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Serial values for hematologic and biochemical analysis after myocardial infarction in rats

Mi-Jin Lee, Hyun-Jin Tae, Ying-Hua Li, Do-Hyeon Yu, In-Ae Han, Seok-Won Lee, Dong-Choon Ahn[1], In-Shik Kim, Jin-Ho Park*

College of Veterinary Medicine & Bio-Safety Research Institute, Chonbuk National University, Jeonju, 561-756, South Korea; [1]School of Veterinary Medicine, Kangwon National University, Chuncheon, 200-701, South Korea

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Abstract

To diagnose acute myocardial infarction (MI), many cardiac markers have been used in hematologic and biochemical analysis, and many studies have been published for hematologic and biochemical analysis associated with human acute MI. However, after occurrence of acute MI, the serial investigation for values in hematologic and biochemical analysis including chronic MI has rarely been performed. To observe the change of the serial values in hematologic and biochemical analysis, we induced artificial MI. The left main descending artery (LMDA) of the left coronary artery was ligated during the progression (day 1, 3, 5, 7, 14 and 30) of MI. Total 66 Sprague-Dawley rats were divided into the sham group (n=24, thoracotomy without LMDA ligation) and the experimental (MI) group (n=42, with LMDA ligation). And all individual in each group was sacrified at day 1, 3, 5, 7, 14 and 30 for the hematologic and biochemical analysis. In comparison of hematologic analysis between the sham and MI groups, the mean values of red blood cell (RBCs), hemoglobin and hematocrit (HCT) showed a steady increase. In biochemical analysis, the mean values of glucose, cholesterol, total creatine kinase (CK) and isoenzyme MB, and lactate dehydrogenase (LDH) were increased in all MI groups compared with the sham groups. The results of this study suggest that early hematologic and biochemical mean values occurred after acute MI are similar to those of human acute MI. In conclusion, we could observe the

*Corresponding author
Phone : +82-63-270-2557, Fax : +82-63-270-3780
E-Mail : jpark@chonbuk.ac.kr

alterations and serial values in hematologic and biochemical analysis to the extent of chronic status after acute MI.

Key words: Hematology, Biochemistry, Myocardial infarction, Serial values

Introduction

The main cause of myocardial infarction (MI) is a focal complete obstruction in one of the main coronary arteries as a result of platelet aggregation and coagulation. This event causes irreversible ischemic damage or death of myocardial cell, which results in impaired contractility of the heart muscle within seconds[1]. Thus, MI acts as a significant factor of acute heart failure or left ventricular dysfunction. Clinical signs of MI can present differently in individual patients, with symptoms that range from none at all to sudden cardiac death. Despite the diversity of the symptoms of MI, some characteristic symptoms exist including characteristic chest pain, looking pale, sweating, syncope and impairment of cognitive function without other cause[1-5].

When these symptoms are encountered, clinicians perform diagnostic tests, including serial electrocardiograms (ECG), X-rays and blood tests, to determine if MI is involved[5-6]. Quick and reliable diagnosis of MI is the most important factor affecting the mortality and morbidity of this condition. The diagnosis of MI has been based on the presence of two of the three following conditions: characteristic chest pain, diagnostic ECG changes and elevation of the cardiac markers in blood samples[2-3,5]. For example, because several markers with different sensitivities and specificities are used for the diagnosis of MI, it is very difficult to make an accurate diagnosis using only a single marker[5]. In addition, ECG could been a poor diagnostic test for acute MI patients because approximately half of all acute MI patients show normal or no diagnostic ECG[6]. Therefore, many researchers have constantly performed the study for accurate diagnosis of MI.

Among some conditions for the evaluation of acute MI, cardiac markers in serum have been facilitated by the development of highly sensitive and specific determinations. Many studies for changes in the activity of serum enzymes, such as aspartate aminotransferase (AST), lactate dehydrogenase (LDH), creatine kinase (CK) and isoenzyme MB (CK-MB), have been widely performed in the early phase of suspected ischemic myocardial injury[5,7-9]. In addition, recently several studies have been reported for the correlation between the value such as WBC[10,11], hematocrit (HCT)[12,13] and glucose[14-16] and the mortality in MI patients. Like this, many studies have been published for hematologic and biochemical analysis in relation to acute MI in human. However, few studies for the serial observation of alterations in hematologic and biochemical analysis from acute to chronic MI has been accomplished. Therefore, we observed the serial alterations in hematologic and biochemical

analysis after occurrence of artificial MI.

