Cardio specific Troponin I in patients with Aortic stenosis

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Abstract

To evaluate the association of Cardio specific Troponin I (CTnI) and aortic stenosis. Cross sectional study was conducted among 20 aortic stenosis patients and 20 control groups. A structured questioner and checklist was used to collect data through face to face interview, Echocardiography findings, and laboratory estimation of cTnI. A total 20 patients and 20 healthy control subjects were investigated for cTnI. cTnI level was within normal physiological limits in all the control subjects. The mean value was 0.02±00. The mean cTnI level in Aortic stenosis patients were 0.67±0.81. EJection fraction of Aortic stenosis patients were normal, indicating that cases yet not develops complication (eg; Heart failure). The cTnI in control group and Aortic stenosis patients shows significant difference of mean (p<0.001). cTnI level in Aortic stenosis patients increases in absence of heart failure indicating that it can expose cardiomyocyte injury prior to the development of over Left ventricular dysfunction. So that, serial monitoring of cTnI could help clinician to give definitive treatment before development of complications.

Keywords: *Cardiospecific Troporin I. Aortic Stenosis.*

Introduction

Valvular heart disease is a common cardiac disability in our country. The valvular heart disease which we face in our day to day hospital & private practice is due to chronic sequilae of rheumatic fever. In chronic rheumatic heart disease mitral valve is affected in more than 90% of cases and aortic valve is the next most frequently affected valve (Bloomfield et al., 2002).

In aortic stenosis there occurs obstruction to left ventricular our flow during systole. Pressure gradient across the aortic valve (pressure higher in Left ventricle than aorta during systole), causes chronic left ventricular pressure overload and compensatory Left ventricular hypertrophy. Increased wall thickness and decreased cavity size oppose the increased wall stress. The Left ventricular hypertrophy initially leads to diastolic dysfunction and lettter on systolic dysfunction. In the late stage, LV systolic function can be reduced as a result of myocardial fibrosis (Rediberg et al., 2004). The sequence of cardiac decomposition begins with ventricular dilatation, which further raises wall stress, leading to increased Left ventricular hypertrophy and probably reduced blood flow to the hypertrophied myocardium, in term leading to ischemia with reduced LVEF. Furthermore, aortic stenosis is itself associated with 50% increased risk of cardiovascular mortality and Myocardial infarction (otto et al., 1999).

Lt. ventricular systolic dysfunction and heart failure predict poor prognosis including a less favorable outcome after valve replacement in aortic stenosis. The onset of heart failure proceeded by structural & functional alterations in the heart muscle with degeneration and death of the cardiac myocytes (Hein et al 2003). Disease of the heart valves may progress with time and selected patients require regular review, usually every 1 or 2 years, to ensure that deterioration is detected before complication such as heart failure ensue (Bloomfield et al., 2002). Detection of ongoing myocardial injury before the outbreak of overt Left ventricular dysfunction could help promote earlier surgery in patients without symptom or with vague symptom.

Troponin, a protein molecule that plays an essential role in the contraction of the striated muscle. cTnI has been established as reliable and highly heart specific markers of myocytes injury. Their background concentrations in the circulation are normally undetectable or very low and they are therefore sensitive to even minor heart muscle

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damage. Measuring circulating cardiac Troponin-I would help expose ongoing silent myocytes damage in Aortic stenosis (Kupari et al., 2005). Elevated CTnI even in the absence of heart failure indicating that it can expose cardiomyocyte injury prior to the development of overt Left ventricular dysfunction. Serial monitoring of cTnI during follow up of asymptomatic Aortic stenosis will show whether cTnI can assist in the timing of therapeutic interventions.

The aim of the study was to explore the relation ship of serum cTnI and Aortic stenosis. So that it will help in the detection of ongoing myocardial injury in aortic stenosis patient before the outbreak of overt Left ventricular dysfunction which will help to promote earlier surgery in patients without symptom or with vague symptom.

