

Evaluation of Noninvasive Tests of Cardiac Damage in Suspected Cardiac Contusion

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SUMMARY Nonpenetrating trauma to the chest can result in cardiac damage that may be overlooked because of associated injuries and the lack of obvious thoracic injury. The clinical diagnosis of important cardiac damage in this setting is difficult. We evaluated noninvasive tests for detecting myocardial damage in 100 patients with severe, nonpenetrating chest trauma. The noninvasive tests included serial ECG, serial total CPK and CPK-MB enzymes, continuous Holter monitor recording to detect dysrhythmia, and technetium-99m pyrophosphate scintigraphy.

Peak CPK-MB elevations occurred in 72 patients. ECG abnormalities were noted in 70 patients, and 27 patients had Lown grade 3 or greater dysrhythmias. Fifteen patients died and all had autopsies. The noninvasive abnormalities were nonspecific and did not reflect myocardial contusion that led to clinically important cardiac complications.

BLUNT TRAUMA to the chest can result in cardiac damage. Significant cardiac damage may be overlooked because associated injuries may be recognized more easily and because evidence of thoracic injury is absent.¹⁻⁶ The reported incidence of cardiac involvement in trauma varies from 6% to 76%,^{6,7} and may represent the most common unsuspected visceral injury responsible for death in fatally injured accident victims.⁸ Parmley et al. believe that the true incidence of nonpenetrating trauma to the heart is not well established, primarily due to inability to make an accurate clinical diagnosis.⁷

Recent advances in detecting myocardial damage (principally in the setting of acute myocardial infarction) have included the use of CPK isoenzymes and technetium-99m pyrophosphate myocardial imaging. Experimental and clinical evidence suggests that these tests may be helpful in detecting cardiac "damage" in the setting of major trauma.⁹⁻¹⁵ Because of potential lethal complications of undetected cardiac damage in patients with chest trauma, as well as the uncertainty of appropriate medical management, we used noninvasive tests to study patients with suspected cardiac damage. We also sought to determine the outcome of patients with abnormal tests, and to determine the utility of the tests in the setting of trauma.

Methods

Protocol

One hundred adult patients were prospectively studied over a 9-month period at Harborview Medical Center, Seattle, Washington, the Northwest regional trauma

center. All patients were studied in the medical or surgical intensive care units. Criteria for admission into the study included admission to the intensive care unit with a history of nonpenetrating chest trauma and at least one of the following: severe precordial chest pain, fracture of the rib, clavicle or sternum, hemothorax or pneumothorax.

Patients (table 1)

Seventy-three men and 27 women, mean age 37 years (range of 18-86 years), were studied. Eighty patients were injured in motor vehicle accidents. Ten patients had sustained significant falls, three had crush injuries, and seven had other injuries.

Tests

All patients had 12-lead ECGs upon admission to the emergency room. Serial tracings were obtained for the first 3 days of hospitalization. Cardiac enzymes, including total CPK and CPK-MB, were drawn upon admission, repeated at 12 hours and then serially for the first 3 days. CPK-MB was determined by a modification of the method of Henry, Roberts and Sobel by DEAE sepharose absorption and electrophoretically by a commercially available kit (Helena Laboratories).^{16, 17} In all patients, continuous Holter monitoring was started within 12 hours of admission and continued for 24-48 hours. The tapes were scanned manually using an Avionics cardioscanner. Technetium-99m pyrophosphate scintigraphy was performed on days 1 and 3 of the hospitalization. Images were obtained approximately 2 hours after injection of a 20-mCi dose using a General Electric portable scintillation camera with high-resolution collimation and a 15% window centered on the photopeak. Four or more views were obtained for 700,000 counts per view (typically 30° right anterior oblique, anterior, and 30° and 60° left anterior oblique).

Definitions

The ECG was considered abnormal if there were any abnormalities of the ST segment, T wave, QT interval, or atrioventricular or intraventricular conduction.

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TABLE 1. *Patient Profile*

	n
Males	73
Females	27
Age (years)	
Mean	37
Range	18-86
Previous cardiac disease	15
Mode of injury	
Motor vehicle accident	80
Fall	10
Crush injury	3
Other	7

Rhythm disturbances were categorized according to the modified Lown classification.¹⁸ CPK-MB was recorded as IU/l and as a percentage of the total CPK. A CPK-MB fraction greater than or equal to 2% of the total CPK was considered abnormal. Technetium-99m pyrophosphate myocardial imaging was scored on a scale of 0-4. Using this scale, 2-4 + discrete uptake was considered positive, 2 + diffuse uptake equivocal, and 0-1 + diffuse uptake negative.

