EFFECTS OF ANABOLIC ANDROGENIC STEROID USE ON CARDIOVASCULAR STATUS AND REPRODUCTION IN ADULT MALE ATHLETES

By

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ABSTRACT

Anabolic androgenic steroid (AAS) abuse by man represents a significant health risk due to the potential long-term negative physical sequelae. The present work aims to detect the effects of chronic (AAS) use in adult males and their consequences on cardiovascular and gonadal status. This study was divided into two parts: The human part was carried on eighteen strength athletes self administered several steroids (oral and intramuscular) simultaneously for about 14 weeks. Serum lipids and lipoproteins were assessed during use and 6 weeks after drug cessation, as indicators of the risk for cardiovascular disease. Plasma sex hormones as testosterone (T) , luteinizing hormone (LH) and follicle stimulating hormone (FSH) were also determined to clarify the effect of AAS on the testicular endocrine function. The experimental part was carried on 40 rats. They were divided into two groups: Control and an AAS-treated group. Each rat in AAS group administered 5 mg/kg body wt testosterone propionate (TP) intramuscularly twice weekly for 8 weeks. At the end, plasma sex hormones, intra-testicular T levels were assessed and testes of the sacrificed animals were examined histologically as well. The results of the human part showed significant increase in LDL level and significant fall in HDL. These changes were paralleled by significant increase in Apo lipoproteins-B concentration and decrease in both Apo-A1& Lp(a) concentration. On the other hand, sex hormones were significantly suppressed. Six weeks after AAS withdrawal, lipoprotein variables had not returned to baseline concentrations. As regard the experimental part; testes weight and testicular sperm numbers were markedly decreased compared with the control. Testicular tissue examination revealed arrest of advanced spermatogenesis in the seminiferous tubules and a severe depletion of the number of Leydig cells. Plasma levels of FSH and LH were decreased. Plasma T levels remained at the normal range throughout the entire treatment duration. Intra-testicular T levels were significantly reduced. It was concluded that administration of AAS produces comparable profound unfavorable effects on lipids

and lipoproteins, leading to an increased atherogenic lipid profile and to great extent may be responsible for developing male subfertility during use.

INTRODUCTION

The use of drugs to enhance physical performance and appearance has been observed for thousands of years. Today individuals including adolescents, continue to employ a wide variety of anabolic androgenic steroid (AAS) drugs for improving their athletic performance and looking better. Most studies reported that 3-12% of adolescent males used an AAS at some time during their life (Yesalis and Bahrke, 2000).

Anabolic androgenic steroids are synthetic derivatives of testosterone. The legal and illegal use of these drugs is gaining popularity. Testosterone has potent anabolic effects on the musculoskeletal system. They were added to the list of Schedule III Controlled Substances in 1990. They have a powerful lure, despite the risk of subjective side effects (Evans, 2004).

Anabolic androgenic steroid use cause interrupted growth and virilization in children, birth defects in the unborn and a number of other disorders including unpredictable changes in mood, aggression, and libido. The current regimens used for steroid doping include combinations of injectable and oral preparations of steroids at doses 10 to 40 times greater than those prescribed therapeutically (Lamb, 2004).

The present study aims firstly to investigate the adverse effects of AASs on lipid variables in healthy, young strength athletes and recovery of these variables after drug cessation, as risk indicators for cardiovascular disease. Secondly to clarify the effect of anabolic steroids on the testicular endocrine function through measuring the sex hormones i.e. testosterone (T), luteinizing hormone (LH) and follicle stimulating hormone (FSH) both in human and animals to explain the sub-fertility attributed to its use.

SUBJECTS & METHODS

Human part :

Subjects :

Twenty nine male strength athletes classified as eighteen self administered AASs courses on the basis of their own experience and beliefs *AAS group* and eleven controls had never used AASs before *Control group*.

They volunteered by flyers in three regional gym clubs and agreed to participate in this study after they had been discussed about the experimental protocol. Inclusion criteria were: male; athletic training expe-

rience of at least three years, aged 20-40 years. The exclusion criteria were : hypertension, hereditary hypercholesterolaemia, diabetes mellitus, infertility and smoking.