Materials and Methods

Induction of experimental MI

Sprague-Dawley (SD) rats aged 60-day-old were obtained from the Bio-Safety Research Institute at Chonbuk National University (Jeonju, South Korea), and were conditioned in conformance with the standards described by the US National Institutes of Health (NIH). The number of rats in the each sham (control) and MI groups was 4 and 7, respectively, and rats were subjected to treatment for 1, 3, 5, 7, 14 and 30 days in both the sham and MI groups. After being conditioned, the rats were anesthetized by ketamine (80.75 ㎎/㎏) and xylazine (5.13 ㎎/㎏), which was administered via an intraperitoneal injection. The rats were then provided with artificial ventilation by inserting a 16-gauge catheter into the trachea through the oral cavity, which was then connected to a rodent ventilator that administered 80 strokes per min of room air. A thoracotomy was performed through the fourth intercostal space to expose the heart. The left main descending artery (LMDA) of the left coronary artery was ligated just below 2-3 ㎜ from origin of LMDA using a 7.0 silk suture as shown in Fig 1. The sham rats were treated in a similar manner except that the LMDA was ligated. After surgery, all individuals received antibiotic treatment for only 1 day. Each individual was sacrificed at day 1, 3, 5, 7, 14 and 30 after occurrence of MI.

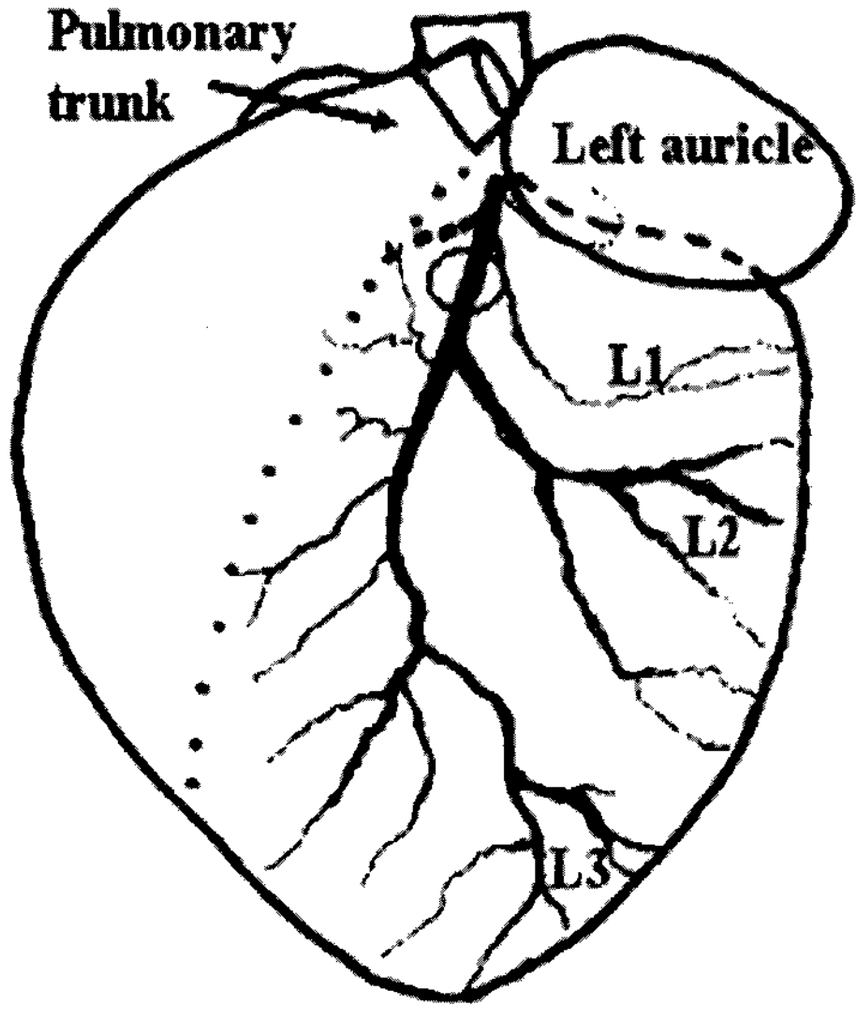

Fig 1. A diagram for ligation (circle) at the left main descending artery (LMDA) of the left coronary artery. L1~L3, the branches of left coronary arteries.

Hematologic and biochemical analysis

Blood samples were collected from the sham and MI rats sacrificed. A total 10 ㎖ of blood was collected from each individual, 2-3 ㎖ of that was placed into EDTA tubes for hematologic analysis, and the remainder was used for biochemical analysis and fibrinogen measurement. Hematologic analysis was promptly performed.