Statistical Analysis

Chi-square tests were used for statistical analysis. Fischer's exact tests were performed on the autopsy data because the number of patients was small. Tests for trends in proportions were also used. Two-sided hypotheses were tested.¹⁹

Results

Table 2 lists the electrocardiographic findings in the study. Seventy patients had abnormal ECGs. The majority of the changes were nonspecific ST- and T-wave changes (58%). No patient developed new Q waves. Sixteen patients (23%) had intraventricular conduction

TABLE 2. *Electrocardiographic Abnormalities During the First 3 Days of Hospitalization*

	n
Abnormal ECG	70
ST segment	39
T wave	19
Low voltage	2
New Q waves	0
Abnormal intraventricular conduction	16
RBBB	11
LBBB	2
IVCD	3
Atrioventricular conduction	2
Mobitz type II	1
3° AV block	1

Abbreviations: RBBB = right bundle branch block; LBBB = left bundle branch block; IVCD = intraventricular conduction delay; AV = atrioventricular.

abnormalities. Most of them had transient right bundle branch block. Two patients had transient atrioventricular block (one Mobitz type II block and one transient 3° atrioventricular block). Neither patient required temporary pacing.

Holter Monitor

Seventy-three patients had rhythm disturbances (table 3). Unifocal premature ventricular complexes occurred frequently (54%); complex ventricular dysrhythmias were less frequently seen. Of the 60 patients with ventricular ectopy, only 27 had dysrhythmias of Lown grade 3 or greater.

Myocardial Enzyme Determinations

The peak total CPK varied from 198 to 200,000 IU (median 1985 IU). The peak CPK-MB fraction also varied widely, 0-1400 IU (median 27 IU). Seventy-two patients had CPK-MB fraction greater than 6 IU (the upper limit of normal at our institution). However, only 27 patients had CPK-MB fraction greater than or equal to 2% of the total CPK.

Technetium-99m Pyrophosphate Scintigraphy

All 100 patients were scanned on two occasions during the first and third days of hospitalization; only two patients had clearly positive scans. Multiple projections frequently localized technetium-99m pyrophosphate accumulation to the chest wall (fig. 1). Five patients had equivocal studies. Thirty-three of 93 negative studies were categorized as 1 + diffuse.

Necropsy

Fifteen patients died, and an autopsy was performed on each. These patients were 19-70 years old (mean 40.6 years). None underwent open-chest cardiac massage before death. Five autopsied patients had had cardiac contusion; gross evidence was noted in one patient and all had histologic findings that included extravasation of red blood cells into and between myocardial muscle fibers and selective myocardial necrosis of the epicardium and myocardium. Only one of the patients had transmural myocardial contusion (fig. 2). Damage limited to the subendocardium was excluded

TABLE 3. *Results of Holter Monitoring*

Any dysrhythmia	73
Lown grade ≥ 3	27
Received treatment (Lown grade ≥ 3)	7
PSVT/AF	6
Unifocal PVCs	54
Multifocal PVCs	16
Bigeminy/trigeminy	11
Ventricular couplets/triplets	9
Ventricular tachycardia	3
Ventricular fibrillation	0

Abbreviations: PSVT = paroxysmal supraventricular tachycardia; AF = atrial fibrillation; PVC = premature ventricular complex.

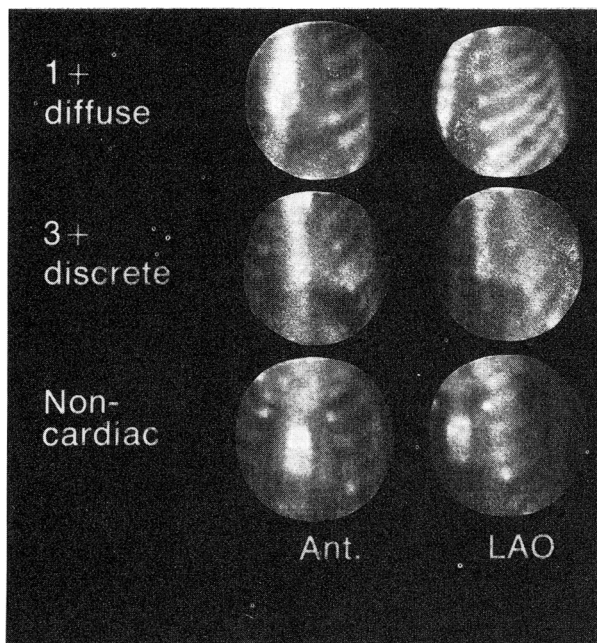


FIGURE 1. Scintigrams showing 1+ diffuse uptake, discrete cardiac uptake, and noncardiac accumulation of the radio-nuclide, a common finding in this patient population. ANT = anterior view; LAO = left anterior oblique view.

since its presence is nonspecific and found frequently in patients who die of hypoxia and other noncardiac causes.³⁰ The cause of death in the five patients with cardiac contusion was noncardiac in all cases: three patients died of central nervous system injuries and two of respiratory failure (table 4).

