Cardiac contusion in blunt chest trauma: a combined study of transesophageal echocardiography and cardiac troponin I determination

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Cardiac troponin; Myocardial contusion; Transesophageal echocardiography.

Background. **The role of cardiac troponin I (cTnI) is well established in acute myocardial ischemia. However, its role in myocardial contusion remains to be clarified. Since transesophageal echocardiography (TEE) appears, at present, to be the best method for the diagnosis of myocardial contusion, the aim of this study was to measure the concentration of cTnI in patients with blunt chest trauma studied using TEE.**

Methods. **Thirty-two patients (27 males, 5 females, mean age 44 ± 20 years), admitted to the Trauma Center of our Institution with clinical and/or radiological signs of acute blunt chest trauma, underwent biplane TEE within 24 hours of injury; serial blood samples were taken to measure cTnI levels (normal values < 0.4 ng/ml), using fluorimetric enzyme immunoassay.**

Results. **Abnormal levels of cTnI were found in 17 patients (53%): 7 patients had levels of cTnI between 0.4 and 1 ng/ml, whereas 10 patients had levels > 1 ng/ml. Segmental wall motion abnormalities consistent with myocardial contusion could be identified by echocardiography in 6/10 patients with cTnI levels > 1 ng/ml (60%) but in no patients with normal cTnI levels or with titers between 0.4 and 1 ng/ml; mean cTnI levels showed a significant difference between the two groups of patients with and without echocardiographic signs of myocardial contusion (2.6 ± 1.6 vs 0.6 ± 1.4 ng/ml, p < 0.001).**

Conclusions. **Abnormal titers of cTnI suggesting myocardial contusion may be found in more than half of patients with blunt chest trauma; however, myocardial injury can be detected by TEE only for cTnI levels > 1 ng/ml; cTnI concentrations ranging between 0.4 and 1 ng/ml might be indicative of myocardial microlesions, not detectable by echocardiography, even if TEE is used; cTnI assay could therefore be suggested as a screening test before performing TEE after blunt chest trauma.**

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Introduction

Myocardial contusion has been reported in 0 to 63% of patients with blunt chest trauma depending on the criteria adopted for establishing the diagnosis[1,2]. The clinical spectrum ranges from isolated arrhythmias to severe consequences such as cardiac failure, ventricular aneurysm formation and/or cardiac rupture[3-12]. The potential need for subsequent general anesthesia, for heart transplantation (donor status) and resultant legal controversies require that any possible form of cardiac damage be ruled out. A correct diagnosis of myocardial contusion also appears to be important in patients without significant complications because the prognostic significance of cardiac injury is not clear, particularly in young patients.

Difficulties in the detection of myocardial contusion in these patients are well known: whereas transthoracic echocardiography (TTE) has several technical limitations, transesophageal echocardiography (TEE) is currently considered one of the best techniques for the evaluation of patients with chest trauma[13-24]. Elevated blood levels of cardiac troponin I (cTnI) have been indicated as a specific marker of cardiac contusion and may offer a new and reliable diagnostic tool that could help the clinician in decision making[25,26]. Since we have been unable to find any report, in the Italian medical literature, on this subject (MEDLINE source 1986-1999) and because the correlation between TEE and cTnI levels in patients with suspected myocardial contusion has not yet been studied, we report the results

of a series of patients who had blunt chest trauma and were studied both by determination of cTnI levels and combined TTE and TEE.

Methods

Over an 18 month period, from January 1998 to June 1999, a prospective study including victims of blunt chest trauma admitted to the Trauma Center of Careggi Hospital (Florence, Italy) was performed. Patients were eligible if they had clinical and/or radiological signs of acute blunt chest trauma: chest pain, subcutaneous emphysema, anterior chest wall contusions; rib, sternal or clavicular fractures, radiographic signs of pulmonary contusion, hemothorax or pneumothorax.

Only those patients in whom a complete echocardiographic study (TTE and TEE) was performed within 24 hours of injury were considered. Patients with a previous history of cardiac disease were excluded; particular attention was paid to exclude a history of coronary heart disease.

