Myocardial pathological changes in overtraining exercise

Made Kurnia Widiastuti Giri,¹ Ketut Indra Purnomo,¹ Muchsin Doewes,² Bambang Purwanto,² Ambar Mudigdo³

ABSTRACT

Background: An overtraining or a practice of an excessive intensity without a sufficient recovery period may induce myocardial injury. Objective: The aim of this study was to reveal myocardial changes in overtrained rats through a histo-pathobiological analysis. Methods: We enrolled 24 male Wistar rats for 6 weeks. As many as 8 rats or the control group swam 15 minute/day, 5 days/week. The overtrained group consisted of 24 rats swam for 90 minutes, twice a day, daily. After sacrificing, the hearts were excised for pathological preparation slides.

INTRODUCTION

Numerous studies showed that an exercise prevents degenerative and cardiovascular diseases, and has an anti-aging effect.¹⁻³ An overload training is sometimes endured by sports athletes to prepare themselves for a competition.⁴ When not accompanied by an adequate recovery period; an exercise overload causes an overtraining. During the training period of an overtraining, Reactive Oxygen Species (ROS) may be discharged exceeding the protective capacity of the anti-ROS system. Thus, causing a dysregulation of the inflammatory, oxidative phosphorylation, and neuroendocrine systems.⁵⁻⁶ Increasing the intensity of an exercise has been shown to increase the free radical production in a cell.⁷⁻⁸ Overtraining may also cause a muscle damage in the rarely exercising people who experienced a sudden overload training.⁹⁻¹⁰

The cases of a sudden cardiac death in athletes had not been well documented, but do exist.¹¹⁻¹² The incidence of sudden cardiac death in athletes in the United States varied greatly from 1:23,000 to 1:300,000 athletes per year.¹²⁻¹⁶ Some animal studies showed cardiac myocytes changes happened in overtraining. Myocardial damage occurred in overtraining may induce a sudden cardiac death in an exercise.¹⁷⁻¹⁹ We analyzed histopathological changes of the myocardium in rats with overtraining exercise to see the effect of overtraining in the myocardium.

METHOD

We conducted an in vivo rat hemodynamic study. The samples were 24 young adult male Wistar rats, each weighted 80 to 225 gr.¹⁷ For six weeks, the rats were housed in a room with a constant temperature of 22±2°C with a 12/12 hours light-dark cycle. They were fed a standard laboratory rat diet.

The rats were divided into two groups: control and overtrained groups. The control group consisted of 8 rats, received a proportional exercise. The overtrained group consisted of 16 rats, was treated with an overtraining protocol modified from Olah's study protocols.¹⁸⁻¹⁹ In a rat swimming pool, the control group, swam for twenty minutes per day, five days a week. The overtrained group swam for ninety minutes, twice a day daily. After six weeks of treatment, on the last day of the treatment, the rats in both groups were sacrificed. Each heart was made into 2 slides. The left ventricle was cut into 3 transverse sections: apex, middle ring, and base. From the middle ring, 5μm sections were cut and stained with Hematoxylin-Eosin. Two independent histopathology experts separately examined the myocardium slides for histopathological changes.

RESULTS: The overtrained group’s slides showed an increase in chromatin fragmentation in the myocardial structure. The left ventricle wall thickness was also greater in overtrained groups than the control. Necrotic bodies were found in the myocardium of overtrained groups, but none in the control group.

CONCLUSION: We found the negative effects of an overtraining to the myocardium. Avoiding overtraining is necessary to avoid a myocardial injury.

Keywords: overtraining, myocardial injury, overtraining histopathology


¹Lecturer, Faculty of Sports and Health Sciences, Universitas Pendidikan Ganesha, Singaraja, Bali, Indonesia
²Lecturer, Department of Medical Sciences, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia
³Clinical Pathology Specialist and Lecturer, Department of Medical Sciences, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

¹¹Correspondence to: Made Kurnia, MD, Faculty of Sport and Health, Universitas Pendidikan Ganesha, Singaraja, Bali, Indonesia dmnia82@gmail.com

Received: 2017-03-16
Accepted: 2017-08-28
Published: 2017-09-1

Open access: www.balimedicaljournal.org and ojs.unud.ac.id/index.php/bmj

Published by DiscoverSys
The slide conclusion was based on 5 visual fields observation using a 100-magnification.

RESULT

Both groups (48 slides) showed a left ventricle hypertrophy (LVH). The hypertrophy area in the overtrained group was larger than the control group as shown in Figure 1 and Figure 2. Moreover, the left ventricular wall thickening was thicker in the overtrained group than in the control group. The wall thickness of the control group showed a moderate LVH, while the overtrained group showed a severe LVH.

In 14 out of 32 slides of the overtrained group, the left heart ventricle showed a necrosis. There were nucleus changes such as loss of chromatin and a wrinkled nucleus. The nucleus became more solid and darkened (pyknosis), divided into fragments, and shredded (karyorrhexis). A necrotic area was shown in Figure 3.

An increased chromatin activity as shown in Figure 4 was present in 6 out of 32 slides in the overtrained group.

DISCUSSION

In rat experiments, an overtraining-induced changes in the kidney function and caused a skeletal muscle fatigue. Previous studies showed an excessive apoptosis in overtrained rats' cardiomyocytes. A study by Benito with a sedentary rat group and another group which rats ran in a 60 cm/second treadmill for 60 minutes for 5 days a week showed a concentric LVH after 8 weeks and at the end of the sixteenth week the hypertrophy became eccentric. A study investigating the effects of an anaerobic and aerobic exercise in rat myocardium
showed that signs of hypertrophy in myocardicocytes appeared on day 3, signs of ischemia on day 7, and infarction appeared on day 10 in both groups.27 Our study showed a left ventricle hypertrophy as a myocardium structural compensation in both groups. Known as a physiological response to an exercise, an LVH may appear after some exercises. However, an excessive wall thickness leads to a structure rigidity. Thus, it initiates a myocardial contraction impairment. Our overtrained group showed a pathological hypertrophy of the left ventricle.

Our study revealed cardiomyocyte necrosis and an increased chromatin activity. It can be assumed the overtrained rats had pathological signs of myocardial damage. Necrotic cardiomyocytes can be found in the end stage of an ischemic cardiac injury. A massive cardiomyocyte necrosis manifests as a rapid worsening of the clinical condition. However, our study could not explain what underlay the condensed chromatin. To better explain the pathomechanism of an overtraining, more studies are needed.

CONCLUSION

An overtraining may be harmful to the heart and may lead to a rare sudden cardiac arrest. Histopathologically, an overtraining in rats causes an excessive left ventricle hypertrophy, necrosis and an increased chromatin activity in the myocardium.

ACKNOWLEDGEMENT

The authors are grateful to the researchers who had helped greatly in the study:

1. Suradi, a professor at Sebelas Maret University who supervised and helped in designing the study.
2. Wiranatha, a laboratorian who maintained the rats in Udayana University.
3. Brian Wasita, a histopathology specialist who examined the slides and did a software analysis to the pathological changes.
4. Dono Indarto, whose suggestions improved the quality of the final manuscript.
5. The reviewer and the UPI ICSSPE’s team.

REFERENCES

2. D’Silva A, Sharma S. Exercise, the athlete’s heart, and sudden cardiac death. The physician and sports medicine. 2014;42(2):100-13


This work is licensed under a Creative Commons Attribution