Discussion

In our series, 70% of the patients had electrocardiographic changes during the first 3 days of their hospitalization. The most frequent ECG changes were ST-segment and T-wave changes. Other investigators have stressed the importance of ECG changes in diagnosing cardiac contusion.^{8, 21, 22} However, ECG changes may be common in patients with noncardiac trauma, and may not offer the necessary specificity in diagnosing myocardial contusion.^{4, 7-9, 12, 24} In critically ill patients, electrocardiographic changes are not necessarily due to myocardial damage. Dolara et al.²⁵ studied ECG changes in 98 patients without open-chest injuries and found that 63% of the patients with severe trauma had ECG changes. Blair et al.⁴ described patients who had autopsy-proved myocardial contusion and normal ECGs. Parmley et al.⁷ showed that experimentally induced chest trauma in animals produced ECG changes and mediastinal or pulmonary hemorrhage, yet cardiac findings at necropsy were normal.⁷ Of the five patients in this study with documented myocardial contusion at autopsy, four had ECG changes. However, all 10 patients who died with no evidence of myocardial contusion at necropsy had transient ECG abnormalities.

Eighteen patients had conduction abnormalities, 16

of whom had intraventricular conduction abnormalities. Previous authors have documented the occurrence of conduction abnormalities in clinical and experimental myocardial contusion, including bundle branch block, left anterior hemiblock, and bifascicular block.^{5, 7, 8, 26-32} Fourteen of our patients had transient right bundle branch block, two patients had left bundle branch block (one transient), and two patients had atrioventricular block (one transient third-degree atrioventricular block). No patient required atropine or cardiac pacing. Only one of our five patients with myocardial contusion confirmed by autopsy had transient conduction abnormalities (transient right bundle branch block). Factors other than cardiac damage, such as vagal tone, sympathetic tone and electrolyte status (especially hyperkalemia) can induce conduction disturbances in a normal heart; hence, conduction disturbances are nonspecific and are not diagnostic of cardiac damage in the setting of trauma.³⁴⁻³⁶

Suspected Cardiac Contusion and Dysrhythmias

Previous studies document dysrhythmias in both human and experimentally induced cardiac contusion. Virtually all dysrhythmias have been produced experimentally or have been seen in patients with



FIGURE 2. Photomicrograph showing selective myocardial necrosis and extravasation of red blood cells in myocardial tissue from a patient with cardiac contusion. Magnification $\times 100$.

TABLE 4. *Clinical Profile of Autopsy Patients*

Pt	ECG	CPK-MB	Lown 3 dysrhythmias	Technetium scan	Cause of death
Necropsy positive for contusion					
1	Normal	1%	Yes	Neg (1 + dif)	CNS
2	+	1%	No	Neg	CNS
3	+	3%	No	Neg	Pneumonia
4	Normal	1%	Yes	Neg (1 + dif)	CNS
5	+	1%	Yes	Neg (1 + dif)	ARDS
Necropsy negative for contusion					
1	+	4%	Yes	Neg	Pneumonia
2	+	1%	No	Neg (1 + dif)	Pneumonia
3	+	2%	Yes	Neg	CNS
4	+	2%	Yes	Neg (1 + dif)	ARDS
5	+	2%	Yes	Neg	CNS
6	+	1%	No	Neg	ARDS
7	+	1%	No	Neg	Pneumonia
8	+	4%	No	Neg (1 + dif)	Pneumonia
9	+	7%	Yes	+ 3 discrete	CNS
10	+	1%	No	Neg	Pneumonia

Abbreviations: Neg = negative; CNS = central nervous system injury; ARDS = acute respiratory distress syndrome; + = at least one abnormal ECG.

myocardial contusion; these dysrhythmias include atrial fibrillation, atrial flutter, sinoatrial block, nodal dysrhythmias, atrioventricular and idioventricular rhythms.^{1, 3, 4, 5, 8, 21, 23, 24, 26, 27, 30} Parmley has observed that asystole can occur in myocardial contusion without other clinical signs.⁷ The severity and duration of dysrhythmias may be related to the extent of myocardial contusion and the reduction in cardiac output.^{1, 37} Ventricular fibrillation has been precipitated by blunt trauma in animal studies^{1, 3, 7, 38} and may be an important mechanism for immediate death in man in this setting. We found that 73% of our patients had some dysrhythmia during the period of observation. The most common dysrhythmia was unifocal premature ventricular complexes. Only 27% of our entire study group had complex dysrhythmias (at least Lown grade 3), and only three patients had ventricular tachycardia, two of whom were treated. One episode was noted on the Holter recording analysis and was unnoticed clinically. One patient with ventricular tachycardia died, but had no evidence of cardiac contusion at autopsy.

