Recovery Does Not Prevent Myocardial Damage Due to Overtraining (Biomolecular and Pathobiology Studies)

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Abstract—Overtraining the body with exercises and without adequate recovery time would lead to various health conditions. The overtraining conditions, usually leads to oxidative stress which damages the body tissue. This study aims to analyze the effect associated with a recovery duration of three and seven days in order to prevent myocardial damage due to overtraining. The research made use of a post experimental test of 32 rats which were divided into 4 groups namely 1) proportional sport, 2) overtraining, 3) overtraining with recovery of 3 days and 4) overtraining with recovery of 7 days. Histopathology examination was performed at the end of the experiment period on the rats. The results obtained has it that there were myocardial damage in the left ventricular hypertrophy, necrosis and chromatin condensation of these rats. Posttest comparative analysis was done to analyze and obtain a posttest mean for each group. For MDA myocardium concentration, Analysis Of Variance (ANOVA) results showed F to be equal to 6,248 with p = 0.002; SOD myocardium concentration was F = 19.519 with p = 0.0001; Left Ventricular Hypertrophy Index was F = 140.732 with p = .0.0001; Index of myocardium necrosis between groups, was F = 201.911 with p = .0.0001; Chromatin condensation index on the cardiomyocyte between the study groups was F = 524.466 with p = 0.0001. Post hoc tests showed different significant concentration at MDA and SOD without any different in the left ventricular hypertrophy, necrosis and chromatin condensation. The results of this study proves that there is no effect in myocardial damage as a result of overtraining from three and seven days, notwithstanding the fact that recovery decreases MDA myocardium concentrations, increases SOD myocardium concentrations, but does not prevent left ventricular hypertrophy, necrosis and chromatin condensation.

Keywords— recovery, myocardium damage, overtraining

I. INTRODUCTION

Overtraining or overloading exercises is often experienced when an athlete is preparing for a competition either with a limited timeframe or with adequate timeframe. Overtraining the body without adequate recovery periods, would lead to some myocardium damages on the athlete. According to [21] and [18], an athlete's body condition is very vulnerable if faced with overload dose of exercise without adequate rest at intervals.

Furthermore, during the period of overtraining training, Reactive Oxygen Species (ROS) is released from the body. This ROS released, exceeds the protective capacity of the endogenous antioxidant system and causes deregulations in the inflammatory system, oxidative phosphorylation and neuroendocrine [5]. Several studies has it that increased exercise intensity increases the oxidative stress and free radical production in cells [2].