Participants completed an extensive questionnaire on health status, training habits, past history and the previous use of AASs, then had a full physical examination to exclude any relevant diseases. All subjects gave their written informed consent before participating.

All members in AAS group administered several steroids (oral and i.m injection) simultaneously for periods ranged from 10-18 weeks during the study. The total amount administered by each participant exceeded the recommended therapeutic dose. They also had previous experience of AAS self administration for about 2-11 years. Table (1) shows the used AAS drugs, the routes and the average dose. They purchased the AASs from market. The researchers were not involved in purchasing or administering these compounds.

From information supplied by the subjects, the AAS users had been drug free for 6-9 months before the start of the study. However, to objectively exclude recent drug use, urine was collected from all subjects for drug analysis at the beginning of the experiment.

At the beginning of the experiment "base line" and after 14 weeks of AAS use, and six weeks after cessation, blood was drawn from the antecubital vein after 12 hour fasting, for the measurement of serum concentrations of total cholesterol, triglycerides, HDL-cholesterol (HDL-C), LDL- cholesterol (LDL-C), apolipoprotein A1 (Apo-A1), apolipoprotein B (Apo-B), and lipoprotein (a) (Lp(a)).

Methods:

Lipid measurements:

Plasma Total Cholesterol (CHOD-POD; Spinreact, S.A. , Santa Coloma , Spain) and triglyceride (GPO-POD; Spinreact, S.A. , Santa Coloma, Spain) were determined by commercial enzymatic colometric methods on Spekol 11.

HDL-C (Spinreact, S.A., Santa Coloma Spain) was measured by enzymatic colometric methods for total cholesterol after precipitation of the very low density (VLDL) and low density(LDL) lipoproteins from plasma by using phosphotungstate in the presence of magnesium ions and using the supernatant for determination of HDL-C (Grove,1979). LDL cholesterol calculation was based on the Friedewald formula according to Schectman et al., (1996) .

Apolipoprotein A1 and apolipoprotein B determination:

Apolipoprotein A1 was measuered ac-

cording to Miller et al., (1980) by electro-immuno- assay where pretreated samples and standards were run on agarose gel with anti- ApoA1 antiserum (Sigma Chemical Company, St. Louis, MO) at 130 V for 6 hours. The Apo-AI particles continue to migrate and react with anti-apoAI antibodies. After washing and drying the gel, the height of the resulting immunoprecipitation rocket is proportional to the ApoAI concentration. The rocket heights (ApoAI concentration) were compared with the calibration curve generated with the apoAI standard serum pool.

Apolipoprotein B was measured by electroimmunoassay. Anti-Apo B antiserum (Sigma Chemical Company, St. Louis, MO) was used. The samples were used untreated and undiluted unless the Apo-B concentrations were greater than the top of the standard (Marcovina et al., 1993).

Lipoprotein (a) measurement:

Lp(a) was analyzed according to Bostom, (1994) by using a commercially available enzyme-linked immunosorbent assay (Strategic Diagnostics, NewYork, DE).

Methods of hormonal assay:

Serum FSH , LH , and T (both in human and rats) were measured by immuonometric assay according to the protocol of Immulite (Diagnostic Product Corporation, U.S.A.) using Immulite Automated Immunoassy Analyzer (Diagnostic Product Corporation, U.S.A.) which automates the entire assay process. Inter- and intraassay variation coefficients for LH were 9.6% and 5.7%, respectively, for FSH were 7.5% and 6.4%, respectively and for testosterone were 7.8% and 7.0%, respectively.

Experimental part:

Animals:

Forty healthy male rats obtained from the animal house of Faculty of Medicine, El-Minia University. They were 14-22 weeks of age and weighing 200 ± 50 grams. All animals were provided with the ordinary animal food and water and conditioned at room temperature for one week before the experiment. They were divided into two groups:

Control group (n = 20) which were subdivided equally into two subgroups:-

Blank group where rats received nothing and., vehicle treated group where each rat was injected i.m by 0.1ml stafflower oil.

AAS-treated group (n = 20)

Each rat in AAS group was administered 5 mg/kg body wt testosterone propionate (TP) intramuscularly twice weekly for 8 weeks. At the end, Serum FSH, LH, T and intra testicular T were measured. Testes of the sacrificed animals were examined histologically for the resultant changes.