Baseline data of these patients included the Glasgow coma score (GCS), the injury severity score (ISS), the vital signs at admission, the mechanism of injury, and operative procedures. Immediately after admission, all patients underwent an electrocardiogram and an echocardiogram. All echocardiograms were performed with a Toshiba SSH 140 (Tokyo, Japan) or an Acuson 128 XP (Mountain View, CA, USA) ultrasound imaging system; TTE was performed with a 2.5 or 3.5 MHz probe, whereas TEE was performed with a biplane 5 MHz probe. Patients first underwent TTE including a complete time-motion, two-dimensional and color Doppler study; images were obtained as recommended by the American Society of Echocardiography[27,28], using all possible views (parasternal, apical, subcostal and suprasternal). The quality of the images was judged as good, sufficient, poor or insufficient.

TEE was performed by an experienced operator[29,30] in the emergency department or in the intensive care unit, possibly after informed consent. Twenty-three patients were examined during oro-tracheal intubation. Unless the patient had already been sedated prior to oro-tracheal intubation, introduction of the probe was accomplished after intravenous administration of a benzodiazepine. TEE evaluation was performed in order to evaluate myocardial contusion and other possible lesions such as valve dysfunction, pericardial effusions and aortic hematoma or rupture.

None of the echocardiographic studies delayed ongoing evaluation or treatment, as many echoes were performed while patients were undergoing other diagnostic or therapeutic procedures.

Regional left and right ventricular wall motion abnormalities were investigated. The prospective criteria to diagnose myocardial contusion were the presence of a segmental wall motion abnormality which resolved at subsequent echocardiography or a fixed segmental asynergy associated with the development of new Q waves at electrocardiography. Attention was focused on the wall motion of the right ventricular antero-apical wall and left ventricular apex[16]. All echocardiographic images were interpreted by a cardiologist expert in echocardiography who was unaware of the biochemical results.

Measurement of cardiac troponin I and of the MB isoenzyme of creatine kinase. Blood samples were taken to measure total creatine kinase (CK), its MB isoenzyme (CK-MB), and cTnI levels. Samples were taken 6, 12, 24, 48 and 96 hours after the trauma. Each blood sample, obtained at the reported intervals after chest injury, was collected in evacuated tubes without additives. The samples were immediately centrifuged at 2000 g for 15 min and measurement of cardiac markers was performed. CK-MB and cTnI assays were performed by one of the investigators (A.O.), a physician specializing in clinical chemistry, unaware of the clinical and echocardiographic data.

cTnI was measured, using the Baxter Stratus II analyzer, by means of fluorimetric enzyme immunoassay (Dade, Miami, FL, USA). This technique uses two different antibodies that recognize two different epitopes of protein and show no detectable cross-reactivity with skeletal muscle troponin I. In serum specimens from a group of 50 healthy individuals with no evidence of cardiac disease, we found that the cTnI concentration was below the minimal concentration detectable by the assay or 0.35 ng/ml (the lowest concentration that can be distinguished from zero). CK-MB mass assay was performed using the Stratus II instrument and employing an immunoassay technique (Dade, Miami, FL, USA). The upper reference limit was ≥ 6.0 ng/ml.

Statistical analysis. Data are reported as mean values ± SD. Statistical comparison to assess the differences in outcome between groups was performed using the unpaired Student's t test and the Fisher's exact test. A p value < 0.05 was considered to be statistically significant.

Results

In accordance with our criteria, 32 consecutive patients (27 males, 5 females, mean age 44 ± 20 years) were enrolled. The reason for blunt chest trauma was a motor vehicle accident in 29 and an accidental fall in 3 patients.

Echocardiography feasibility. Due to the poor acoustic window, TTE was judged insufficient in 12 out of 32 patients (36%); in 14 of the 20 remaining patients, the subcostal view was the best ultrasound window. TEE was successfully performed in all patients but one, due to the presence of cervical trauma with severe vertebral lesions

which did not allow introduction and adequate manipulation of the probe. The feasibility of TEE (97%) was significantly higher than that of TTE ($p < 0.002$).