Overtraining is a condition associated with oxidative stress which affects various organs of the body [18]. The study of the effects of overtraining on cardiac organ is debatable. However, in some studies on cardiac myositis, overtraining leads to pathological changes in the heart. Animal test studies was carried out to examine the causes of sudden death in athletes as a result of cardiac organ caused by the hypertrophy of cardiomyopathy. Hypertrophy of cardiomyopathy is defined as a congenital aberration, obtained through autopsy results which in some cases is one of the major causes of sudden cardiac death in athletes. This is said to be one of the causes of sudden cardiac death on athletes though this analysis is yet to be properly documented. Athletes are described as those individuals in a community that are more fit and healthy than non-athletes. The presence of sudden cardiac death in athletes has seized public attention. This is simply because it is a paradoxical ailment, leaving the public to assume the health status of the athletes [14]. Through autopsies performed in some cases of sudden cardiac death in athletes, cardiomyopathy hypertrophy is reported to be the major causes of sudden death in young athletes [8], [5], [10]. However, in cases where autopsy was not conducted on the body of these young athletes, the cause of death cannot be ascertained. Similarly, experiment carried out using animals, has shown incorrect training to cause changes in heart rate and stroke volume of the heart. In this study, myocardium damage to histologic
overtraining will be demonstrated on the basis of patho-
mechanism that begins with the formation of excessive ROS. 
Damage to myocardium which occurs as a result of 
overtraining is suspected to result in an increased risk of 
cardiovascular diseases. Proper training management to 
avoid overtraining is carried out by decreasing the training 
load and recovery time [12]. Based on previous researches, it 
is evident that ROS is an underlying etiology used to 
determine the body system adaptation to sport. However, its 
major failure in adaptation is patomechanism in cardiac 
organ, as well as the inability to effectively explain the 
effectiveness of the recovery period using some previous 
studies. Recovery will induce the body’s hormonal ability to 
decrease the ROS in the system and enable it meet the 
production capacity of endogenous antioxidants, one of 
which is Superoxide Dismutase (SOD). SOD is an 
endogenous antioxidant formed by mitochondria. [1], [7]. 
Detraining or recovery is defined as a management 
timeframe given in overtraining conditions. Recovery will 
restore the exhausted cells so that it can return to its 
homeostatic conditions [12], [4]. The basic recovery 
principle is the recovery process which is dependent on the 
type and duration of physical stress within the given training 
period [3]. The right recovery duration required by the body 
cells is explained through several recovery mechanisms 
studies that occur in the skeletal muscle. Recovery is 
tended to restore phosphor creatin, glycogen and to 
increase blood flow for oxygen consumption (reperfusion) 
[15], [9], [11]. The time required for the recovery process is 
dependent on the intensity of the exercise, as well as the type 
and frequency of the exercise. In a study conducted on 
mouse to ascertain the quantity of phosphocreatine reserved 
during exercises, it was proven that there was a significant 
difference between 24 hours and 72 hours recovery. The 
study analyzed that phosphocreatine reserves were higher 
during recovery with a duration of 72 hours [12]. This study 
analyzes the effect associated with recovery with regards to 
the protection of heart organs in overtraining conditions. The 
duration of recovery used in this study is same as those used 
in previous studies (72 hours or 3 days) and modification of 
extension of 7 (seven) days. The research examines the 
damages caused by myocardium in overtraining, as well as 
the proper effect of recovery in the exercise program, 
thereby, making this study to aim at analyzing the effect of 
recovery on myocardial damage as a result of overtraining 
during exercises. Obtained results from this study is likely to 
give birth to a step in the preparation of new protocols in the 
management and training of athletes against cardiovascular 
diseases.

II. METHOD

The methodology used in carrying out this research is the 
"randomized post-test only control group design". Data were 
collected at the end of the study by comparing the various 
outcomes in overtraining treated groups, overtraining with 3 
(three) days’ recovery and 7 (seven) days recovery with the 
untreated group (proportional exercise as control group). Four treatment groups were randomly created these are 1 
(one) control group (proportional sport), 1 (one) group 
without recovery treatment overtraining, 1 (one) group 
overtraining treatment with recovery for 3 days and 1 (one) 
others group were given overtraining treatment a recovery 
period of 7 days. The study made use of male white rats 
(Wistar Ratus Norwegicus) between the ages of 3-4 months, 
and weighing between 180-220 grams. The research was 
approved by the Research Ethics Commission of Faculty of 
Medicine, of Sebelas Maret University No: 717 / VIII / 
HREC / 2016. The treatment given to mice was reinforced in 
a swimming pool of 50x20x60 cm (Giri, 2017). In the 
control group: the mice was allowed to swim in the afternoon 
for approximately 15 minutes after an hour of eating and 
drinking This was done throughout the days (1,2,4,5,7 every 
week). However, on the third and sixth day they were 
allowed to rest. The treatments were carried out for 8 weeks. 
At the end of the treatment, an autopsy was carried out on 
the mice to ascertain the condition of the blood and the heart 
organs. The overtraining treatment group was swim 2 (two) 
times daily (except on the 7th day) until fatigue occurred and 
they could not swim again. The treatment was administered 
for 8 weeks. At the end of the treatment, an autopsy was 
immmediately carried out on the mice to ascertain the blood 
and the heart organs The overtraining treatment group with 3 
(three) recovery experienced the same treatment with the 
overtraining group and was given a rest period of 3 (three) 
days at the end of the treatment period. The overtraining 
treatment group with recovery 7 (seven) days, experienced 
the same treatment with the overtraining group and given a 
rest period of 7 (seven) days at the end of the treatment 
period an autopsy was carried out to ascertain the condition 
of the heart. Histologic preparations was done by making 
incisions in the heart muscle using microtome. The 
preparations were the stained with a Hematoxylin-Eosin 
(HE) staining technique to facilitate observation using a light 
microscope with 40 times magnification. This was used to 
assess hypertrophy, necrosis and increase the chromatin 
activities occurring in the myocardium. To detect the 
concentration of protein the mitochondria of MDA and SOD, 
the ELISA technique was used which precipitated antibodies 
as well. The data obtained were analyzed using the one-way 
ANOVA parametric test method followed by a Post Hoc 
Least Significant Difference (LSD) test.

