대 한 외 상 학 회 지
Vol. 24, No. 1, June, 2011

• 원 저 •

다발성 외상환자에서 폐좌상과 심근좌상의 상관관계

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— Abstract —

Correlation Between Pulmonary Contusion and Myocardial Contusion in Patients with Multiple Injuries

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Purpose: This study was conducted to evaluate the correlations among pulmonary contusion severity, trauma score and cardiac troponin I (cTnI) level.

Methods: We prospectively evaluated patients with multiple injuries who had been admitted to the emergency department (ED) from July 2007 to July 2008. We first measured the total creatinine kinase (CK), the MB fraction of CK (CK-MB), TnI, and myoglobin within 2 hours after the injury. We then checked the electrocardiogram, x-ray, and computed tomography (CT) results. Finally, we assessed the injuries as variables and then compared the results for patients with elevated TnI levels (group A) and patients whose TnI levels fell within the normal range (group B).

Results: Eighty-six of the 92 patients admitted to the ED were enrolled. The pulmonary contusion score (PCS) was well correlated with PaO_2/FiO_2. TnI levels were correlated with PCS. When TnI levels were above 0.86 ng/ml, the mortality was estimated with 100% sensitivity and 86.1% specificity.

Conclusion: Pulmonary contusion severity is correlated with TnI level. When the PCS is high and the cTnI level is elevated in multiple-injury patients, we recommend continuous cardiac monitoring and further evaluation. (J Korean Soc Traumatol 2011;24:31-36)

Key Words: Troponin I, Multiple injuries, Injury severity score, Cardiac contusion, Pulmonary contusion

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접수일: 2010년 10월 7일, 심사일: 2010년 11월 3일, 수정일: 2010년 12월 1일, 승인일: 2011년 5월 30일

This paper was supported by clinical research grant from Pusan National University Yangsan Hospital, in 2009

I. Introduction

Myocardial contusion can be defined as any myocardial lesion by blunt chest trauma accompanied by pathological, histological, or biochemical abnormalities. The exact incidence of myocardial contusion in patients with blunt chest trauma is unknown, and various estimates range from 3 to 70%.(1-5) Myocardial contusion during blunt chest trauma is common and may lead to fatal cardiac complications such as arrhythmias, conduction defects, cardiogenic shock, valvular dysfunction, or hemopericardium. The causative mechanisms of myocardial contusion include road traffic accidents (for example, a direct blow to the anterior chest from a steering wheel), thoracic wall compression of the heart between the sternum and the spine, rapid deceleration, a fall from a great height, indirect compression on the abdomen with upward displacement of the abdominal viscera, etc. Previous studies have been conducted to try to pin down the diagnosis of myocardial contusion. However, to date the diagnosis of myocardial contusion is complicated by nonspecific symptoms and signs. In particular, as myocardial contusion is generally combined with multiple injuries, the diagnosis of myocardial contusion is made more difficult by symptoms that mimic hypovolemic shock, neurogenic shock, and hypoxia due to pulmonary contusion, or vascular injury. However, the diagnosis of myocardial contusion is difficult and includes nonspecific findings in multiple injuries. The initial diagnosis of myocardial contusion may be delayed or obscured in multiple-trauma patients, particularly those with hypovolemic shock or neurogenic shock. Therefore, early detection, evaluation, and management are important for the patients' prognosis. Many investigators have studied biochemical analyses such as total CK, CK-MB, myoglobin, and troponin T or I, searching for the key to diagnosis of myocardial contusion.

Recently, the authors reported that serum cardiac troponin I (cTnI) is a specific marker of myocardial contusion.(1-10) cTnI present in the heart muscle cells is detected by enzyme-linked immunoassay (ELISA) analysis. The cardiac muscle isoform of TnI is a 24-kD protein uniquely expressed in the human heart.(2) Some investigators have demonstrated that cTnI is released after traumatic myocardial injury. Therefore, elevated serum cTnI after blunt chest trauma is a good predictor of myocardial contusion. One experimental study demonstrated that cTnI is elevated immediately after the chest trauma and returns to baseline levels after 20 min. Therefore, cTnI is helpful in an early diagnosis of acute myocardial injury.(7)

In contrast, total CK, CK-MB, and myoglobin have low sensitivity and specificity in the detection of traumatic myocardial injury because most trauma is combined with muscular injuries to multiple extremities.(4,5,11) Multiple-injury patients usually present with pulmonary contusion and elevated serum cTnI levels or within normal range in the chest trauma. Therefore, the present study was performed to evaluate the correlation between pulmonary contusion score (PCS) and PaO_2/FiO_2 ratio (an index of arterial oxygenation efficiency that corresponds to ratio of partial pressure of arterial O_2 to the fraction of inspired O_2) that the parameter of pulmonary contusion severity, trauma scores and serum cTnI during the initial evaluation process in the emergency department.

