

## EVALUATION OF MYOCARDIAL CONTUSION USING BIOCHEMICAL MARKERS IN CHEST TRAUMA PATIENTS

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**ABSTRACT: BACKGROUND:** Controversy surrounds diagnostic tests and their ability to predict the early cardiovascular complications in blunt cardiac trauma patients. **AIMS:** We evaluated electrocardiogram (ECG), cardiac troponin-I (Tn-I) and creatine kinase-MB (CK-MB) levels to diagnose myocardial contusion (MC) in chest trauma patients. **SETTINGS AND DESIGN:** A total of 50 chest trauma cases were randomly included in the study. Patients with a history of ischemic heart disease or myocardial infarction and/or treatment with anti-anginal or antihypertensive drugs were excluded. **MATERIAL AND METHODS:** ECG, CK-MB, creatinine phosphokinase (CPK), and Tn-I were recorded at admission and repeated at 24 and 72 hours. Patients were grouped as suspected or non-suspected MC group based on any of the following: Abnormal ECG; CK-MB >50 U/L; CK-MB/CPK ratio>5; Tn-I >1.5ng/ml. Suspected MC group patients were sub-grouped as significant MC group: If they had-abnormal echocardiographic findings such as regional wall motion abnormalities, regurgitant lesions; unexplained hemodynamic instability and shock with systolic blood pressure <90mmHg; patients unresponsive to fluid resuscitation in spite of replacement of estimated blood loss; or patients requiring vasopressor for cardiogenic shock. **STATISTICAL ANALYSIS:** Student's t-test, Z-test and sensitivity analysis were performed for statistical analysis. **RESULTS:** Of 50 patients, 44 belonged to suspected MC group; 15 of them had significant MC. ECG was the most sensitive (93.3%) indicator of MC whereas Tn-I showed high specificity (86.20%) for MC. **CONCLUSIONS:** Troponin-I is a specific marker of MC while ECG is a sensitive diagnostic tool.

**KEYWORDS:** Thoracic injuries, Contusion, Troponin I.

**INTRODUCTION:** Myocardial contusion (MC) is an important entity among thoracic trauma cases and needs special attention because of difficulties in its diagnosis and its implications on outcome of these victims. In United States of America, about 13.4 million motor vehicle related accidents occurred in the year 2000. Forty percent of these victims had associated blunt thoracic trauma. Incidence of MC varies from 16-76% in polytrauma patients.<sup>[1]</sup> The widely varying incidence results from lack of well accepted gold standard diagnostic tests and standardized study protocols. Many trauma patients are suspected to have associated cardiac injury but there is no established criterion to diagnose these injuries. Confirmatory histopathological diagnosis of MC is impractical in live victims. Relying on biochemical markers and electrophysiological studies always remain an issue of concern because of their variable sensitivities, specificities, and therefore, the clinical significance. Various authors have described different diagnostic tools and criteria for diagnosing MC in trauma victims. These include electrocardiography (ECG), echocardiography and various biochemical markers such as creatine phosphokinase (CPK), creatine kinase-MB (CK-MB), CK-MB/CPK ratio, etc. Although transesophageal and transthoracic echocardiography (TTE and TEE) are good investigative

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tools, the quality of images in chest injury patients may be compromised; moreover, presently these imaging modalities are not readily available in many centers of developing countries. In last decade, cardiac troponin-I and T (Tn-I and Tn-T) have been established to diagnose myocardial injury in myocardial infarctions because of high sensitivity and specificity. As earlier available biochemical markers are non-specific, we hypothesized that these specific biochemical markers may help in diagnosing myocardial injury in polytrauma patients. Thus we evaluated various biochemical markers; CK-MB, cardiac Tn-I and CK-MB fraction and ECG to find incidence of MC and their clinical relevance in diagnosing significant MC.

