Biochemical markers an indirect method for evaluating Delayed Onset Muscle Soreness among recreational athletes

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Abstract: The aim of this study was to determine the role of biochemical markers in identifying the delayed onset muscle soreness among recreational athletes. Methods: This study was conducted in Saveetha University, college of physiotherapy, outpatient department. Eighty recreational athletes participated in this study. For all we measured the biochemical markers, Creatine kinase (CK), and Lactate dehydrogenase (LDH) enzymatic activity at the baseline and Delayed Onset Muscle Soreness (DOMS) were induced to elbow flexors by eccentric loading protocol and again the biochemical parameters measurement were repeated at 24 hours and 48 hours. Results: Paired t test analysis revealed significant differences (p<0.05) between 0 hr and 48 hours, as well as between 0 hr and 24 hours. Conclusion: Serum creatine kinase level and lactate dehydrogenase level remains elevated until 48 hours after inducing delayed onset muscle soreness and these biochemical markers can be used to identify the level of muscle soreness.

Exercise-induced muscle fibre micro injury has been hypothesized to be initiated by disruption of the force generating and/or transmitting structures and loss of sarcolemma integrity followed by a calcium overload phase resulting in an influx of Extracellular calcium (Ca2+) that activates several intrinsic degradative pathways [1, 2, 3]. The specific event that serves to initiate exercise-induced muscle fibre injury is not known. It is generally recognized that this type of injury is associated with eccentric contractions [1, 4, 5]. Damage to the sarcolemma and extra cellular matrix (ECM) creates an altered chemical environment within the muscle. Release of proteins and ions into the plasma as a result of inflammation is similar to that found in acute strains. Increases in these levels indicate damage to the sarcolemma. Elevations of intracellular molecules such as CK LDH, protein metabolites, and myoglobin have been found in plasma up to 48 hours following eccentric exercise. Liberation of these biochemical substances occur from the muscle cells and begin approximately 24 hours post exercise [6], before phagocytic cells enter the injury site. Time-specific clinical events (such as peak soreness at 2 to 3 days) may correspond to the time of increased enzyme levels (such as CK increase at 2 days). While Tiidus [7], reported such a correlation between soreness and enzyme levels, Clarkson et al [6] cautioned against claiming a cause-and-effect relationship based on limited research. Structural disruption leads to the normal inflammatory response: an increase in chemical mediators such as histamine, bradykinin, prostaglandin, and serotonin [8] causing pain and swelling. The products of the inflammatory response sensitize free nerve endings in muscle, thus increasing soreness. Stauber et al [9] concluded that the DOMS after repeated eccentric muscle action is not because of actual myofiber damage, but more likely results from inflammation.

Creatine Kinase (CK) is found predominantly in muscle and is released into the circulation during muscular lesions. Therefore, serum CK activity has been theoretically expected to be useful as a marker in exercise physiology and sports medicine for the detection of muscle injury and overwork [10]. However, previous studies on CK release have not clearly demonstrated its value as a marker for these states [11]. The purpose of this study is to...
2.4.1. Serum CK level:

2.5. Serum LDH level:

3. Results:

All subjects developed DOMS when examined. Participant characteristics of age, weight, height and their initial 1 RM level are presented in table-1 which gives us the information on the homogenous population that participated in the study.

2. Materials and Method

2.2. Eccentric loading protocol

2.2.1. RM calculation:

The subject was asked to lift a fixed weight in his hand from a fully extended to a fully flexed position in standing position. The amount of weight was determined by subject’s perception. Initially 1 Repetitive maximum (RM) calculated by using the formula:

\[ \text{[NO of repetitions + 1] X Wt used}. \]

80 % of 1 RM calculated and used for inducing DOMS. Concentric contractions followed by eccentric contractions for 7 seconds. Assistance given for concentric contractions and no assistance given for eccentric contractions and all the subjects were verbally encouraged. The subjects were instructed to perform 4 sets, 1 set consisting of 10 repetitions, with a rest period of 3-5 minutes between each set.