Hematologic analysis (WBCs, RBCs, hemoglobin, HCT and platelets) was performed using automatic coulter counters (ABC VET, France), and the differential count of WBC (lymphocytes, neutrophils, monocytes, eosinophils and basophils) was manually measured. Biochemial analysis [alkaline phosphatase (ALP), gamma-glutamyl transpeptidase (γ-GT), AST, alanine

aminotransferase (ALT), amylase, blood urea nitrogen (BUN), glucose, phosphorus, calcium, albumin, cholesterol, uric acid, CK, creatinine, total bilirubin, total protein and globulin] was accomplished in serum using Analyst® (benchtop chemistry system, Hemagen, USA). Also, CK-MB and LDH value was measured using a Fuji dry-chem analyzer (Japan). Fibrinogen was manually measured using Millar method. If the value in biochemical analysis shows an undetectable result due to high concentration, the serum was diluted with distilled water to obtain the definite result and then was rechecked using a Fuji dry-chem analyzer.

Statistical analysis

All values represent the mean ± the standard error (SE). Values were considered statistically significant when a $P<0.05$ or a $P<0.01$ was obtained. The variance of variables observed in the sham and MI groups at the same time points was measured using an ANOVA test. All analyses were conducted using GLM SAS Version 8.0 (USA).

Results

In evaluation of WBC associated with the inflammation, the mean values of the sham groups were higher than MI groups at day 1 and day 3. However, the mean values of the MI groups were higher than the sham groups from day 5 to day 30. Especially, the mean value of the MI group at day 7 was increased over 100% than that of the sham group.

The differential count (lymphocytes, neutrophils, monocytes and eosinophils) of WBC in both all sham and MI groups was irregularly changed. Significant differences in the mean values for WBC, lymphocytes and neutrophils at day 7 and for eosinophils at day 1 were observed between the sham and MI group (Table 1).

