of rheumatic carditis on the myocyte. Therefore it has limited value in the diagnosis or the prognosis of rheumatic carditis. Whether slight, but significant, increases of cTnT may be observed in rheumatic carditis will be a subject of further investigation.

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