2.4. Data Collection:

2.4.1. Serum CK level:

Blood samples of 2 ml were collected from all the subjects using a disposable syringe and centrifuge used to separate the serum. Then the blood serum added with 1 ml of CK reagent and kept in CK analyzer and incubated at 37 degree Celsius for 100 seconds. Changes in absorbance / minute (OD / Min) during 3 minutes were measured. The following formula was used to calculate the CK activity:

\[ \text{CK activity (U/L) = (OD/Min) X 4127} \]

2.5. Serum LDH level:

The blood serum was added with 1 ml of LDH reagent kept in the LDH analyzer and incubated at 37 degree Celsius for 1 minute. The Changes in absorbance / minute (OD / Min) during 3 minutes were measured. The following formula was used to calculate the LDH activity:

\[ \text{LDH activity (U/L) = (OD/Min) X 16030} \]

This procedure was done at baseline, 24 hours and at 48 hours.

Our study results showed significant inverse relationship between BMI and HRV parameters like SDNN, E/I ratio and HF nu, but at the same it showed significant positive relation of BMI and LF nu, LF/HF.

Table -1 Anthropometric characteristics of the subjects participated in the study

<table>
<thead>
<tr>
<th>Sample size</th>
<th>Age</th>
<th>Height</th>
<th>Weight</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>80</td>
<td>21.5±1.87</td>
<td>164.85±5.57</td>
<td>62.3±5.10</td>
<td>20.4±4.81</td>
</tr>
</tbody>
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Trend Line over time at baseline, 24 hours and at 48 hours among CK mean values

Trend Line over time at baseline, 24 hours and at 48 hours among LDH mean values

Both Creatine kinase and Lactate Dehydrogenase enzymatic activity started to elevate after inducing delayed onset muscle soreness and after 48 hours the enzymatic activity increased more when compared to baseline and 24 hours values.

4. Discussion

In the present study serum CK was measured at time points 24 hours and after 48 hours to gain an accurate picture of changes after eccentric exercise. The serum CK value was the balance between the value produced by exercise loading and the lost value.
through renal clearance. We hypothesized that there might be a break point of CK release after the same absolute workload exercise, depending on individual physical characteristics. Although variation in each subject after exercise may be due to differences in the degree of physical workload in performing exercise, individual muscle strength is an important factor affecting serum CK activity.

Exercise programs that include eccentric muscle contractions can result in significant serum CK elevations. One study followed 203 participants to evaluate the magnitude of CK elevation and the effect on renal function produced by exercise [12]. After performing 50 maximal eccentric elbow flexor contractions, 55% of participants had CK elevations > 2000 IU/L at 4 days after exercise; 25% had CK elevations > 10,000 IU/L; 13% had levels > 20,000 IU/L. Another study found significant increases in CK (approximate mean of 15,000 IU/L) after repetitive eccentric elbow flexor contractions in college-age males [13]. Commonly accepted mechanisms of CK release are damage to muscle tissue and changes in myocyte membrane permeability. The pattern of CK response after eccentric exercise in the present study is likely be due to additional CK release from damaged muscle tissue. With regard to membrane permeability, there are various theories of ion-distribution change, enzyme deficiency, and ATP depletion. When the exercise intensity is within the normal range of metabolism, the muscle tissue is exercised without marked changes in membrane permeability. However, when the exercise intensity exceeds this permissible range, the membrane permeability temporarily changes, resulting in CK release from the active muscle. The boundary of this permissible range is its break point. In untrained individuals, relatively greater muscle tension was required than in well-trained individuals to complete the same exercise. In addition, mobilization of free fatty acids, which acts as an energy substrate during exercise, tended to be lower in the untrained individuals than in the well-trained individuals. Therefore, the relative exercise intensity for the muscle seemed to rise according to developing muscle fatigue with continued exercise. It was estimated that metabolic enhancement of the glycolytic pathway induces the production of lactic acid. Thus untrained individuals like recreational athletes seem to have exercised beyond this break point, resulting in an increase in serum CK activity [14]. It is also possible that the CK response to exercise depends on the individual's physical characteristics or training background. Therefore, detailed studies are required on the association between serum CK activity after exercise and the body composition and other characteristics of subjects, as well as on the exercise conditions.

It has been widely acknowledged that blood lactate is removed more quickly during active recovery because blood flow remains elevated through the active muscle, which in turn is believed to enhance lactate removal from the muscle cell [15]. This has been researched extensively and has been given the green light as the most prominent way to enhance recovery from excessive lactic acid levels. This may be the probable reason to support the present study, because the data in the present study shows that, Lactate dehydrogenase (LDH) enzymatic values remained elevated for at least 2 days after exercise induced muscle damage.

5. Conclusion
Serum creatine kinase level and lactate dehydrogenase level remains elevated until 48 hours after inducing delayed onset muscle soreness and these biochemical markers can be used to identify the level of muscle soreness.

6. References