Segmental wall motion abnormalities consistent with myocardial contusion could be identified in 6 patients (18.5%): in 1 patient at both TTE and TEE, in 4 at TEE only and at TTE in the patient with severe cervical trauma, in whom TEE was not performed. Asynergy was observed in the free wall of the left ventricle in 3 patients, in the interventricular septum in 1 and in the free wall of the right ventricle in 2 patients. Among the remaining 26 patients, in whom the transthoracic and transesophageal echocardiograms were negative for myocardial contusion, mild pericardial effusion was found in 3 patients, pleural effusion in 8 and a mediastinal hematoma in 2; no injuries were detected in the remaining 13 patients.

Age, sex, cause of trauma and thoracic lesions did not significantly differ between patients with and without echocardiographic signs of myocardial contusion (Table I). In patients with myocardial contusion a tendency towards a greater prevalence of vertebral lesions was observed. Electrocardiography showed no specific ST-T changes in 5 patients: 2 of them also had echocardiographic signs of myocardial contusion.

Comparison of echocardiographic findings with biochemical markers. Fifteen patients had normal blood levels of cTnI (< 0.4 ng/ml); none of them had echocardiographic findings suggesting myocardial contusion. As progressively higher levels of cTnI seem to indicate a large amount of injured myocardium[31], patients with abnormal values of cTnI were divided into two subgroups: in 7 patients cTnI levels between 0.4 and 1 ng/ml were found; this group also had echocardiograms negative for myocardial contusion; 6 out of 10 patients with levels > 1 ng/ml had an echocardiogram positive for myocardial contusion (Table II, Fig. 1).

Mean cTnI levels were significantly higher in patients with echocardiographic signs of myocardial contusion (2.6 ± 1.6 vs 0.6 ± 1.4 ng/ml, $p < 0.001$) (Fig. 1), whereas the difference in CK-MB levels was not statistically significant (9.5 ± 6.4 vs 10.8 ± 9.6 ng/ml, $p = 0.77$).

Analysis of the temporal curve obtained with different samples showed that cTnI levels reached a peak after 12 hours, whereas the increase disappeared after 48 hours (Fig. 2). Elevated concentrations of CK-MB (> 6 ng/ml) were associated with abnormal cTnI levels in 17 patients, but with normal titers in 8 other patients.

In the acute phase no patient had relevant arrhythmias or hemodynamic instability due to cardiac injury.

During the hospital stay 2 of the 6 patients with myocardial contusion died of cerebral lesions; in 3, echocardiographic abnormalities resolved whereas left ventricular wall motion abnormalities remained unchanged in 1 patient.

Discussion

Myocardial damage is often found at autopsy of patients dying from blunt multisystem trauma including chest injuries[32-38]. Yet, the detection of lesser degrees of cardiac injury has been hampered by the lack of accu-

Table I. Demographic and clinical characteristics of patients with and without echocardiographic signs of myocardial contusion.

	MC+ (n = 6)	MC- (n = 26)	p
Age (years)	42 ± 17	44 ± 21	0.85
Sex (male)	6/6 (100%)	19/26 (79%)	0.55
Motor vehicle accident	6/6 (100%)	21/26 (81%)	0.40
Abdominal lesions	1/6 (17%)	3/26 (12%)	1.00
Limb lesions	2/6 (33%)	15/26 (58%)	0.38
Rib fractures	2/6 (33%)	15/26 (58%)	0.38
Pulmonary contusion	5/6 (83%)	13/26 (50%)	0.19
Pleural effusion	5/6 (83%)	16/26 (62%)	0.64
Vertebral lesions	3/6 (50%)	2/26 (8%)	0.034

MC+ = echo positive for myocardial contusion; MC- = echo negative for myocardial contusion.

Table II. Comparison between echocardiographic results and blood levels of cardiac troponin I.