III. RESULTS AND DISCUSSION

Based on the results of the study obtained, the mean 
MDA concentration of myocardium was found to be in the 
overtraining exercise group. It was found that the seven days 
recovery was lower than the control group (proportional 
sport) and those of the other two treatment groups with a 
control group mean MDA of 86.45 ng/ml, with an 
overtraining group of 112.90 ng/ml. The overtraining 
exercise group with a three-day recovery was found to be 
109.53 ng/ml and the seven-day overtraining exercise group 
had a value of 69.27 ng/ml.

The measurement result of SOD Miocardium concentration in this study showed that the mean SOD 
myocardium concentration in the control group (sport 
proportional) was higher than the other three treatment
groups. The mean SOD myocardium control group was found to be 1516.05 ng/ml, the overtraining exercise group was 829.94 ng/ml, overtraining exercise with 3 (three) days recovery was 115.09 ng/ml and group of overtraining exercise with 7 (seven) days recovery was 939.44 ng/ml.

Description of observation and measurement result using a microscope showed that the overtraining exercise group with 3 (three) days recovery had the largest index of cardiomyocyte hypertrophy with a 77.625% compared to the mean result of the hypertrophy index control group which had a percentage of 26.5%. The overtraining exercise group had a percentage of 75.5% and the overtraining group with 7 (seven) days recovery was 75.125%.

Based on the observation of the anatomical pathology it can be seen that in the control group the index of myocardium necrosis was lower than the other three treatment groups. Signs of necrosis in the form of hypereosinophilic and curolisis, picnosis, curolisis, inflammatory cells and remnants cell was seen. The histopathological features of the necrosis are presented in following figure.

Using One Way ANOVA (Analysis Of Variance) test, the differences between groups were found. This was then followed by LSD (Least Significant Difference) test which presented some of the following as a result:

1. The damage on myocardium is proportional to the amount of exercise, which is better than in the overtraining exercise. Although it has been given recovery because it resulted in lower MDA myocardium concentrations.

2. The condition of myocardium damage is proportional to the amount of exercise, and better than the overtraining exercise despite its given recovery state, since it shows the concentration of endogenous antioxidants (SOD) in the myocardium is higher.

3. The condition of myocardium damage in proportional exercise is better than the overtraining exercise load though it has been given recovery because it resulted in a lower left ventricular myocardium hypertrophy index.

4. The condition of myocardium damage is proportional to exercise and is better than the overtraining exercise despite its recovery state owing to its results in a lower Myocardium Necrosis Index.

5. Myocardium damage in proportional to exercise and it is better than the overtraining exercise despite its recovery timeframe. This is because it resulted in a lower cardiomyocyte chromatin condensation index.

IV. CONCLUSIONS

The results of this study proves that there is no overtraining effect on myocardial damage in three and seven day recovery. Although recovery decreases such as MDA myocardium concentrations, increases SOD myocardium concentrations, it does not prevent left ventricular hypertrophy, necrosis and chromatin condensation...
REFERENCE