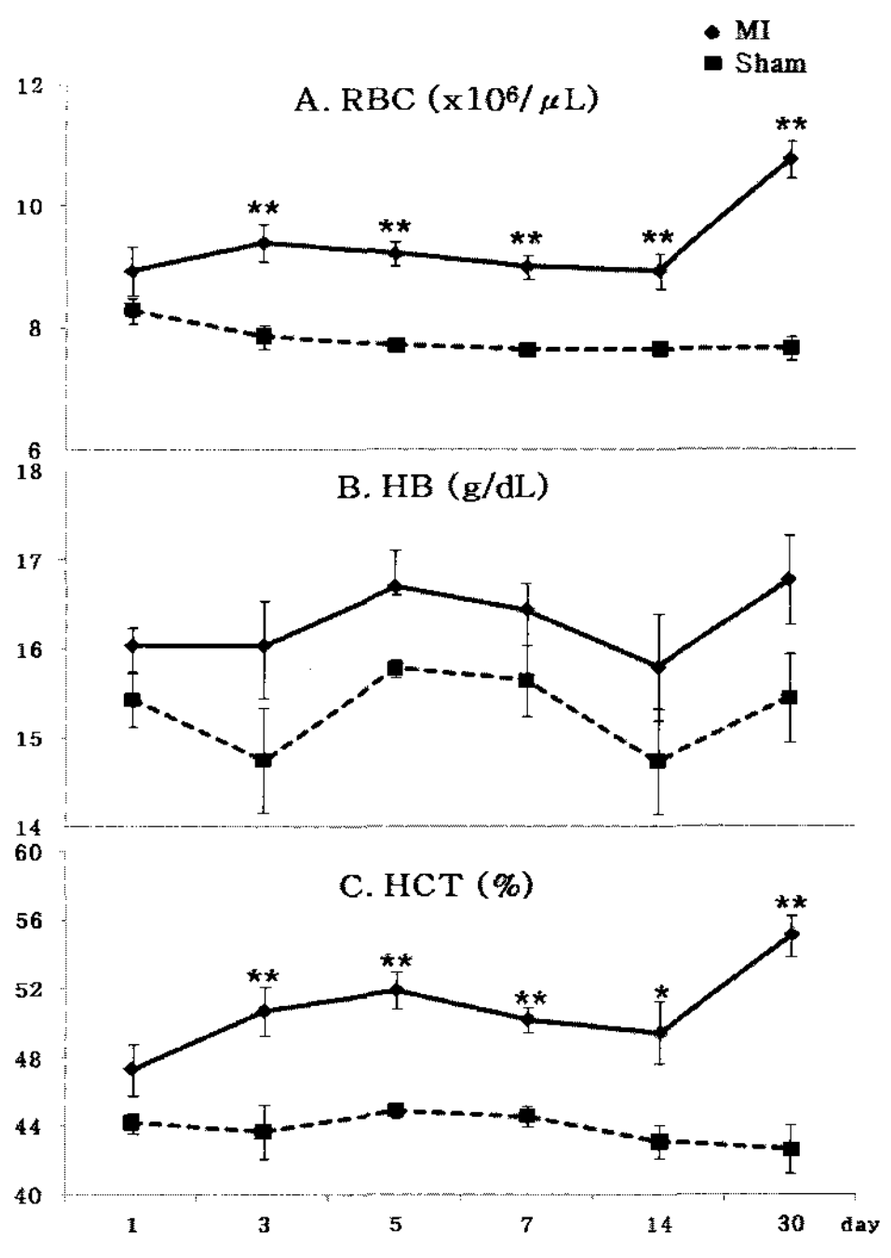

Fig 2. Serial values for the differences of RBC ($\times 10^6/\mu\ell$), hemoglobin (Hb, g/dℓ) and hematocrit (HCT, %) between the sham and MI groups ($^{*}P<0.05$; $^{**}P<0.01$).

The mean values of all MI groups are higher than the sham groups. In A and C, the relationship between the sham and MI groups is significant except day 1.

The RBC profiles (RBC, hemoglobin and HCT) were found to be high the mean values of all MI groups compared with the sham groups. In the MI groups, the mean values of the RBC profiles were slightly increased at day 1, but as MI

goes on chronic status, the increasing rate of the mean values was higher. Significant differences for the mean values of RBC and HCT were observed between the sham and MI group except day 1 (Fig 2). The mean values of platelet in the MI groups were lower than the sham groups except day 3. The mean values of fibrinogen were generally lower in the MI groups than the sham groups, but the variation of the mean values was irregular (Table 1).

Table 1. The mean ± standard error(SE) values for hematologic analysis of the myocardial infarction (MI) groups and the differences between the sham and MI groups (the increasing or decreasing rate in the MI group compared with the sham group)

	Day					
	1	3	5	7	14	30
WBC ($\times 10^3/\mu\ell$)	10.7 ± 1.0 (27.6% ↓)	9.1 ± 0.8 (4.3% ↓)	10.4 ± 1.5 (6.3% ↑)	17.0 ± 1.7** (127.8% ↑)	12.0 ± 1.3 (35.2% ↑)	8.0 ± 1.0 (1.3% ↑)
Lymphocyte (%)	73.9 ± 3.3 (9.4% ↑)	72.4 ± 2.8 (3.4% ↓)	72.4 ± 2.1 (5.3% ↓)	66.6 ± 2.5* (12.4% ↓)	76.3 ± 3.4 (0.8% ↑)	68.0 ± 2.7 (9.0% ↓)
Neutrophil (%)	24.6 ± 3.2 (17.4% ↓)	22.0 ± 2.8 (8.0% ↑)	24.3 ± 2.0 (14.3% ↑)	32.1 ± 2.7* (47.8% ↑)	20.5 ± 3.5 (3.5% ↓)	28.1 ± 2.6 (30.9% ↑)
Monocyte (%)	1.3 ± 0.6 (14.3% ↓)	4.9 ± 1.1 (61.9% ↑)	2.3 ± 0.8 (1.6% ↑)	1.0 ± 0.4 (42.9% ↓)	2.3 ± 1.2 (6.7% ↓)	2.6 ± 0.9 (14.3% ↑)
Eosinophil (%)	0.3 ± 0.2* (77.1% ↓)	0.7 ± 0.6 (56.0% ↓)	1.0 ± 0.5 (−)	0.1 ± 0.1 (71.4% ↓)	0.8 ± 0.6 (66.7% ↑)	1.3 ± 0.5 (14.3% ↓)
Platelet ($\times 10^3/\mu\ell$)	691.9 ± 34.1 (9.9% ↓)	854.0 ± 13.8 (5.4% ↑)	631.6 ± 28.3 (10.0% ↓)	668.0 ± 57.0 (18.5% ↓)	708.8 ± 62.0 (12.6% ↓)	704.6 ± 84.6 (14.4% ↓)
Fibrinogen (㎎/dℓ)	171.4 ± 28.6 (23.8% ↓)	171.4 ± 36.0 (14.3% ↓)	128.6 ± 18.4 (35.7% ↓)	314.3 ± 26.1** (109.5% ↑)	200.0 ± 26.1 (−)	128.6 ± 28.6 (14.3% ↓)

↑: the increasing rate, ↓: the decreasing rate, *: $P < 0.05$, **: $P < 0.01$

Especially, the MI group at day 7 showed the increase of the mean values in WBC as well as fibrinogen.