Cardiac troponin I	> 1 ng/ml	0.4-1 ng/ml	< 0.4 ng/ml	Total
MC+	6	0	0	6
MC-	4	7	15	26
Total	10	7	15	32

Abbreviations as in table I.

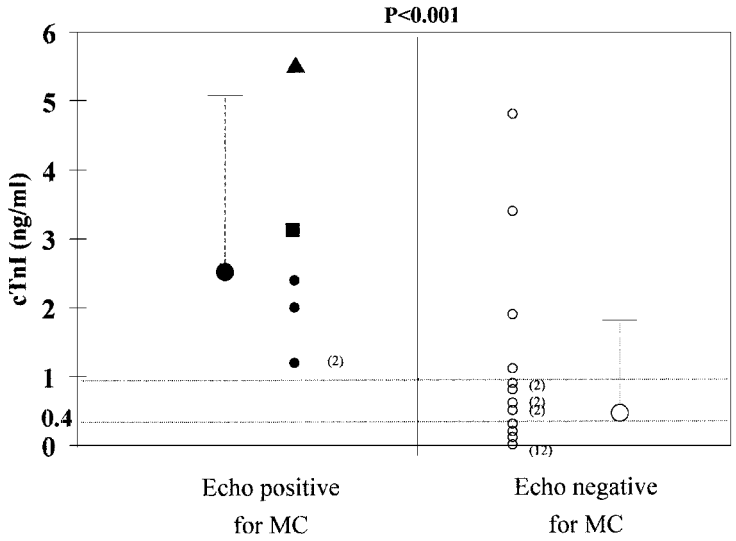

Figure 1. *Values of cardiac troponin I (cTnI) in patients with and without echocardiographic findings of myocardial contusion (MC).* Solid dots *indicate patients with echocardiographic signs of MC;* solid circles *indicate patients with only transesophageal echocardiography positive for MC; the* solid triangle *indicates the patient with transthoracic echocardiography positive for MC and the* solid square dot *indicates the patient in whom both transesophageal and transthoracic echocardiography showed signs of MC. Mean values of the two groups are shown by* large dots. *Numbers between brackets show patients with the same level of cTnI.*

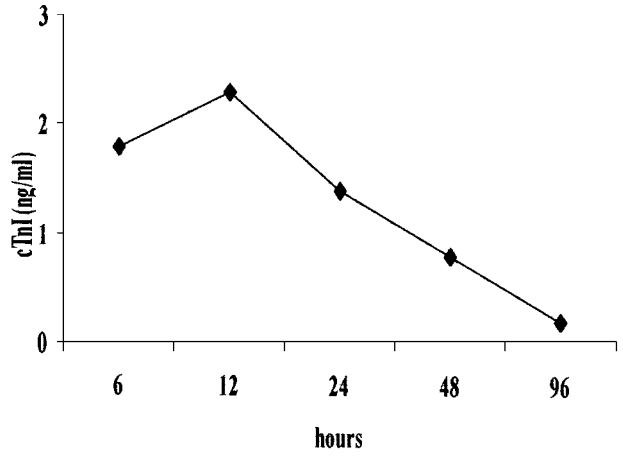

Figure 2. *Temporal curve of cardiac troponin I (cTnI): mean levels of cTnI in samples obtained 6, 12, 24, 48 and 96 hours after blunt chest trauma are reported. Peak values are observed 12 hours after trauma.*

rate diagnostic criteria. Clinical symptoms such as chest pain and/or dyspnea are not specific and often present in patients with chest trauma, even without cardiac injury. Also, electrocardiographic abnormalities, even if frequently observed in these patients, are not specific[33,39,40], and the electrocardiogram may be normal, particularly when myocardial contusion is limited to the right ventricle. The role of radioisotopic methods (radionuclide ventriculography, Tc99m pyrophosphate and thallium perfusion scanning) is also uncertain[15,34,35,41].