Methods :-

Testes were immersion fixed in Bouin fixative overnight and then stored in 70% ethanol. Testes were dehydrated through a standard series of increased ethanol concentrations and were embedded in paraffin wax, sectioned at 10 µm thickness and stained with hematoxylin and eosin.

Spermatozoa were obtained by making small cuts into the epididymis of fresh unfixed testis and placed into 1 ml of modified Krebs Ringer- bicarbonate solution (pH 7.4). After 10 minutes at 37°C. The epididymis was removed, and the resultant sperm suspension was smeared on slide and counted.

Statistical Analysis

Data are expressed as mean ±(SD). Results were tested for statistical significance using the Post Hoc test after one-way ANOVA to compare changes between groups. P value <0.05 was considered significant.

RESULTS

Human part:-

At baseline study, both control and AAS groups were comparable with respect to age, height, weight, training experience and weekly training hours. During the study all participants maintained their regular training regimens without any changes.

Serum lipids and lipoproteins:

Anabolic androgenic steroid use caused insignificant changes neither in serum triglycerides nor in total cholesterol.A significant increase in LDL-C (from 99.73 ± 6.15 to 128.45 ± 2.14 mg /dl) and a significant fall in HDL-C (from 41.91 ± 1.78 to 23.36 ± 2.91) mg/dl) were found when compared with baseline levels.

These changes were also associated with significant increase in apolipoprotein-B concentrations (from 123 ± 9.61 to 144.73 ± 9.42 mg/dl) and significant decrease in apolipoprotein-A1 concentrations from (120.55 ± 9.94 to 95.55 ± 10.14 mg/dl). Also significant reduction in lipoprotein (a) concentrations from (25.55 ± 5.66 to 9.18 ± 2.86 mg/dl) were observed.

Six weeks after AAS regimen withdrawal, the lipoprotein variables had not returned to baseline concentrations particularly lipoprotein (a) concentrations which remained decreased. Tables (2, 3) and figure (1) illustrate the results.

Reproductive hormones:

At baseline, no significant differences in serum reproductive hormones between the control and AAS groups.

Mean serum testosterone level was increased in AAS group when compared with baseline level, but remained within the normal range.

Both serum FSH and LH levels were significantly suppressed in AAS group (from 4 ± 1.3 and 3.59 ± 1.59 to 0.86 ± 0.79 and 1.45 ± 0.49 IU/L) respectively when compared with the baseline and control levels at the end of the 14th week of treatment period.

Changes in serum reproductive hormones were reversible. They returned to baseline levels 6 weeks of recovery period. Results were shown in tables (4,5) and figure (2).

All urine samples analyzed at baseline were free of any AAS metabolites.

Experimental Part :

No significant differences between the blank and the vehicle treated group administered stafflower I.M as regard testicular weight, testicular sperm numbers, plasma FSH, LH, T and intra-testicular T were observed.

Testicular weight and testicular sperm numbers:

A significant decrease in testicular weight was observed in AAS group 8 weeks after testosterone propionate (TP) administration, the mean testis weight was reduced from 2.29 ± 0.43 gm to 1.03 ±0.33 gm when compared with the control group. Also there is a significant decrease in mean testicular sperm content from 101.6 ± 31.02 to 43.63 ± 25.46 million / testis of the control values (Tables 6, 7 & Fig. 3).

Reproductive hormones:

Eight weeks after TP injection, plasma levels of FSH and LH significantly decreased from (4.25 ± 1.6 and 0.88 ± 0.46 to 2.4 ± 1.03 and 0.15 ± 0.22ng/ml) respectively.Plasma testosterone levels remained at the normal range, where intra-testicular testosterone levels were significantly reduced from 491.5 ± 21.08 to 14.2 ± 10.23 ng\ testis) of control levels (Tables 6, 7 & Fig. 4).

Histological examination of the testis of the adult rats in AAS group showed arrest of advanced spermatogenesis in the seminiferous tubules and severe depletion of the number of leydig cells in the interstitial compartment (Figs. 5,6,7).