**MATERIALS AND METHODS:** After approval of the Hospital Ethical Committee 50 patients of chest trauma of all age and either sex were randomly selected for the study. A written informed consent was obtained from all patients and/or their attendants. Patients were considered to have chest injury if they had any of the following: 1) Significant contusion on the chest wall with or without respiratory distress, 2) Clavicle fracture, 3) One or more rib fractures, 4) Pneumothorax, 5) Hemothorax and, 6) Surgical emphysema. Patients with known history of ischemic heart disease, old infarction or on chronic therapy with nitrates, digoxin, antihypertensive, and anti-anginal medication were excluded from the study. The selected patients were grouped as:

1. Suspected MC group: If they had any of the following-Abnormal ECG, CK-MB>50 U/L, CK-MB/CPK ratio>5, Tn-I>1.5ng/ml.
2. Non-suspected MC Group: Patients not having any of the above Suspected MC group patients were further sub-grouped as;
  - A. Significant MC group: If they had any of the following;
    - a. Abnormal echocardiographic findings such as regional wall motion abnormalities, regurgitant lesions, etc.
    - b. Unexplained hemodynamic instability and shock with systolic blood pressure < 90 mmHg
    - c. Patients unresponsive to fluid resuscitation in spite of replacement of estimated blood loss.
    - d. Patients requiring vasopressor support for cardiogenic shock.
  - B. Non-significant MC Group: Patients other than those described in-group A.

Chest radiograph, ECG, and biochemical markers-CPK, CK-MB and cardiac Tn-I were done at the time of admission. ECG and biochemical markers were repeated at 24 and 72 hours after admission and more frequently if clinical circumstances indicated. In addition to the above, TTE was performed in patients with positive biochemical markers if they were stable for transportation to the cardiac laboratory. The ECG criteria used for the diagnosis of a myocardial injury was defined as a rhythm other than sinus or transient sinus tachycardia or ventricular dysrhythmias, supraventricular tachyarrhythmia (Atrial fibrillation or flutter), junctional tachycardia, sinus bradycardia, conduction abnormality, a bundle branch block pattern, a prolonged QT interval, Q-wave formation, ST segment depression or elevation of greater than 1mm, or flat or inverted T waves or both in two or more leads. Abnormal echocardiography included pericardial effusion, regional wall motion abnormality (Defined as an alteration in wall motion and in systolic thickening) and acute valvular dysfunction. Patients were managed as per the emergency protocols. Trauma patients were resuscitated initially using crystalloids, colloids and blood for hypovolemia. Inotropic support with dopamine or other drugs was given if patients failed to respond to fluid therapy or had unexplained hypotension.

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Patients having airway obstruction or respiratory distress or desaturation were managed with oral airway or endotracheal intubation or tracheostomy and mechanical ventilation as indicated clinically. Patients were provided with adequate analgesia. Vital parameters were recorded at the time of admission and then half hourly for 2 hours and then 6 hourly for first day and daily until 72 hours. Patients were observed for complications such as hypotension, bradycardia, tachycardia, arrhythmias, shock, angina, and myocardial infarction.

Blood samples for measurement of CPK, CK-MB and Tn-I was drawn in collecting tubes without an anticoagulant and was kept at room temperature for 20 minutes to allow clotting. CPK and CK-MB were measured without delay. CK-MB was assessed by immune-inhibition assay; the assay range at 37°C is 0-125U/L and a value more than 50U/L was considered significant. For the quantitative determination of Tn-I an immune-enzymatic assay was used.

Statistical analysis was performed using chi square analysis to compare the various markers in significant and non-significant MC groups. Sensitivity and specificity were drawn for individual and combination of tests.

**RESULTS:** We enrolled 50 chest trauma patients in the study. Most of the chest trauma patients were young males, weighed between 61-70 kg and belonged to ASA grade II and III. Forty-four patients of these 50 were suspected of MC based on either raised biochemical markers or abnormal ECG. Of these 44 patients, 15 had significant MC and 29 had non-significant MC [Fig. 1]. There was no significant difference between the significant MC group and non-significant MC group with respect to age, sex and ASA physical status. Most of the patients in this study had injury severity score (ISS) in the range of 9-19 [Table 1]. There was no correlation between ISS and significant MC.