Patients and Methods

This cross sectional study was conduction for a period of 6 months (July 2005- June 2006). 20 patients (2 male and 18 female) between the age ranges of 5-60 years were selected having Aortic stenosis that was confirmed by Echocardiography. All the patients came to the outpatient department of NICVD with mild symptoms such as- palpitation, chest pain, and exertional dyspneoa. Ischemic heart disease was excluded by Echocardiography. 20 control subjects (3 male male and 17 female) between the age ranges of 5-60 years were selected randomly. A blood sample for determination of cTnI was obtained by vein puncture. Micro particle Enzyme immuno assay was done for the quantitative determination of cTnI.

Data are presented as mean±SD. Mann Whitney U test was done as the test of significance. A probability value of P<0.05 was considered significant. All calculations were done with SPSS system 10.0.

Result

cTnI level was within normal physiological limits in all the control subjects. The mean value was 0.02±0.00. Among the patients of Aortic stenosis cTnI was undetectable in two patients, in two patients there was detectable cTnI but it was within normal physiological limits, and another sixteen patients had elevated levels of cTnI. The mean cTnI level in aortic stenosis patients was 0.67±0.81. cTnI level among Aortic stenosis patient and control subjects shows significant difference of mean (p<0.001).

Table : Ejection fraction and cTnI conc of study subjectsTable:

Group	Ejection fraction (Mean ±SD)	cTnI (ng/ml) (Mean ±SD)
Aortic stenosis patients	63.10 ±6.33	0.67 ±0.81
control	65.25 ±3.26	0.02 ±0.00

The normal ejection fraction is 55%. The mean ejection fraction of Aortic stenosis patients and control subjects were 63.10±6.33% and 65.25±3.26% respectively, having no significant difference between the groups. This indicates that the Aortic stenosis patients yet not develops complication, such as heart failure (as shown by lowered ejection fraction).

Discussion

It is evident from the findings of our present study that there is a significant higher level of serum cTnI in Aortic stenosis patients with normal ejection fraction. The survival rates of the patients with asymptomatic aortic stenosis patients are nearly normal, until the symptoms of angina, syncope or heart failure develops. The presence of symptoms of heart failure in patients of Aortic stenosis causes a bad prognosis within a short period of time (mean survival<2 years) (Park et al., 2001). We found that cTnI elevated in the absence of heart failure indicating that it can expose cardiomyocyte injury prior to the development of overt LV dysfunction. So that, serial monitoring of cTnI could help clinicians to give definitive treatment before development of complications.

Cardiac Troponin I have been established as reliable and highly heart specific markers of myocytes injury. Their background concentrations in the circulation are normally undetectable or very low (normal level is 0.00-0.05ng/ml) and they are therefore sensitive to even minor heart muscle damage (kupari et al., 2005). The triggers of cardiomyocyte death in Aortic stenosis have not been detailed, the possible pathophysiology is that-in Aortic stenosis there occur narrowing of aortic valve which causes decreased blood flow from the Lt. Ventricle to the aorta. This increases workload of the Lt. Ventricle. This forces the left ventricle to squeeze harder, as a result the walls become thicker in time and left ventricular hypertrophy develops. The hypertrophied Lt. ventricular muscle mass elevates myocardial oxygen requirements. Even in the absence of obstructive coronary artery disease there may be interference with coronary blood flow. This is because of the compression of the coronary arteries by the hypertrophied myocardium. The coronary arteries

that give nutrition to the heart muscle with blood and oxygen arise just valve may not get enough blood to adequately supply the heart muscle (Myo foundation 2005). This in turn, results in the relative ischemia of the Lt. ventricular myocardium and consequently death of the cardiac myocytes and causes release of cTnI. Whatever their mechanisms, injury and death of the cardiac myocytes ultimately leads to the development of depressed systolic function and heart failure (Kupari et al., 2005). However, it is noted that circulating cTnI was undetectable in two of our patients with severe AS. This suggests that myocytes injury can be intermittent and may therefore escape detection)

An important limitation of our work is that we have financial constrain in serial measurement of cTnI and LV function in our pationts. Therefore True predictive value of circulating CTnI could not be studied. Only serial monitoring of CTnI during follow up of asymphomatic Aortic Stenosis will show whether cTnI can assist in the timing of therapeutic interventions.

From our present study findings we can conclude that circulating cTnI concentrations are frequency detectable and elevated in patients with severe Aortic stenosis even in the absence of heart failure. Circulating cTnI deserves a potential warning signal in patients with server but still asymptomatic Aortic stenosis.

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