

## Echocardiographic Findings in Power Athletes Abusing Anabolic Androgenic Steroids

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- A** Concept / Design
- B** Acquisition of Data
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- D** Manuscript Preparation
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- F** Funds Collection
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Received: Apr 28, 2012

Accepted: Jun 10, 2012

Available Online: Sep 29, 2012

## Abstract

**Purpose:** Anabolic androgenic steroids (AAS) abuse for improving physical appearance and performance in body builders is common and has been considered responsible for serious cardiovascular effects. Due to disagreement about cardiovascular side effects of these drugs in published articles, this case control study was designed to evaluate the echocardiographic findings in body builder athletes who are current and chronic abusers of these drugs.

**Methods:** Body builder athletes with continuous practice for the preceding two years and were training at least twice weekly were selected and divided into AAS abuser and non user and compared with age and BMI matched non athletic healthy volunteers (15 cases in each group).

**Results:** There was no significant difference in left ventricular size or function either systolic or diastolic in comparison to cases and control groups. The only difference was in diastolic size of septum and free wall but observed differences were only significant ( $P = 0.05$ ) between first (athletic with AAS abuser) and third group (non athletic and nonuser). The difference between the above-mentioned indexes were not significant between two groups of athletes.

**Conclusion:** Observed differences in diastolic size of septum and free wall is in favor of that long term abuse of anabolic steroid results in accentuation of physiologic hypertrophy due to long term sport most probably due to higher rate pressure product. Furthermore long term abuse and supra pharmacologic doses do not have significant effect in size and left ventricular function.

**Key Words:** Androgenic Anabolic Agent; Body Builder; Left Ventricular Hypertrophy; Echocardiography

*Asian Journal of Sports Medicine, Volume 4 (Number 1), March 2013, Pages: 10-14*

## INTRODUCTION

Anabolic androgenic steroids (AAS) abuse for improving physical appearance and performance in body builders has been considered responsible for sudden cardiac death, malignant ventricular arrhythmia, myocardial infarction, left ventricular hypertrophy, reduced left ventricular function, arterial thrombosis and pulmonary embolism [1].

AAS are usually synthetic derivatives of testosterone with both anabolic and androgenic effects. A limitation of human studies is represented by the fact that information about the intake of anabolic steroids

are, generally, self reported and it is hardly possible to assess the exact dosage. AAS are often used in combination with other drugs or substances, so it is difficult to separate their toxic effects from those caused by the other drugs abused. Many of these side effects are only reported in case reports [2-5] and have not been systematically investigated. Experimental studies for cardiovascular effects of anabolic steroids in some animal studies have shown that supraphysiologic doses of AAS administration lead to structural and functional disturbances of myocardial cells in rabbits and rats [6-8].

The true incidence of AAS related medical problems

is not known due to several shortcomings in human studies. D'Andrea and his coworker in their study about chronic misuse of AAS concluded that several years after chronic misuse of AAS, body builders show a subclinical impairment of both systolic and diastolic myocardial function, strongly associated with mean dosage and duration of AAS abuse [9]. Also Montisci et al in a case control study concluded that left ventricular mass was significantly higher in AAS users than in AAS non-users and controls. There was some disturbance in relaxation without significant change in systolic function [10]. In contrast to these studies Hartgens et al in a prospective study showed that short term (8-16 weeks) misuse of AAS did not lead to significant functional or structural cardiac side effects [11]. Dickerman and his coworkers in a case control study showed that long term use of AAS leads to mild hypertrophy without significant change in systolic or diastolic function [12].

Data primarily from Framingham's heart study have shown left ventricular hypertrophy (LVH) either physiologic or pathologic is an independent risk factor for arrhythmia and sudden cardiac death [13, 14]. Trans thoracic Echocardiography is the most useful noninvasive tool for evaluation of LVH and myocardial function. Septal or free wall thickness of more than 11 mm is abnormal. Heavy exercise in professional athletics and especially isometric exercise leads to physiologic LVH but whether anabolic steroids accentuate this hypertrophy or not is not clear [12]. The entity of side effects depends on the sex, the dose, the duration of treatment, whether they are taken during exercise training or under sedentary conditions; and the susceptibility of the individuals themselves to androgen exposure partly depending on genetic factors [15]. The current study has been designed to evaluate the effect of anabolic androgenic steroid misuse in body builder athletes on myocardial function and LVH.

## METHODS AND SUBJECTS

Study cases were selected from more than hundreds of body builder athletes and more than ten training

centers. Thirty body builder athletes with daily exercise for two hours and at least five times per week for the preceding two years were selected and divided into two groups (15 cases in each group): group one with chronic anabolic steroid abuse for the preceding two years and at least twice weekly or eight times per month of testosterone or nandrolone injection or daily oxymetholone ingestion and group two, which included athletes without history of anabolic androgenic steroid abuse. The history of long term sport and AAS abuse was taken from the athletes themselves and their coach and a questioner used for recording of data. For ethical reasons and also because the use of AAS usage is unethical and forbidden and also for better cooperation of the athletes it was promised to them that this data is just for research purpose and all data will remain secret. As a control group 15 healthy sedentary volunteers with same age and BMI of previous two groups were selected.