Abnormal ECG was found in 30 patients (60%) and was the most frequent finding [Table 2]. Abnormal ECG was found in 14 (93.3%) of 15 patients with significant MC and 16 (55.17%) of 29 patients of non-significant MC [Table 4].

Twenty five (50%) chest trauma patients had raised CK-MB levels [Table 2]. Eight of these (53.3%) belonged to significant MC group (n=15) and 17 patients (58.62%) belonged to non-significant MC Group (n=29) [Table 4]. In non-significant MC group, the mean CK-MB levels gradually decreased from 85.76±115.90 at zero hours to 37.89±31.41 at 24 hours to 23.89±25.62 at 72 hours. In the significant MC group the mean levels initially decreased from 57.53±98.59 at zero hours to 55.73±97.04 at 24 hours and then again increased to 116.20±248.15 at 72 hours [Fig. 2]. The difference in the mean CK-MB levels in the two groups at zero and 24 hours was not significant while the difference at 72 hours was statistically significant (p<0.05).

The Tn-I was found positive in 10 (20%) of 50 chest trauma patients [Table 2]. Out of these 6 (40%) patients belonged to significant MC group (n=15) and 4 (13.79%) patients belonged to non-significant MC group (n=29) [Table 4]. The mean Tn-I values gradually decreased from 12.24±30.11 at zero hours to 1.97±5.47 at 24 hours and 1.77±4.70 at 72 hours in significant MC group [Fig. 3]. The Tn-I values in non-significant MC group also gradually decreased from zero to 72 hours. However, this difference in the decline in two groups was not significant.

The values of CK-MB in relation to Tn-I at 72 hours were more in significant MC group (51.77) than in non-significant MC group (36.6) thus making Tn-I an important indicator at 72 hours (p=0.312) [Fig. 4].

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Of 44 patients of suspected MC, 5 (11.36%) patients showed all markers positive i.e. raised CK-MB and Tn-I levels, and abnormal ECG [Table 3]. Six (13.63%) patients of suspected MC group had both CK-MB and Tn-I raised and 7 (15.9%) patients of suspected MC had raised Tn-I and abnormal ECG [Table 3].

ECG showed highest (93.3%) sensitivity while Tn-I showed highest (86.20%) specificity [Table 5]. The Tn-I had the highest overall predictive value of 70.45 followed by ECG with a predictive value of 61.36 [Table 5].

Receiver operating characteristic (ROC) curves of ECG, CK-MB, CK-MB/CPK and cardiac Tn-I are shown in fig 5. The area under curve (AUC) of ECG was 0.691(p-0.040), AUC of CK-MB was 0.474(p-0.776), AUC of CK-MB/CPK was 0.461(p-0.674) and that of Tn-I was 0.631(p-0.158).

Echocardiography could be performed in only four patients. Two patients with significant MC showed abnormal echocardiography findings. The other two patients with normal echocardiography were grouped into non-significant MC group. Hypotension was the commonest complication in significant MC group (p<0.01) followed by tachycardia (p<0.05) and cardiac arrest (p<0.01). Five patients had cardiac arrest for which cardiopulmonary resuscitation was done. Out of these two patients had return of spontaneous circulation and were put on ventilatory support. But three patients could not be revived.

Out of 15 patients in the significant MC group 7 patients underwent surgery under anaesthesia; intraoperatively, four of them (57.14%) developed tachycardia and 2(28.5%), developed hypotension. While in non-significant MC group, 12 patients underwent surgery under anaesthesia; two of them (16.66%) had tachycardia and none developed hypotensive episode. The difference between the two groups was statistically significant (p<0.05). Tachycardia was the commonest intraoperative arrhythmias seen.