Among the enzymes related to the hepatobilliary system and the liver, the mean values of ALP were highly maintained from day 3 after acute MI and all MI groups compared with the sham groups showed significant differences except day 1. The mean value of γ-GT was a little low in the MI group at day 1, however, after that, was higher than the sham groups. The mean value of AST, enzyme related to muscle, MI and liver damage, in the MI group was similar to the sham group at day 1, and at day 3 and 5, the mean value of AST was increased about 26% and 39% than the sham group, respectively. The mean values of ALT, enzyme specific for liver damage, were generally lower than the sham groups except day 5 and significant differences were observed compared with the sham group at day 1 and 3 (Fig 3 and Table 2).

Among enzymes related to kidney dysfunction, the mean values of BUN were found to be lower for all MI groups than the sham groups, and the mean values of

creatinine in the MI groups were diverse-

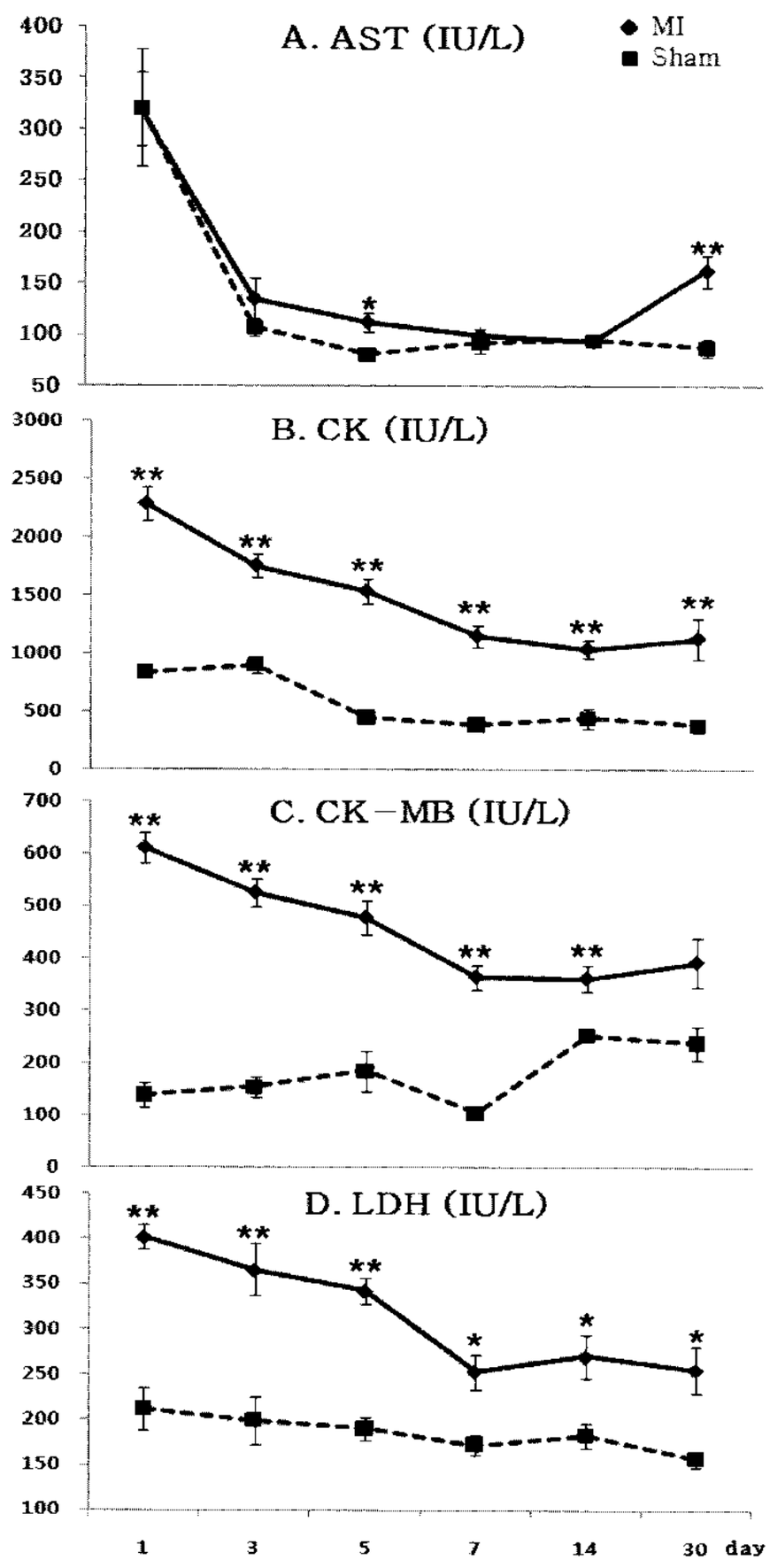

Fig 3. Serial values for the differences of aspartate aminotransferase (AST, IU/ℓ), creatine kinase (CK, IU/ℓ), creatine kinase isoenzyme MB (CK-MB, IU/ℓ) and lactate dehydrogenase (LDH, IU/ℓ) between the sham and MI groups (* $P<0.05$; ** $P<0.01$).