DISCUSSION

Nowadays, lots of individuals especially adolescents, are using -in a markedly increasing manner- a wide variety of anabolic androgenic steroids (AAS), running behind the shape of the perfect man.

As regard the human part:

AAS use in this work caused significant increase in LDL-C and significant fall in HDL-C which is compatible with Wu, (1997) who explained that AASs stimulate

hepatic triglyceride lipase, (an enzyme that regulates serum lipids) resulting in decreased serum HDL-C (the good cholesterol) and its sub-fractions which may have long-term consequences in increasing the risk to ischemic heart disease .

Similarly, Parssinen and Seppala (2002) recognized association of AAS use with hypertension and dislipidaemia (raised low density lipoprotein and reduced high density lipoprotein cholesterol, and raised triglycerides).

AAS administration in this study also led to significant decrease in both serum concentrations of Apo-A1(the major component of the HDL particle) and serum atherogenic Lp (a) whereas Apo-B increased significantly. Total cholesterol and triglycerides did not change significantly. Altered lipoprotein profile have also been reported by Kutscher et al., (2002).

Lippi et al., (1997) stated that the fat composition of Lp (a) is comparable to that of LDL-C, but the most important difference is the presence of a specific apoprotein(a). A close correlation has been reported between the serum concentration of Lp(a) and accumulation of this particle in the vascular wall.

In contrast with the detrimental effects of AAS on lipids, AASs may favorably lower Lp(a) concentrations. The exact mechanism by which AASs decrease Lp(a) concentration was suggested by Zmuda et al., (1996) to be mediated through decreasing apo-A synthesis.

Six weeks after AAS withdrawal, lipoprotein variables had not returned to baseline concentrations. Recovery was significantly slower in particular Lp (a) concentrations which remained decreased. This is highly consistent with Hartgens et al., (2004).

Moreover, AASs appear to drive exaggerated left ventricular hypertrophic (LVH) growth response to any hypertrophic stimulus as exercise. Left ventricular wall thickness and cavity dimensions were assessed by Urhausen et al., (2004) in 17 AAS users and 15 non users using echocardiography. They found evident LVH and absolute LV muscle mass measures were significantly greater for users than non users.

Additionally, the hypothesis that AAS may induce coronary vasospasm and powerful influences on coagulation and platelet aggregation in susceptible individuals was reported by Payne et al., (2004) who recorded several cases of increased thrombo-embolic risk as well.

This work showed that both serum FSH

and LH levels were significantly suppressed in AAS group while serum testosterone level was increased but remained within the normal range. The same effect was observed by Torres-Calleja et al., (2001). This effect was explained by MacIndoe et al., (1997) via the negative feedback loop of the hypothalamic pituitary gonadal axis.

Androgenic anabolic steroids use induce suppression of sex hormones which result in hypogonadotrophic hypogonadism with subsequent azoospermia in many couples investigated by Lioyd et al., (1996).

This work also exhibited that sex hormones changes returned to baseline levels 6 weeks of recovery period, this agree with Hartgens et al., (2004).

Regarding the experimental part:

Administration of TP to adult male rats in this work resulted in significant decreases in plasma LH, FSH and intra-testicular T. This occurred without significant changes in plasma T levels. This agree with Nagata et al., (1999) and Bhasin et al., (2001) who found these parameters decreased rapidly to very low levels by AAS use. But does not agree with Clark et al., (1997) as they proved that serum testosterone levels were also suppressed as they used high doses of AAS compounds for longer periods than those used in this work (12 weeks).

Farrell & McGinnis, (2004) stated that ejaculation was affected by AAS administration and only recovered to control levels at 15 weeks of withdrawal.

This work also showed that both testis weight and testicular sperm counts were markedly decreased by TP treatment. A nearby results were found by Lue et al., (2000) in both heat and testosterone treated animal groups compared with the control one.

Similarly, Noorafshan et al., (2004) found the mean testis weight and length of the seminiferous tubules reduced approximately by 32% and 31% respectively in comparison with the control group in animals received high doses of nandrolone decanoate.

McGinnis et al., (2002) found all reproductive hormones and tissue weights return to normal after very long period of AAS withdrawal (eighteen weeks), suggesting possible long lasting effects of chronic AAS exposure.