**DISCUSSION:** MC is histologically described as cellular injury, extravasation of red blood cells between myocardial muscle fibers, occasional necrosis and polymorphonuclear leukocytic infiltration. Cardiac concussion is the mild form of injury without any cellular disruption whereas the contusion leads to cellular membrane disruption and release of cytoplasmic and membranous enzymes in the plasma. Thus it is cellular disruption with subsequent release of CK-MB fraction that differentiates myocardial contusion from concussion. Forces involved in blunt cardiac injury (BCI) include compression of the heart between the spine and sternum, abrupt pressure fluctuations in the chest and abdomen, shearing from rapid deceleration and blast injury, and fragments from rib fractures causing injury to the heart. The patients may have clinical manifestations as arrhythmias (Including ventricular tachycardia and ventricular fibrillation), ventricular dysfunction with congestive heart failure, acute valvular involvement with regurgitation, ventricular aneurysm with thrombus formation, pericardial effusion with or without pericardial tamponade, intracardiac structural damage, or cardiac rupture.<sup>[2]</sup>

Continuous ECG, radionuclide perfusion scan, echocardiography, and CK-MB fractions have been employed to detect MC.<sup>[3]</sup> Controversy exists regarding the utility of ECG, cardiac enzymes, and echocardiography to diagnose BCI. ECG has traditionally been relied on extensively for screening patients at risk of developing complications. Most recent investigators defined concussion and contusion by the presence of CK-MB in the blood following injury.<sup>[4]</sup> Some authors have defined MC by abnormalities noted on ECG gated ventriculo-scintigraphic angiography.<sup>[5]</sup> The presence of CK-MB above 5% of total CPK, as measured electrophoretically, has also been employed for definition of

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contusion.<sup>[6]</sup> Two recent studies, by Mori et al<sup>[7]</sup> and Collins et al<sup>[8]</sup> have highlighted the usefulness of serum troponin levels in evaluating cardiac injury, suggesting that Tn-I is a useful screening test when performed prior to echocardiography following BCI. Collins et al,<sup>[8]</sup> further concluded that measuring troponin in presence of a normal ECG is not necessary and if the admission ECG has minor abnormalities and the troponin at 4 to 6 hours after injury is normal then the risk of BCI related complications is low.

Nuclear studies using radionucleotide imaging may indicate the presence and extent of myocardial damage. However, <sup>99m</sup>Tc pyrophosphate scans have high false negative rates and are not recommended for use in diagnosing myocardial injury. Radionuclide ventriculography may reveal reduced ejection fraction and focal defects in the wall motion of the ventricles, particularly in the right ventricle. Thallium or sestamibi imaging is excellent for evaluating perfusion of the left ventricle but is a poor imaging modality for right ventricle perfusion. However, right ventricle contusions are twice more common as compared to left ventricle contusion. Myocardial injury should be suspected with reversible findings on radionucleotide angiography. Maenza et al concluded that radionucleotide scans were not useful in the evaluation of blunt thoracic trauma.<sup>[9]</sup> The incidence of BCI may vary due to diagnostic tools; however, all tests, mechanism of injury, as well as clinical picture should be considered when diagnosing and managing a patient with suspected BCI.<sup>[10]</sup>

We studied 50 chest trauma patients to evaluate cardiac injury on the basis of abnormal ECG findings, elevated CK-MB, CK-MB/CPK ratio and high Tn-I levels. Hypotension was the commonest complication seen in almost all patients at one or the other time during the study followed by tachycardia observed in 53.3% of the patients having Significant MC. Overall 5(33.3%) patients had cardiac arrest. In our study, 60% patients were found to have ECG abnormalities, 50% were found to have high CK-MB levels, 28% had high CK-MB/CPK ratio and 20% had high Tn-I levels in 50 chest trauma patients. Out of these, 5 patients were found to have all diagnostic tools positive for significant MC. ECG and Tn-I were found to have significant correlation with significant MC whereas no statistical significance was seen with CK-MB or CK-MB/CPK ratio. Similar results were seen by Salim and coworkers, they found that 50% and 23.5% of their patients had abnormal ECG findings and elevated Tn-I levels respectively.<sup>[11]</sup> We further found that ECG is the most sensitive (93.3%) diagnostic tool to screen significant MC in suspected patients whereas Tn-I is the most specific (86.20%) tool for MC while CK-MB and CK-MB/CPK ratio showed poor sensitivity (53.33% and 26.67%) and specificity (41.37% and 65.51%). While comparing the positive predictive values, Tn-I, ECG, CK-MB, CK-MB/CPK ratio has positive predictive values of 60, 46.6, 32, and 28.57 respectively. Negative predictive values of ECG, Tn-I, CK-MB, CK-MB/CPK ratio were 92.85, 73.53, 63.15, and 63.33 respectively. The Tn-I showed the best overall predictive value (70.45) followed by ECG (61.36), CK-MB/CPK ratio (52.27) and CK-MB (45.45).

