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EFFECTS OF ECCENTRIC EXERCISE ON SKELETAL MUSCLE MARKERS IN HUMANS

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ABSTRACT

Most of the studies consider the significance of creatine kinase (CK) and myoglobin (Mb) as the best markers of skeletal muscle injury after high force eccentric exercise (ECE). To confirm this and the activity of other injury markers mainly lactate dehydrogenase (LDH), C-reactive protein (CRP), aldolase, fatty acid binding protein (FABP) and aspartate aminotransferase (AST), eighteen physically active and healthy male Saudis (mean age: 22.5 years (range: 18-27 years); basal body weight: 70 kg (range: 58-78 kg) from Makkah (Saudi Arabia) were included in the ECE program using cycle ergometer. Highly significant post-ECE increase compared to pre-ECE in CK, Mb, LDH, FABP, CRP and AST were recorded whereas no significant variation occurred in aldolase activity.

Key-words: Eccentric exercise, muscular injuries, skeletal muscle injury markers

INTRODUCTION

Several studies consider the significance of creatine kinase (CK) and myoglobin (Mb) as the best markers of skeletal muscle injury after high force eccentric exercise as reviewed by Sorichter *et al*. (1999). The involvement of CK in causing muscle injury after eccentric activity and influencing the other muscle markers, has been suggested as the foremost important factor (Armstrong, 1990; Nosaka and Clarkson, 1996; Ide *et al*., 1997; Sorichter *et al*., 1998; Duarte *et al*., 1999; Schwane *et al*., 2000; Byme and Eston, 2002; Szlachcic *et al*., 2002; Liu *et al*., 2005; Milias *et al*., 2005; Twist and Eston, 2005; Bloomer *et al*., 2006; Ascensao *et al*., 2008; Banasik *et al*., 2008; Lavender and Nosaka, 2008; Pantoja *et al*., 2009).

Myoglobin (Ide *et al*., 1997; Sorichter *et al*., 1998; Rev: Sorichter *et al*., 1999; Banasik *et al*., 2008) and LDH (Nosaka and Clarkson, 1996; Ide *et al*., 1997; Milias *et al*., 2005) are the other major players considered as causing damage after eccentric activity.

The other associated enzymes and biochemicals are CRP (Nosaka and Clarkson, 1996; Milias *et al*., 2005), aldolase (Brancaccio *et al*., 2010), FABP (Sorichter *et al*., 1998) and AST (Nosaka and Clarkson, 1996; Banasik *et al*., 2008). The present study has been undertaken to measure CPK, Mb, LDH, CRP, aldolase, FABP and AST in normal healthy subjects participating in the eccentric exercise program (mentioned elsewhere in this article). The purpose of this study was to assess the change in the mentioned parameters and to interpret the cause of damage occurring in eccentric exercise activity.

MATERIALS AND METHODS

Eighteen physically active and healthy male Saudis from Makkah took part in the present study. Average age of these subjects was 22.5 years (range: 18-27 years) and average basal body weight of 70 kg (range: 58-78 kg). All subjects participated in the exercise program with informed consent. The volunteers were asked not to carry out any damaging exercise or strenuous physical activity a week prior to the start of eccentric exercise of cycling till the collection of blood samples.

No any subject was under-medication or involved in any physical training or athletic program. All participants were active and healthy with no history of cardiac, muscular, neurological, pulmonary, renal or any other disorder and genetic/ familial disease.

The standard cycling exercise test using cycle ergometer was carried out by the subjects starting with the specified work at 60 revolutions per minute with work increase for every 3 minutes till the occurrence of exhaustion.

Blood samples from all subjects were taken before the start and immediately after the eccentric exercise session. Blood samples were allowed to clot at room temperature and were centrifuged at 3000 rpm for about 10 minutes. The serum was collected and stored at -80º C to be analyzed later. Measurement of serum CK was done at 37º C using commercial kits. Serum Mb, aldolase, CRP, FABP, LDH, aspartate aminotransferase and carbonic anhydrase were measured by using the respective commercial kits.

RESULTS AND DISCUSSION

The effects of eccentric exercise program on muscle injury in human volunteer male subjects was pre- and post ECE determined (Table 1).