Echocardiography appears to be superior to other noninvasive tests in diagnosing myocardial contusion[13-16,18,42,43]. The main limitation consists of the difficulty in obtaining optimal images when a transthoracic approach is used in patients with severe chest injury. In the present study, 36% of patients had poor-quality TTE examinations. Our findings are similar to those of previous studies in which the percentage of suboptimal examinations of the patients with blunt chest trauma ranged from 13 to 28%[15,16,42]. TEE, allowing cardiac imaging from the esophagus, has proved to be clearly superior to TTE in these patients. The equipment is portable and, in case of critical injury, permits quick and safe evaluation by the patient's bedside or else in the operating theatre, emergency room or in the intensive care unit. The safety of the examination is confirmed by our series: no complications were observed in the 31 patients in whom TEE was performed. Other authors[44] have demonstrated the safety and efficacy of TEE for the detection of mediastinal lesions in patients with multiple injuries. The time required to perform and interpret TEE images in patients with chest trauma has been evaluated by Brooks et al.[45] who reported that, in patients with serious lesions, TEE was completed in a mean time of 27 min. In our experience the entire examination was performed in 10 to 20 min. Our data confirm that TEE is more accurate than TTE in identifying segmental wall motion abnormalities in patients with blunt chest trauma since, in 4 patients, myocardial contusion was detected only by TEE. Therefore, TTE and TEE can be considered complementary techniques in these patients and even when the former permits satisfactory evaluation of myocardial function, TEE may provide further information about lesions of the thoracic aorta or other mediastinal organs.

With regard to biochemical markers, previous studies have stressed a poor correlation between CK-MB levels and the presence of myocardial contusion[1,16,19,46], since titers of the enzyme may also be elevated in response to skeletal muscle injury. Shapiro et al.[19] found no correlation between CK-MB levels and TEE results

in patients with blunt chest trauma. In our study, CK-MB titers were elevated in all patients with echocardiographic signs of myocardial contusion, but also in 19/26 patients (73%) without echocardiographic signs of myocardial contusion and in 8/15 patients (53%) with cTnI levels < 0.4 ng/ml. These data confirm the poor specificity of CK-MB titration for cardiac lesions in patients with massive skeletal injury such as in case of multisystem lesions and chest trauma, probably due to the release of CK-MB from skeletal muscles[47]. Conversely, cTnI is a regulatory protein found only in cardiac tissue and elevated levels of this enzyme have a high sensitivity for the detection of myocardial injury[25]. It has been demonstrated that, despite severe acute and/or chronic muscle injury, cTnI titers are not elevated unless concomitant cardiac injury is also present[25,48]. In our study, 17 of the 32 patients (53%) had abnormal cTnI levels. Thus, in view of the assay we used, one might conclude that myocardial contusion is very frequent in patients with blunt chest trauma. However, TEE images indicating myocardial contusion were observed in only 6 out of 10 patients with cTnI levels > 1 ng/ml. Even Adams et al.[26], who found a good correlation between cTnI levels and myocardial contusion diagnosed using TTE, suggested that echocardiography may be less sensitive than protein markers in detecting myocardial injury. One might presume that many patients with abnormal troponin levels have microlesions of the myocardium which are undetectable even at TEE. Our data show that the peak elevation in cTnI levels occurs within the first 12 hours and is probably caused by release of a cytosol pool of this protein[49]. Elevated levels persist until approximately the 48th hour. This time interval of our cTnI elevation was shorter than in previous studies[26], perhaps due to a minor degree of cardiac damage.

The prognostic significance of myocardial contusion is still controversial: in our study 2 of 6 patients with echocardiographic signs of myocardial contusion died of severe cerebral lesions. No other patient died or presented with any other cardiovascular dysfunction. While some authors reported a very low incidence of cardiac complications in patients with myocardial contusion[16,18,46,50,51], others have observed a higher morbidity and mortality and many potentially life-threatening complications[5-12].

In conclusion, the combined use of cTnI determination followed by TEE appears the best suited method to detect and quantify myocardial contusion in patients with blunt chest trauma. Abnormal levels of cTnI are frequently found and TEE appears unnecessary when cTnI levels are normal or low.

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