Adams and coworkers studied 100 patients with extensive skeletal muscle injury for non-cardiac surgery and found that only one had a slight elevation of cTn-I.<sup>[12]</sup> Bertinchant and associates compared Tn-I and T with conventional markers in chest trauma patients to detect cardiac contusion.<sup>[13]</sup> Sensitivity, specificity and negative and positive predictive values of Tn-I and T in predicting a myocardial contusion in blunt trauma patients were 23%, 97%, and 77%, 75%, and 12%, 100%, and 74%, 100%, respectively.

In our study, we analyzed Tn-I and CK-MB at time of admission ( $t^1$ ), at 24 hours after admission ( $t^2$ ) and at 72 hours after admission ( $t^3$ ). A decreasing trend from  $t^1$  to  $t^3$  was seen with Tn-I in all the 50 patients. In myocardial injuries Tn-I levels start to rise about 4-6hrs after the onset of

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myocardial injury and they remain elevated for 7-10 days with a peak at 24-36hrs. CK-MB had a similar declining trend in non-significant MC group while in significant MC group the mean CK-MB levels initially decreased from 0-24 hours and then showed a slight rise at 72 hours. Probably this difference in trend in our study is because of delayed admission of our patients.

**CONCLUSIONS:** Although area under curve of ECG has been found to be significant in relation to troponin-I, but troponin-I have highest OPV among all the cardiac markers. Thus we conclude that Tn-I is a specific marker to detect MC in chest trauma patients while ECG was found to be a sensitive diagnostic tool to diagnose MC in chest trauma patients. As high percentage of patients were found to have MC, trauma cases should be evaluated for myocardial contusion in routine using ECG and Tn-I.

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ISS	Suspected MC(44)	Non suspected MC(6)	z-value	p-value
9-19	23(52.27%)	2(33.33%)	0.87	>0.01 NS
20-29	13(29.54%)	2(33.33%)	0.19	>0.10 NS
30-39	6(13.63%)	1(16.67%)	0.20	>0.10 NS
40-49	1(2.27%)	0(0)	0.37	>0.10 NS
50-59	1(2.27%)	1(16.67%)	1.69	<0.10

**Table 1: Distribution of patients according to Injury severity score(ISS)(n=50)**

Markers	No. of MC Patients(50)	Incidence/'000
ECG	30(60%)	600
CK-MB	25(50%)	500
CK-MB/CPK	14(28%)	280
Tn-I	10(20%)	200

**Table 2: Incidence of myocardial contusion(MC) in chest trauma patients with various diagnostic tools**

Markers	No. of Patient's (44)
ECG	30(68.18%)
CK-MB	25(56.81%)
Tn-I	10(22.72%)
ECG + CK-MB	15(34.09%)
ECG + Tn-I	07(15.90%)
CK-MB + Tn-I	06(13.63%)
ECG + CK-MB + Tn-I	05(11.36%)

**Table 3: Correlation of different combinations of various diagnostic tools to detect myocardial contusion in Suspected Myocardial Contusion patients(n=44)**

Parameters		Significant MC		Non-Significant MC	
		N	Percent (%)	N	Percent (%)
EKG	Abnormal	14	93.3	16	55.17
	Normal	1	6.7	13	44.83
CK-MB	>50 U/L	8	53.3	17	58.62
	<50 U/L	7	46.6	12	41.38
CKMB/CPK	>5	4	26.6	10	34.48
	<5	11	73.3	19	65.52
Tn-I	>1.5ng/ml	6	40	4	13.79
	< 1.5	9	60	25	86.21