II. Materials and Methods

We prospectively evaluated patients with multiple injuries who had been admitted through the emergency department (ED) between July 2007 and July 2008. We measured the total creatinine kinase (CK), MB fraction of CK (CK-MB), troponin I, and myoglobin within 2 hours after injuries and checked electrocardiogram, x-rays, and computed tomography (CT). We also assessed the chest abbreviated injury score (AIS), injury severity score (ISS), revised trauma score (RTS), pulmonary contusion score (PCS).(12) PCS compared with PaO_2/FiO_2 ratio and chest AIS. We divided into the elevated troponin I group (group A) and troponin I group within normal range (group B), and compared with PCS, chest AIS, RTS, ISS, total CK, CK-MB and myoglobin. Each variables was analyzed by ROC curve .Comparisons of the factors affecting the 2 groups were performed by linear regression analysis and the chi-square test or Fisher's exact test. Comparison of PaO_2/FiO_2 ratio and chest AIS by PCS was performed by one-way ANOVA analysis. All tests were analyzed by using the SPSS statistical software (Version 12.0k for Windows, Chicago, IL). Statistical significance was defined as a P value less than 0.05.

III. Results

Eighty-six out of ninety-two patients admitted to the ED were enrolled in the study. Six patients were excluded

because their pulmonary contusion diagnosis was uncertain. The male:female ratio was 71:15. The mean age was 39.7 ±17.3. The trauma mechanisms included 21 passenger traffic accidents (TA), 19 pedestrian TA, 18 motorcycle TA, 23 falls, and 5 miscellaneous injuries. ECG rhythms were nonspecific, and included 36 patients with normal

Table 1. Comparison of PaO_2/FiO_2 ratio and chest AIS by the pulmonary contusion score (PCS)

	PCS			
	Mild (0-2) n=18	Moderate (3-5) n=40	Severe (6-12) n=28	*P* value
PaO_2/FiO_2	380.6±88.75	334.2±107.48	237.4±111.94	< 0.001
Chest AIS	3±0.76	3.3±0.60	3.5±0.58	0.191

AIS; Abbreviated Injury Score
PaO_2/FiO_2: ratio of partial pressure of arterial O_2 to the fraction of inspired O_2

Table 2. Comparisons of Parameters on the two groups

Variables	Group A (44)	Group B (42)	*P* value
PCS	5.84±2.80	3.69±1.80	0.025
RTS	9.41±2.94	11.02±1.85	0.281
Chest AIS	3.50±0.60	3.12±0.67	0.271
ISS	32.91±10.60	24.60±11.41	0.626
Total CK	1974±1990	1164±1393	0.492
CK-MB	20.78±17.02	13.67±15.48	0.856
Myoglobin	2243±2191	884±739	0.088

Group A: elevated troponin I group (> 0.06 ng/ml), group B: troponin I group within normal range (≤ 0.06ng/ml)
PCS: pulmonary contusion score, RTS: revised trauma score, AIS: abbreviated injury score,
ISS: injury severity score, CK: creatinine kinase, CK-MB: MB fraction of CK