In A, the mean values of both groups showed dramatic increases at 1 day after MI. In B, the mean value of MI group at day 1 showed a dramatic increase, and then maintained higher value than the sham groups. In C, the mean values of all MI groups showed higher than the sham groups. In D, the relationship between the sham and MI groups showed significantly at all day after MI.

ly changed compared with the sham groups. However, the relationship between these variant values and MI are not considered to be clinically important (Table 2).

The mean values of amylase in the MI groups were lower than all sham group, however, it was limited to be mentioned for the relation of amylase and MI from this study because amylase is non-specific enzyme for the detection of pancreatic diseases. The mean values of glucose were higher in all MI groups than the sham groups. The mean values of glucose in the MI groups were increased about 30-50% except day 3 and significant differences were observed compared with the sham groups at day 1, 5, 7 and 30. The mean values of cholesterol were continuously increased in the MI groups after day 1 (Table 2).

The mean values of CK, CK-MB and LDH, which are the cardiac markers known to be associated with MI, were increased greatly in response to MI. In particular, the mean values of CK in the MI groups showed dramatic increases after day 1. As acute MI was progressed to chronic, the mean values of CK in all MI groups were still higher than the sham groups. The mean values of CK-MB in the MI groups were also maintained at a higher level, however, the increasing rates in serum of the MI groups were decreased gradually rather than initial level. The mean value of LDH in the MI group was higher over 90% than the sham group at day 1. Like the mean values of CK and CK-MB, the mean values of LDH in the MI groups

were highly maintained till day 30. Also, all MI groups in CK and LDH values and MI groups in CK-MB except day 30 showed significant differences compared with the sham groups, respectively (Fig 3).

The mean values of phosphorus, calcium, uric acid, total bilirubin, albumin, total protein and globulin in this study were found to be varied from comparison of the MI groups with the sham groups (Table 2). Significant differences between the sham and MI group for biochemical analysis were observed (Table 2).

Table 2. The mean ± standard error(SE) values for biochemical analysis of myocardial infarction (MI) groups and the differences between the sham and MI groups (the increasing or decreasing rate in the MI group compared with the sham group)