Serum CK showed highly significant increase in post-ECE ($p<0.0001$; Table 1). This parameter was found most significant as compared to all other parameters assessed. The significance levels for other parameters were as $p=0.0053$ for LDH (IU/ L) , $p=0.0041$ for CRP (mg/ L), $p=0.3238$ for aldolase, $p<0.0001$ for Mb(µg/ L), $p=0.0018$ for FABP (µg/ L) and $p=0.0001$ for AST(IU/ L).

Table 1. Effects of eccentric exercise on skeletal muscle injury markers in humans.

Skeletal muscle markers	**Pre-ECE** (n: 18) (X ± SE)	**Post-ECE** (n: 18) (X ± SE)	**Significance (p)**
CK (IU/ L)	75.39± 8.34	260.23 ±17.15	t =9.693; p<0.0001
LDH (IU/ L)	160.52± 12.34	221.33±16.26	t =2.979; p<0.0053
CRP (mg/ L)	3.24±0.624	6.89±1.009	t =3.076; p<0.0041
Aldolase (IU/ L)	0.48±0.064	0.59±0.089	t=1.001; p<0.3238
Mb (µg/ L)	39.46±3.14	96.61±6.651	t=7.769; p<0.0001
FABP (µg/ L)	3.28±0.502	6.03±0.641	t=3.377; p<0.0018
AST (IU/ L)	16.05±1.471	27.20±1.966	t=4.534; p<0.0001

The values are Mean±SE; ECE refers to eccentric exercise; n is the total number of subjects; CK, LDH, CRP, Mb, FABP and AST are abbreviations respectively for creatine kinase, lactate dehydrogenase, C - reactive protein, myoglobin, fatty acid binding protein and aspartate aminotransferase.

All evaluations (Table 1) showed significant increase in post-ECE compared to pre-ECE except aldolase (IU/ L) that did not differ significantly in post-ECE, though a non-significant increase was obtained for aldolase in post-ECE. The order of the significance in descending order was: CK>Mb>AST>FABP>CRP>LDH. Hence, the most significant biochemicals found to be highly involved in causing skeletal muscle injury in eccentric exercise activity in the present investigation were CK and Mb. The mean± SD values for each of the skeletal muscle injury marker in pre-ECE and post-ECE are given in Table 1.

There is a controversy for the precise involvement of a variety of factors considered as involved in causing injury in eccentric exercise. This disagreement is of a variety of nature. The difference in the extent of involvement of various biochemicals is also present. First, the methodological details of the eccentric exercise programs differ in different reports. There are rarely the set of experiments in literature wherein same or almost same strength of eccentric work or activity was assessed and associated with other variables. It is essentially required to have a clear knowledge of causal factors (especially the extent of their involvements) for a physiological and pathophysiological interpretation of injury occurring in eccentric exercise activity. The second important issue is to use same or similar methods in a series of experiments for the clinical, physiological, biochemical, cellular or molecular determinations. This might help elucidating the underlying causal factors in specific conditions as well.

The other associated enzymes and biochemicals are CRP (Nosaka and Clarkson, 1996; Milias *et al*., 2005), aldolase (Brancaccio *et al*., 2010), FABP (Sorichter *et al*., 1998) and AST (Nosaka and Clarkson, 1996; Banasik *et al*., 2008).

Indeed standardizing a certain procedure in clinical human studies is not quite easy task compared to well organized studies in animals or model studies; there is still a need to make coherence in interacted clinical study programs. This might lead us to associate it for muscular overload after eccentric exercise with the structural injury of contractile apparatus observed as Z- line streaming as well as myofibrillar disruption (Sorichter *et al*., 1999).

There is a need to do more specific work in a progressive study of eccentric activity via including concentric activity or less intense eccentric exercise and comparing and evaluating the comparative influences of various factors in causing and inhibiting the muscle injury. Lavender and Nosaka (2008) found no change in CK after

excessive exercise in those subjects who went through light eccentric activity. Similarly, the CK activity in a group of boys of average 13 years age did not show adult like increase in high level eccentric activity, and that did not show CK as a marker of eccentric exercise (Duarte *et al*., 1999).

In view of the described reasons, the current report comprised only a very specific eccentric exercise program and incorporated same methods of measurement of enzymes and other biochemical parameters. These set of experiments are in continuation with each other and apply same exercise protocol, experimental protocol and methodologies (Rakkah NIA, Unpublished Reports). Conclusively, the present study is helpful in physiological and pathophysiological perspective to understand the significance of skeletal muscle injury markers and for the proper management of the control of muscle injuries caused by excessive eccentric muscular activities.

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