Examination of the testicular tissue indicated arrest of advanced spermatogenesis in the seminiferous tubules and a severe depletion of the number of

Leydig cells in the interstitial compartment as a result of treatment. This is greatly compatible with Feinberg et al., (1997) who stated that high doses of AAS depleted Leydig cell number in the prepubertal and adult rats as well, The depletion occurred in the prepubertal animals after 16 weeks withdrawal was reversible, while the depletion of the adult animals did not return to the control level suggesting a long lasting alteration.

Nagata et al., (1999) treated mature stallions with 800 mg nandrolone decanoate every 3 weeks for 3 months and found same results.

CONCLUSION AND RECOMMENDATION

It was concluded that administration of AAS produces comparable profound unfavorable effects on lipids and lipoproteins, leading to an increased atherogenic lipid profile and to great extent may be responsible for developing male subfertility during use. So men abusing steroids must be warned about their potential long term adverse effects .

Acknowledgment

Many thanks to Prof. Dr. Wafaa Farghaly, professor of pathology for her help in the histopathologic part.

Table (1): The used AAS drugs, the routes and the average used dose by adult male athletes.

Scientific name	*Generic name*	*Route of administration*	*Total amount of drug used*
Nandrolone decanoate	Deca-Durabolin	I.M	350 mg
Testosterone propionate	Primoteston dopot	I.M	275 mg
Testosterone enanthate	Primoteston depot	I.M	800 mg
Testosterone undecanoate	Andriol	Oral capsule	950 mg
Mesterolone	Proviron	Oral tablets	1200 mg

Table (2): Effects of AAS use and withdrawal on serum lipids and lipoproteins in adult male athletes.

Parameters / Groups		Cholesterol mg/dl	Triglycerides mg/dl	H.D.Lmg/dl	L.D.L. mg/dl	ApolipoA1 mg/dl	Apolipo B mg/dl	Lipo (a) mg/dl
G 1	X ± SD	190.90± 19.05	82.64 ± 27.74	44.36 ± 6.77	105.3 ± 8.69	125.18 ± 9.28	105.91 ± 29.80	23.27 ± 7.59
G 2	X ± SD	199.45± 30.56	95.55 ± 16.91	41.91 ± 5.91	99.73 ± 6.15	120.55 ± 9.94	123.00 ± 9.61	25.55 ± 5.66
G 3	X ± SD	203.36± 22.92	87.55 ± 19.62	23.36 ± 2.91	128.5 ± 7.09	95.55 ± 10.14	144.73 ± 9.42	9.18 ± 2.86
G 4	X ± SD	189.18± 11.21	100.55 ± 12.91	30.82 ± 4.07	118.4 ± 12.14	106.36 ± 6.50	129.36 ± 6.70	15.09 ± 2.98
F		1.041	1.755	39.973	23.747	24.400	10.194	23.449
P		0.385	0.171	0.000*	0.000*	0.000*	0.000*	0.000*

Table (3): Comparison of AAS use and withdrawal on serum lipids and lipoproteins between different groups in adult male athletes.

Para. / Groups	Choleste-rol mg/dl		Triglyceri-des mg/dl		H.D.L-C mg/dl		L.D.L-C mg/dl		Apo. A1 mg/dl		Apo. B mg/dl		Lp. (a) mg/dl	
	M D	P	M D	P	M D	P	M D	P	M D	P	M D	P	M D	P
1 vs 2	8.55	1.00	12.91	0.52	2.45	1.00	5.55	0.88	4.64	0.70	17.09	0.14	4.64	0.70
1 vs 3	12.45	1.00	4.91	0.95	21.00	0.00 *	23.18	0.00 *	29.64	0.00 *	38.82	0.00 *	29.64	0.00 *
1 vs 4	1.73	1.00	17.91	0.24	13.55	0.00 *	13.09	0.07 *	18.82	0.00 *	23.45	0.02 *	18.82	0.00 *
2 vs 3	3.91	1.00	8.00	0.83	18.55	0.00 *	28.73	0.00 *	25.00	0.00 *	21.73	0.03 *	25.00	0.00 *
2 vs 4	10.27	1.00	5.00	0.95	11.09	0.00 *	18.64	0.00 *	14.18	0.08	6.36	0.85	14.18	0.08
3 vs 4	14.18	1.00	13.00	0.95	7.45	0.09	10.09	0.06	10.82	0.06	15.36	0.22	10.82	0.06