**Table 4: Comparison of ECG and biochemical markers in patients with Suspected myocardial contusion (n=44)**

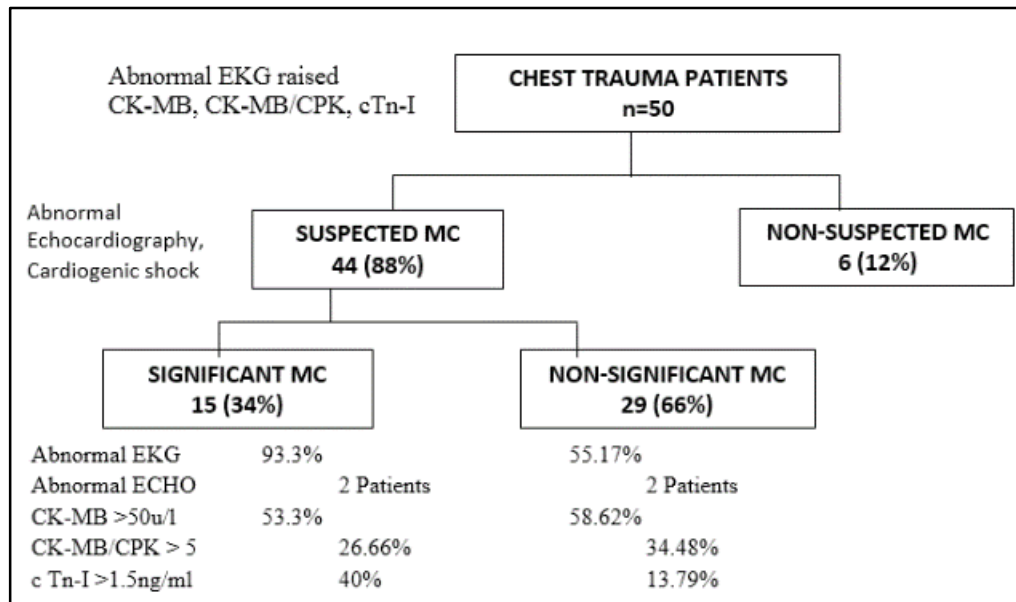
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Parameter	Sensitivity	Specificity	PPV	NPV	OPV
ECG	93.3%	44.83%	46.67	92.86	61.36
CK-MB	53.33%	41.38%	32.00	63.16	45.45
CK-MB/CPK	26.67%	65.52%	28.57	63.33	52.27
Tn-I	40.00%	86.21%	60.00	73.53	70.45

**Table 5: Diagnostic value of ECG and biochemical markers for predicting myocardial contusion in chest trauma victims**

Parameters	Area	P Value	Significance
EKG	0.691	0.040	S
CK-MB	0.474	0.776	NS
CKMB/CPK	0.461	0.674	NS
Tn-I	0.631	0.158	NS

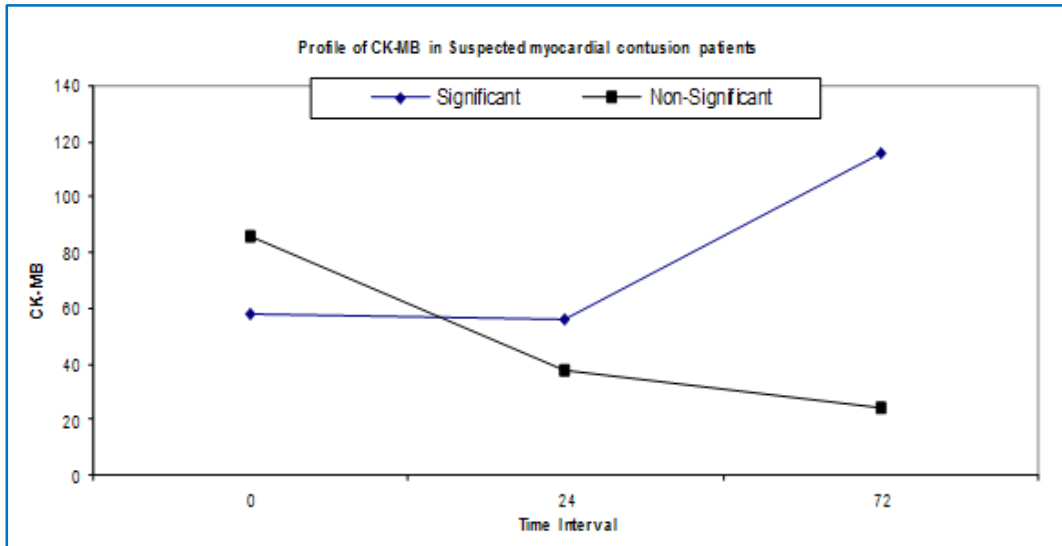
**Table 6: Area under ROC curve analysis**