	Day					
	1	3	5	7	14	30
ALP (IU/ℓ)	171.1 ± 6.2 (1.0% ↑)	211.0 ± 23.4** (159.7% ↑)	260.7 ± 7.6** (167.4% ↑)	247.7 ± 15.8** (104.7% ↑)	237.5 ± 14.5** (127.8% ↑)	126.1 ± 4.5** (62.2% ↑)
γ-GT (IU/ℓ)	2.9 ± 0.3 (4.8% ↓)	3.1 ± 0.3 (57.1% ↑)	3.3 ± 0.4 (9.5% ↑)	3.7 ± 0.2 (14.3% ↑)	3.5 ± 0.3 (40.0% ↑)	3.4 ± 0.2 (24.7% ↑)
ALT (IU/ℓ)	69.1 ± 11.6* (40.4% ↓)	34.7 ± 1.6** (45.1% ↓)	46.0 ± 6.9 (10.2% ↑)	45.6 ± 8.5 (19.0% ↓)	30.0 ± 6.4 (36.5% ↓)	58.3 ± 8.0 (18.8% ↓)
Amylase (IU/ℓ)	1052.0 ± 83.2 (13.9% ↓)	1185.6 ± 66.2 (1.3% ↓)	960.4 ± 61.9 (15.3% ↓)	1125.0 ± 109.0 (16.7% ↓)	1330.8 ± 118.6 (19.1% ↓)	1459.4 ± 73.4 (5.1% ↓)
BUN (㎎/dℓ)	11.1 ± 1.0* (23.5% ↓)	18.7 ± 1.1 (1.2% ↓)	18.9 ± 0.9 (20.4% ↓)	12.9 ± 1.2** (29.4% ↓)	16.5 ± 2.7 (15.4% ↓)	17.8 ± 1.3 (11.2% ↓)
Glucose (㎎/dℓ)	181.1± 11.3* (31.7% ↑)	176.3 ± 9.4 (2.6% ↑)	178.7 ± 5.7** (45.0% ↑)	176.4 ± 5.6** (49.2% ↑)	199.3 ± 13.0 (29.0% ↑)	193.9 ± 12.3** (50.0% ↑)
Phosphorus (㎎/dℓ)	7.9 ± 0.7 (14.1% ↓)	8.0 ± 0.6 (16.2% ↑)	9.6 ± 0.4 (9.0% ↑)	8.1 ± 0.6 (13.9% ↑)	9.8 ± 0.7 (9.2% ↑)	6.6 ± 0.4 (18.2% ↓)
Calcium (㎎/dℓ)	9.5 ± 0.8 (0.3% ↓)	9.5 ± 0.5 (3.1% ↑)	9.6 ± 0.2 (0.3% ↓)	9.7 ± 0.7 (6.7% ↑)	10.6 ± 0.5 (9.0% ↑)	8.3 ± 0.1* (15.7% ↓)
Albumin (g/dℓ)	3.3 ± 0.2 (11.6% ↓)	3.4 ± 0.1 (4.1% ↓)	3.5 ± 0.1 (8.1% ↓)	3.2 ± 0.2 (9.0% ↓)	3.8 ± 0.2 (5.1% ↑)	3.3 ± 0.1* (13.7% ↓)
Cholesterol (㎎/dℓ)	60.3 ± 3.8 (9.1% ↑)	60.6 ± 2.7 (17.6% ↑)	74.6 ± 2.9 (11.3% ↑)	73.0 ± 8.5 (25.9% ↑)	70.0 ± 2.8 (13.4% ↑)	65.6 ± 2.1* (18.7% ↑)
Uric acid (㎎/dℓ)	2.0 ± 0.2 (20.3% ↑)	2.0 ± 0.4 (8.7% ↑)	1.7 ± 0.2 (2.2% ↑)	1.4 ± 0.1 (9.3% ↓)	1.5 ± 0.1 (24.0% ↓)	2.1 ± 0.3 (18.4% ↑)
Creatinine (㎎/dℓ)	0.3 ± 0.1 (16.2% ↓)	0.5 ± 0.1 (71.4% ↑)	0.3 ± 0.1 (8.6% ↓)	0.4 ± 0.1 (23.8% ↓)	0.3 ± 0.0 (26.7% ↑)	0.7 ± 0.1 (16.7% ↑)
T-bilirubin (㎎/dℓ)	0.1 ± 0.0 (−)	0.1 ± 0.00 (−)	0.1 ± 0.0 (−)	0.1 ± 0.0 (20.0% ↓)	0.1 ± 0.0 (−)	0.1 ± 0.0 (14.3% ↑)
T-protein (g/dℓ)	6.0 ± 0.3 (4.0% ↓)	6.5 ± 0.2 (2.4% ↑)	5.6 ± 0.1 (1.3% ↓)	5.7 ± 0.2 (2.7% ↑)	6.2 ± 0.2 (4.8% ↓)	7.0 ± 0.2 (1.7% ↑)

↑: the increasing rate, ↓: the decreasing rate, *: $P < 0.05$, **: $P < 0.01$

Discussion

Despite of the extensive study and the development of new diagnostic tools for MI, the mortality of patients as a result

of acute MI has been continued to increase. An elevated WBC value as an available marker of inflammation is associated with increased mortality after acute MI, and these results suggested that the WBC value can be used as a prognostic factor in patients with acute MI[11].

However, it was also showed that the WBC value is independently associated with mortality in MI states[10,17]. In report by Cannon et al.[10], acute MI showed 6.6% in 1,813 patients with WBC value >10,000/㎕, and 24.8% of 1,256 patients with WBC value >14,100/㎕ were diagnosed as a heart failure[11]. In our study, the elevation of the WBC value in the MI group compared with the sham group was observed from day 5 after occurrence of acute MI. At day 7, the mean value of WBC (>17,000/㎕) of the MI group was increased more 127% than the sham group. And, the mortality in all MI individuals including MI rats of day 7 was 0%. In the result, we confirmed that the increase of WBC is independently associated with mortality in MI states.

Fibrinogen values can be measured in venous blood. Normal values are about 150-300 ㎎/㎗ in human. Higher values are associated with cardiovascular disease (>460 ㎎/㎗). It may be elevated in any form of inflammation, as it is an acute phase protein[1]. At day 7 of the MI group, the mean value of fibrinogen was 314.3 ㎎/㎗ and the rate of plasma protein : fibrinogen was 18.0 : 1.0. In result, we couldn't observe the increase of fibrinogen, although MI states was.