Ventricular fibrillation was not observed in our series. Only seven patients received antidysrhythmic therapy, which was initiated at the discretion of the patient's physician.

Suspected Cardiac Contusion and Enzymes

Cardiac enzymes, including CPK-MB, are considered to be specific in diagnosing myocardial damage.⁴⁰ Lindsay et al.⁹ suggested that the MB isoenzymes early after injury might offer evidence of cardiac trauma. They documented the presence of CPK-MB in the serum of victims of motor vehicle accidents. Normal values for CPK-MB vary up to 3% of total CPK.^{41, 42}

Although the CPK-MB fraction is of great value in a coronary care unit, where patients usually do not have noncardiac disease, it is probably less useful in other settings. CPK-MB elevations have been reported in tachyarrhythmias, trauma, crush injuries, muscle diseases and gas gangrene.⁴¹⁻⁴³

Using our hospital's criterion of CPK-MB greater than 6 IU to be positive, 72 patients would have been suspected of having cardiac damage; but if CPK-MB/total CPK $\geq 2\%$ is considered positive, only 27 patients would have been so categorized. One of our patients had an extensive crush injury from a logging accident; his peak total CPK approached 200,000 IU and his MB fraction was 1400 IU. This CPK-MB value suggests massive myocardial necrosis. Not only did this patient survive, but none of the other noninvasive tests examined were abnormal, including ECG, Holter and scintigraphy, and he did not have any cardiac problems clinically. Four of the five patients with documented myocardial contusion at autopsy had CPK-MB/total CPK less than 2%, yet all four had CPK-MB fraction greater than 6 IU. Thus, microscopic cardiac contusion can be missed by the CPK-MB/total CPK $\geq 2\%$ criterion, and patients with major trauma can have very high CPK-MB levels without other evidence for cardiac damage. Therefore, we do not believe CPK-MB is a useful indicator of cardiac damage in this setting.

Suspected Cardiac Contusion and Scintigraphy

Radionuclide imaging has been used in experimentally induced cardiac contusion in dogs.^{11, 13, 14} Coleman et al.¹⁵ found positive technetium-99m pyrophosphate scans within 24 hours and persistent defects for at least 1 week. In contrast to myocardial necrosis secondary to ischemic heart disease, where technetium-99m uptake is delayed, Downey et al.¹⁴ found that contused myocardium sequestered technetium-99m pyrophosphate within 2 hours. They also found that myocardial uptake was present despite the occurrence of nontransmural damage. However, Gonzales et al.¹³ found conflicting results in experimental myocardial contusion. They noted that the scintigraphic results correlated positively with the extent of myocardial damage: Dogs with pathologic changes that did not involve the inner third of myocardium had negative scintigrams, whereas those with transmural damage had positive scintigrams.¹³

In a clinical evaluation of technetium-99m pyrophosphate scanning, Brantigan et al.²¹ studied 29 patients suspected of having myocardial contusion. The scans were positive in only two patients thought to have contusion. Therefore, they concluded that technetium-99m pyrophosphate scintigraphy was of no clinical value. However, they accepted electrocardiographic changes as equivalent to myocardial damage; but, as we have suggested, this may not be reliable. Go et al.¹² reported 2+ diffuse uptake in one patient and another patient with 4+ localized uptake in the setting of cardiac contusion.

In our study, all patients were scanned within 12 hours of admission and again on the third day of hospi-

talization. Multiple views were used to localize uptake in the chest wall. Almost all of our patients (98%) had negative scans. None of the five patients with positive cardiac necropsy for myocardial contusion had positive scintigrams; however, only one had transmural damage. In one of the two patients with positive scintigrams, all other tests were positive (arrhythmias, CPK-MB/total CPK $\geq 2\%$), but the cardiac necropsy was negative. It is possible that transmural damage is necessary for the development of a discretely positive scintigram, as is frequently the case with myocardial infarction; however, we cannot explain the negative scan in the patient who had transmural myocardial contusion.

In conclusion, none of 100 patients with major non-penetrating chest trauma had clinically important cardiac damage. Noninvasive tests of cardiac damage, including CPK-MB, serial ECG, the presence of complex dysrhythmias and technetium-99m pyrophosphate scintigraphy, were nonspecific and did not reflect myocardial contusion that resulted in clinically important cardiac complications.

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