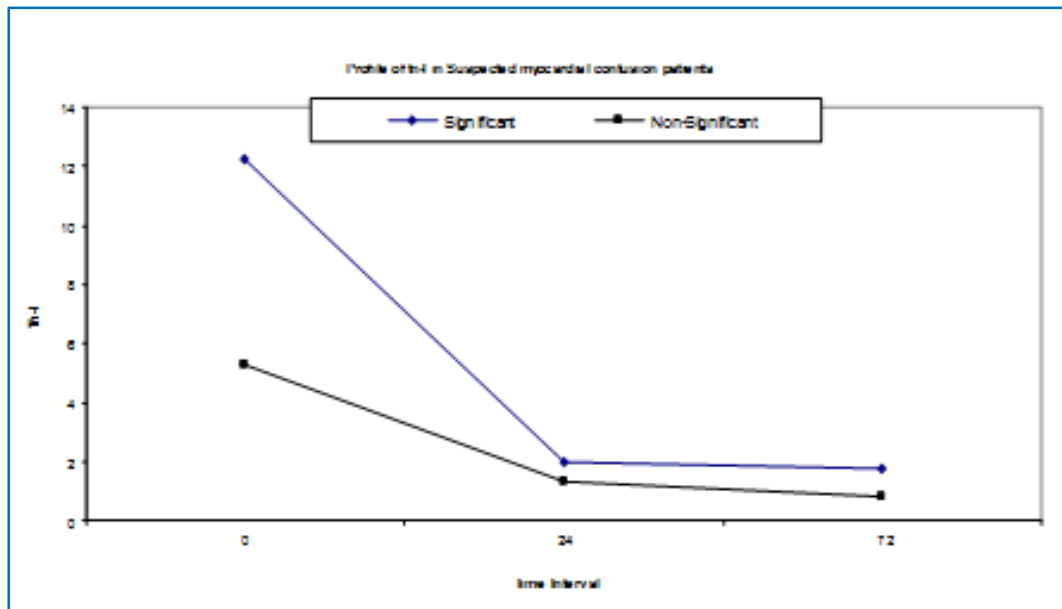
**Fig. 1: Flow diagram to show distribution of patients**