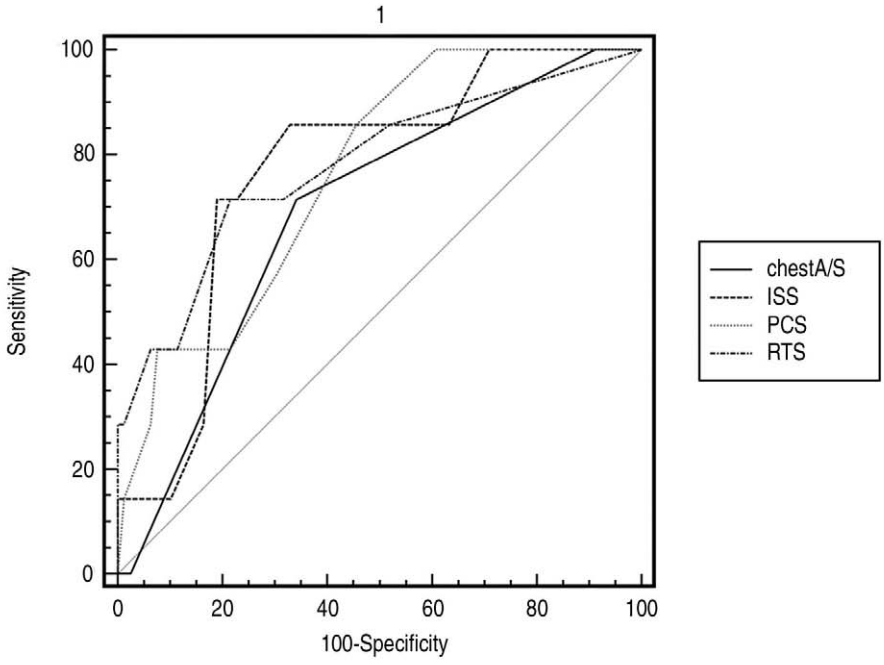

Fig. 1. ROC curves of chest AIS, ISS, PCS, and RTS, ROC: receiver operating characteristic, AIS: abbreviated injury score, ISS: injury severity score, PCS: pulmonary contusion score, RTS: revised trauma score, RTS in the score systems was the most high as 0.779 of AUC (area under curve), but not significant statistically.

sinus rhythms, 38 with sinus tachycardias, and others with abnormal arrhythmias. PCS was well correlated with PaO_2/FiO_2, but not with chest AIS (Table 1). Troponin I levels were correlated with PCS (Table 2). RTS in the score systems was the most high as 0.779 of AUC(area under curve), but not significant statistically (Fig. 1). TnI in the cardiac enzymes was the most high as 0.958 of AUC and statistically significant against CK and CK-MB (p=0.004 vs. CK-MB, p<0.001 vs. CK) (Fig. 2). When TnI levels were over 0.86 ng/ml (cut off of value), the mortality was made estimate of 100% sensitivity and 86.1% specificity. Six out of eighty-six patients expired in the ED. Four out of six patients expired by hypovolemic shock as the cause of death. One patient expired by transtentorial herniation due to severe brain injuries. One patient in our study showed symptoms strongly suggestive of myocardial contusion, but expired without responding to conservative treatment.

IV. Discussion

Clinically, pulmonary contusion is often combined with myocardial contusion in multiple-injury patients. Therefore, we hypothesized that the severity of the pulmonary contusion might be a predictive factor of myocardial contusion in multiple-injury patients with chest trauma. The severity of the pulmonary contusion measured by 3D CT and pulmonary contusion score (PCS) in previous studies. For examples, the exact methods for measuring pulmonary contusion volume had been developed in previous studies that used computer-generated 3D CT. However, there are limitations to this technique in that the measuring time is prolonged and the software is not common. Severe contusion was defined as ≥ 20%, and moderate contusion was defined as <20%.(13,14) Kim(12) et al. reported that the scoring of the pulmonary contusion volume on the chest CT was a good reflection of the pulmonary contusion severity. We measured PCS on the chest CT of trauma patients and compared with PaO_2/FiO_2 ratio as an index of pulmonary function. As a result, PCS was well correlated with PaO_2/FiO_2 ratio.

Screening methods for myocardial contusion include the use of electrocardiography and biochemical cardiac markers such as total CK, CK-MB, myoglobin, and troponin I. Of these parameters, troponin I is the most crucial. In trauma patients, presence of circulating troponin I specifically