A routine physical examination including vital signs, cardiovascular and general physical examination was performed in all cases. Twelve lead resting electrocardiograms were taken in all cases. Only cases without any evidence of systemic or cardiovascular disease were selected for the study. Excluding criteria were history of hypertension, use of antihypertensive drugs, any evidence of valvular stenosis and preexisting bundle branch block in electrocardiogram. The above mentioned excluding criteria lead to impairment in the echocardiographic findings.

Two-dimensional color Doppler echocardiography was done by an expert cardiologist who was unaware of the clinical history of the cases. Routine chamber sizing in standard mode and wall thickness measurement in systole and diastole were performed. Echocardiographic examination included the determination of the aortic root (Ao), left atrial (LA), left ventricular end systolic (LVESD) and left ventricular end diastolic (LVEDD) dimensions. Interventricular septal thickness (IVS) and posterior wall thickness (PW) in diastole and systole and right ventricular diameter (RVD) were measured. Left ventricular global ejection fraction was estimated. For assessment of the diastolic function measurements of E and A peak velocities and a calculation of the E/A ratio was used. Ejection fraction was estimated by visual

**Table 1:** Mean and standard deviation of variables in study groups

| Variable                             | Group 1<br>Mean (SD) | Group 2<br>Mean (SD) | Group 3<br>Mean (SD) |
|--------------------------------------|----------------------|----------------------|----------------------|
| <b>Population</b>                    | 15                   | 15                   | 15                   |
| <b>Age</b>                           | 23.20 (3.58) *       | 21.20 (3.65)         | 23.20 (3.52)         |
| <b>Body Mass Index</b>               | 27.10 (1.85) †       | 24.70 (2.19)         | 26.20 (2.03)         |
| <b>Exercise duration (years)</b>     | 4.10 (1.51) ‡        | 4.00 (1.54)          | 0                    |
| <b>Duration of AAS abuse (years)</b> | 2.2                  | 0                    | 0                    |

\* *P* value = 0.2 ; † *P* value > 0.05; ‡ *P* value = 0.9; AAS: Androgenic anabolic steroids / SD: Standard Deviation

evaluation in different short and long axis views. Simpson and other methods were not useful in exact visualization of endocardial layers in all cases due to obesity or chest wall thickness.

For statistical analysis (because of limited data) non parametric tests (Friedman and  $X^2$ ) were used for comparison of quantitative and qualitative data and in each finding a *P*-value less than 0.05 was significant.

## RESULTS

Table 1 represents demographic characteristics of cases in each group. It is clear there is no significant difference between groups.

Echocardiographic findings are shown in Table 2. Ejection fraction in all groups was normal and there was no significant difference between groups. Left ventricular end systolic and diastolic dimensions in group one were slightly higher than group two and three but this difference was not statistically significant. Mean septal and free wall thickness during

systole and diastole in group one and two were more than group three. The observed differences in group one were mildly more than group two, whereas the differences between group one and group three were statistically significant.

As it is shown in Table 1, the systolic septal thickness in group one is significantly more than the other groups. Diastolic septal and free wall thickness in group one is more than group three. End systolic and diastolic dimensions and fractional shortening were relatively similar between all groups.

Table 2 represents Mitral flow (E/A ratio). It is clear there is no significant change in diastolic function. Other echocardiographic findings were mitral valve prolapse in 3 cases in group one, 8 cases in group two and 4 cases in group three. There was one mild case of mitral regurgitation in each group. There was no aortic or pulmonic valve abnormality. There were two cases of minimal tricuspid regurgitation in group one with normal pulmonary arterial pressure.

There was no asymmetrical septal or free wall hypertrophy in any of the cases and there was no regional wall motion abnormality.

**Table 2:** Mean and Standard Deviation of Echocardiographic Findings

| Variable                                    |                 | Group 1<br>Mean (SD) | Group 2<br>Mean (SD) | Group 3<br>Mean (SD) | <i>P</i> Value |
|---|-----------------|----------------------|----------------------|----------------------|----------------|
| <b>Left Ventricular End Diastolic</b>       |                 | 48.8 (4.3)           | 45.4 (4.1)           | 47.2 (2.6)           | 0.6*           |
| <b>Left Ventricular End Systolic</b>        |                 | 33.8 (3.5)           | 31.9 (3.9)           | 32.00 (2.3)          | 0.2*           |
| <b>Septal thickness</b>                     | <b>Systole</b>  | 14.8 (1.9)           | 13.3 (1.5)           | 13 (1.6)             | 0.05*          |
|   | <b>Diastole</b> | 11.3 (1.4)           | 11.1 (1.5)           | 9.9 (1.6)            | 0.05‡          |
| <b>Left Ventricular Free Wall Thickness</b> | <b>Systole</b>  | 13.9 (2.0)           | 14.4 (1.3)           | 13.3 (1.5)           | 0.2‡           |
|   | <b>Diastole</b> | 12.0 (1.8)           | 11.5 (1.5)           | 10.5 (1.7)           | 0.05‡          |
| <b>Ejection fraction (%)</b>                |                 | 62.3 (3.7)           | 60.0 (3.8)           | 60.3 (4.0)           | 0.2            |
| <b>Fractional shortening</b>                |                 | 31.4 (2.50)          | 29.2 (2.86)          | 31.6 (2.6)           | 0.06           |
| <b>E/A ratio</b>                            |                 | 1.5 (0.1)            | 1.6 (0.2)            | 1.7 (0.1)            | 0.08           |