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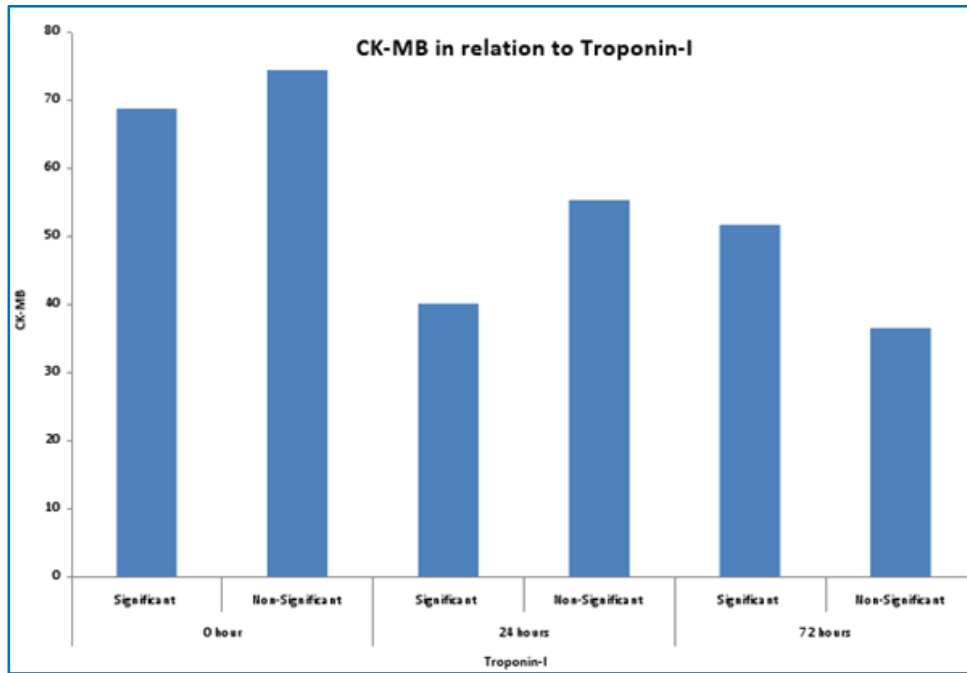


**Fig. 2: Profile of CK-MB in suspected myocardial contusion patients**

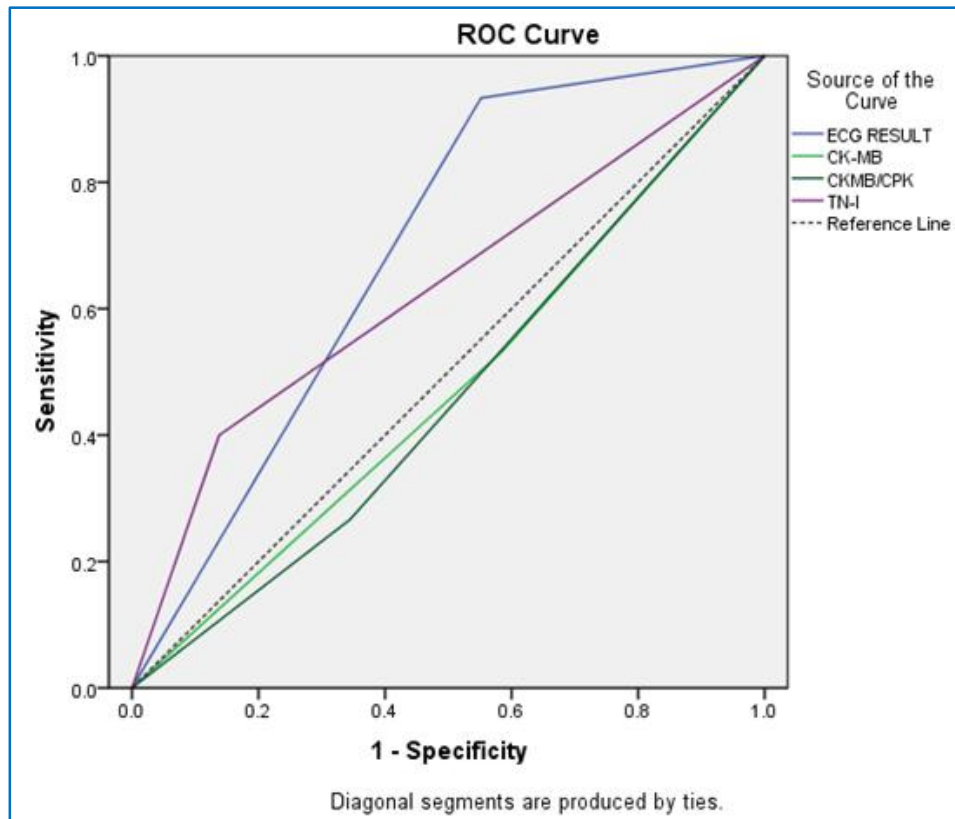


**Fig. 3: Profile of Tn-I in suspected myocardial contusion patients**

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**Fig. 4: CK-MB in relation to Tn-I**



**Fig. 5: Area under curve**

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