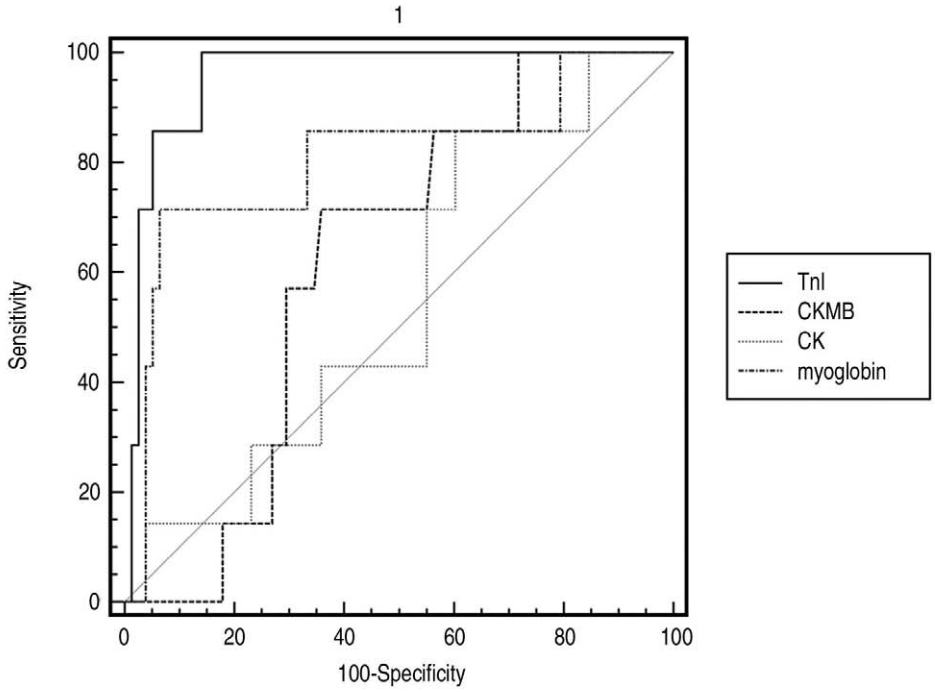

Fig. 2. ROC curves of cardiac enzymes, ROC: receiver operating characteristic, TnI: troponin I, CKMB: MB fraction of creatinine kinase, CK: creatinine kinase, TnI in the cardiac enzymes was the most high as 0.958 of AUC (area under curve) and statistically significant against CK and CK-MB. When TnI levels were over 0.86 ng/ml (cut-off value), the mortality was made estimate of 100% sensitivity and 86.1% specificity.

reflects myocardial damage either through direct contusion or as an indirect effect of hypovolemic shock.(15) In our study, troponin I levels were correlated with PCS. As a result, it suggest that the the severity of the pulmonary contusion is a good reflection of myocardial injury. If the troponin I level is measured at above the normal range, or if elevated tropinin I is suggested by clinical symptoms and signs, close observation and/or serial EKGs must be performed, along with continuous cardiac monitoring. Then, confirming methods such as TTE or TEE, cardiac MRI, scintigraphy, and/or positron emission tomography may be needed.(5,16,17) Other studies have demonstrated that cardiac troponin I is a predictor of arrhythmia, and when it increases over the normal range, the risk of arrhythmia and left ventricular dysfunction also is increased, leading to the need for further cardiac work-up and monitoring.(4) However, troponin I levels were not significant statistically in our study.

Cardiac troponin I also may predict mortality in medical and trauma patients. Wu(18) et al., who reported that elevated cardiac troponin I levels at admission are associated with increased morbidity and mortality in noncardiac critically ill patient during their ICU stay and cTnI levels can also be elevated in patients without acute coronary syndrome, such as in sepsis and trauma patients, and that this is associated with an adverse effect. In our study, when TnI levels were over 0.86 ng/ml (cut off of value), the mortality was made estimate of 100% sensitivity and 86.1% specificity. Therefore, cTnI levels may be a risk factor for mortality in the trauma patients.

Treatment of myocardial contusion is usually conservative. Nonetheless, potentially fatal myocardial contusion may require invasive methods. Masiakos(19) et al. reported successful management of severe combined pulmonary and myocardial contusion with extracorporeal membrane oxygenation. Lindstaedt(20) et al. reported that the incidence of myocardial contusion requiring intensive care monitoring in blunt thoracic trauma was 20% and the outcome and prognosis was favourable, but routine cardiac work-up was not indicated.

There are several limitations to our study. First, the sample size of patients is small, and our trials were carried out at only one emergency care center. In the future, for more accurate results, data from large numbers of patients in multicenter trials is needed. Second, the study subjects did not have follow-ups such as serial checks of cardiac markers, routine laboratory results, and echocardiography for confirmation of the diagnosis after admission. Serial checks of cardiac markers stopped without routine follow-ups, as vital signs are stable. Therefore, we do not know with certainty how many patients actually suffered myocardial contusions.

V. Conclusion

Pulmonary contusion score (PCS) was well correlated with PaO_2/FiO_2 ratio as an index of pulmonary function, and then Pulmonary contusion severity also is correlated with serum cTnI level. Elevated serum cTnI in multiple-injury patients may be a risk factor for mortality. When PCS is high and troponin I levels are elevated, we would like to highlight the possibility of myocardial contusion, and the need for continuous cardiac monitoring and further evaluation.

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