Many studies related with abnormality in the RBC values have been shown that low hemoglobin or HCT value is common in patients with cardiovascular disease and anemia has been identified as an important determinant of prognosis in heart failure[12-13,18-19]. However, other studies have reported that the hemoglobin value is an independent predictor in the occurrence of acute coronary syndromes and acute MI[13,20]. Mahmoodi et al.[13] observed that the prevalence of MI in non-anemic patients with high HCT value was 20.2%. Our result showed high mean values of RBC, hemoglobin and HCT in all MI groups compared with the sham group. Also, the mean value of RBC and HCT showed significant differences between the sham and MI group except day 1. Thus, we could identify that acute and chronic MI affect to the increase in the mean value of RBC, hemoglobin and HCT. And, high RBC, hemoglobin and HCT values were dependent predictors in acute and chronic MI.

Platelets not only promote thrombus formation, but may also trigger acute coronary syndromes through other mechanisms, such as stimulation of inflammatory processes[21-22]. Avramakis et al.[23] reported that acute MI patients showed higher WBC and lower platelet values compared with controls. In this study, the mean value of platelet was lower in the MI groups except day 3. Low platelet values in the MI groups were regarded as a result of chronic MI.

In biochemical analysis of this study, the mean value of ALP was elevated in all MI groups compared with the sham groups. However, the study for the relationship between the ALP value and MI has hardly been reported. In this study, the mean values of ALP in the MI groups

showed dramatic increases after 3 day. Thus, we suggest that the increase of ALP values may be related with chronic MI.

Acute hyperglycemia is a common feature that occurs shortly after the onset of acute MI in patients with or without diabetes mellitus[14-15]. In addition, patients with chronic heart failure due to ischemic heart disease are associated with marked insulin resistance[16]. However, it has remained unclear whether acute hyperglycemia is causally related to adverse outcomes after acute MI. We obtained high glucose values in all MI groups compared with the sham groups. These high values are unrelated with food because we inhibited meal before a day of blood collection. Thus, we observed that hyperglycemia was associated with the occurrence of MI and was independently related with increased mortality risk.

Hypercholesterolemia is not a disease but a metabolic derangement that can be secondary to many diseases and can increase the risk of heart disease, most notably cardiovascular disease[24-25]. In this study, the mean values of cholesterol in the MI groups were slightly elevated compared with the sham groups.

Vega et al.[25] reported that 7.7 of 418 patients with hypercholesterolemia died due to cardiovascular diseases. We observed that the mean value of cholesterol was elevated after occurrence of MI, but the extent of the increase wasn't big. Therefore, it is required further studies for the occurrence of hypercholesterolemia as a outcome of MI.

Heart release cardio-specific enzymes into the blood as a result of myocardial damage, and the amount of enzymes released into the plasma or serum is proportional to the number of heart cells that undergo necrosis[5]. AST among these enzymes has been widely used as a diagnostic aid in cases of MI[8-9]. When the AST value was observed highly in the serum following acute MI, it was considered to indicate the progression of MI, and it was measured even in circumstances of chronic MI[8]. Matsui et al.[8] reported that AST was measured in the infarcted myocardium at 3 weeks after MI, not in serum. In this study, the mean value of AST was highly measured at day 3, 5, 7 and 30. Thus, we confirmed that AST value in serum of patients with MI could be observed in acute and chronic status.

Cardiac markers such as total CK, CK-MB and LDH, which are known early markers for acute MI, are then used to diagnose MI[5,7-8]. Although the total CK value is useful for the detection of skeletal muscle injury or heart disease, it is far too insensitive and nonspecific for use in the diagnosis of acute MI. Therefore, total CK is valuable as a screening test, and serum obtained from patient with abnormal total CK value should be subjected to a CK-MB analysis, as a CK-MB value is very sensitive. The levels of CK-MB have served as essential components for clinical decision in emergency rooms[7]. However, the time course of release and the appearance of these markers in serum must be defined relative to the onset of clinical symptoms. It has been known that total CK, CK-MB and LDH values appear highly in acute MI,

and then disappear in serum after short time[5]. CK-MB, one of MI-specific markers, was released largely to 2 days after onset of acute MI[5]. In this study, we observed that the value of these markers (total CK, CK-MB and LDH) was high due to the release into serum as a result of durable MI.

In conclusion, the results of this study show that acute and chronic MI in rats induced the changes of the RBC, hemoglobin, HCT, AST, glucose, total CK, CK-MB and LDH values in hematologic and biochemical analysis. And these serial values are the results of hematologic and biochemimcal analysis in MI state from day 1 to day 30. Therefore, the eventual value of these results would be suggested for the alterations in blood of acute and chronic MI.

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