\* Between 1 and other groups; ‡ between 1 & 3 / SD: Standard Deviation

## DISCUSSION

In the current study there was not any statistically significant difference in LV systolic and diastolic dimensions between cases and control groups. Systolic and diastolic function in all groups was relatively similar and it is suggestive of no effect, or minimal effect of chronic anabolic steroid abuse on size, function and stiffness of the heart. These findings are comparable with Palatini et al [16] who in his case control study on bodybuilder abusers of AAS, in comparison with control, did not observe any significant differences in size and function of the heart.

Di Bello et al [17] in evaluation of 10 athletes and Urhausen et al [18] in echo evaluation of 21 bodybuilders have found similar results. Another study by Hartgens F. et al [11] on effects of short time (8-16 weeks) abuse of AAS on 32 bodybuilders was similar and did not show any significant effect of AAS on myocardial function. In contrast to the aforementioned studies, De piccolo et al [19] in echo evaluation of LV mass, thickness, systolic and diastolic dimensions showed significant differences in data between AAS abusers, nonuser athletes and healthy volunteers. These differences may be due to genetic differences in susceptibility of the individuals themselves, to androgen exposure or may be due to changes in dose or type or duration of AAS abuse, and whether they are taken during exercise training or under sedentary conditions. Another important difference was that patients with other medical or systemic disorders (like hypertension) that may lead to secondary echocardiographic changes, unrelated to AAS abuse directly, were excluded.

Another interesting finding is the increase in diastolic thickness of septum and free wall in bodybuilders in comparison with non athletes. This shows the positive effects of heavy weight training exercise on myocardial hypertrophy. Although these changes are not significant statistically but they may be important clinically.

These findings are relatively comparable to the Dickerman et al [20] and Karila et al [21] findings. Dickerman and his coworkers in evaluation of 16 athletes showed that AAS lead to increased LV hypertrophy and decreased LV compliance without significant effects on systolic function of the heart [20].

Karila et al's evaluation of 20 bodybuilders from abusers and nonusers of AAS has shown that there is no significant relation between LV mass and AAS abuse [21]. These findings also are in favor of physiologic effects of heavy isometric exercise on myocardial cell hypertrophy with minimal additive effect of AAS abuse on this hypertrophy.

Findings in the current study showed significant LV hypertrophy in group one in comparison to group three. It suggests that chronic abuse of AAS in supraphysiologic doses leads to accentuated hypertrophic response of the heart to strength training exercise. As mentioned by Grace F. et al [22], this accentuated hypertrophic response may be due to increase in rate pressure product and higher blood pressure leading to more hypertrophic response.

In previous studies McKillop et al [23] found similar results. In a comparison study of 23 bodybuilders, including abusers and nonusers of AAS and non athletes, it was observed that septal and free wall thickness in AAS abusers in comparison to other groups significantly increased. In contrast to the above study Salke et al [24] observed that septal and free wall thickness in bodybuilders increased relative to non athletes, and either abuse or non use of AAS has no additive effect in this hypertrophy. This effect can just be seen in individuals who do weight training exercise chronically. So the additive effect of AAS abuse is secondary to the accentuation in hypertensive response (most probably due to the increase in rate pressure product) in other words this hypertrophy may be due to higher blood pressure and not due to AAS abuse per se.

The major limitation of this study and other studies like this is that it is hardly possible to document either misuse or non usage of these drugs by any lab tests. Another limitation of this study is that the prescription of these drugs to athletes is unethical and illegal so they prepare anabolic androgenic steroids from different sources and it is possible that these drugs have different doses and different potencies.

In this present study the only way to exclude the effects of weight and body size on echocardiographic findings is to compare groups according to height and weight. However it should be kept in mind that body composition in vigorous athletes is completely different from non athletes.

## CONCLUSION

AAS abuse has no effect on systolic or diastolic function and LV size of the heart, and just may lead to accentuation of physiologic hypertrophic response to weight training sports.

## ACKNOWLEDGMENTS

A special thank you to Dr. K. Fatahi and Mr. H. Moradi for providing cases and completing the initial examinations. This study had been granted and supported by the research committee of Kermanshah University of Medical Sciences and Health Services.

**Conflict of interests:** None

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