Group 1(G1) : Control (n = 11), adult males athletes never used AASs before.
Group 2 (G2): Baseline (n = 18).
Group 3 (G3): Adult male athletes self administered AASs for 12-16 weeks.
Group 4 (G4): 6 weeks after drug cessation.
Para= parameters. vs= versus M D=mean difference.
Values are expressed as mean X ± SD (standerd deviation)
H.D.L-c: high density lipoprotein cholesterol.
L.D.L-c: low density lipoprotein. Cholesterol.
Apo.A1 : Apolipoprotein A1 Apo.B : Apolipoprotein B
LP. (a) : Lipoprotein (a). P *< 0.05 is significant.

Table (4): Changes in serum reproductive hormones by AAS use and withdrawal between different groups in adult male athletes.

Parameters / Groups		Serum Testosterone (nmol/l)	Serum L. H (IU/L)	Serum F.S.H (IU/L)
Group 1	X ± SD	18.55 ± 5.68	4.32 ± 1.42	3.73 ± 1.79
Group 2	X ± SD	16.55 ± 5.07	4.00 ± 1.30	3.59 ± 1.59
Group 3	X ± SD	24.27 ± 7.44	0.86 ± 0.79	1.45 ± 0.49
Group 4	X ± SD	19.64 ± 7.59	3.79 ± 1.37	3.82 ± 1.38
F		2.760	18.076	7.183
P		0.06	0.000*	0.001*

Table (5) : Comparison of AAS use and withdrawal on serum reproductive hormones between different groups in adult male athletes.

Parameters / Groups	Serum testosterone (nmol/l)		Serum L.H. (IU/L)		Serum F.S.H. (IU/L)	
	M D	P	M D	P	M D	P
1 versus 2	2.oo	0.92	0.32	0.99	0.14	0.99
1 versus 3	5.73	0.26	3.45	0.00*	2.28	0.006*
1 versus 4	1.09	0.99	0.53	0.79	9.09	0.99
2 versus 3	7.73	0.06	3.14	0.00*	2.15	0.01*
2 versus 4	3.09	0.77	0.21	0.98	0.23	0.99
3 versus 4	4.64	0.44	2.93	0.00*	2.37	0.004*

Group 1(G1) : Control (n = 11), adult males athletes never used AASs before.
Group 2 (G2): Baseline (n = 18).
Group 3 (G3): Adult male athletes self administered AASs for 12-16 weeks.
Group 4 (G4): 6 weeks after drug cessation.
Values are expressed as mean X ± SD (standerd deviation)
M D = mean difference.
P* < 0.05 is significant
L.H. : Luteinizing hormone.
F.S.H. : Follicle stimulating hormone

Table (6): Changes in testicular weight, testicular sperm count and reproductive hormones in adult rats administered Testosterone propionate 5 mg/ kg i.m twice weekly for 8 weeks.

Parameters / Groups		Testicular weight (g)	Testicular sperm count (million/testis)	Testicular testosterone (ng/testis)	Plasma testosterone (ng/ml)	Serum L. H. (ng/ml)	Serum F.S.H. (ng/ml)
Group 1	X ± SD	2.29 ± 0.43	111.60 ± 10.22	491.50± 21.08	2.85 ± 0.47	0.88 ± 0.46	4.25 ± 1.60
Group 2	X ± SD	2.36 ± 0.46	112.60 ± 9.71	496.50± 29.91	2.95 ± 0.44	0.90 ± 0.30	4.85 ± 1.87
Group 3	X ± SD	1.03 ± 0.33	36.60 ± 10.72	14.20± 10.23	2.70 ± 0.39	0.15 ± 0.22	2.40 ± 1.83
F		32.92	181.741	1594.295	0.026	16.32	6.855
P		0.000*	0.000*	0.000*	0.974	0.000*	0.004*

Table (7) : Comparison between changes in testicular weight, testicular sperm count and reproductive hormones between the different experimental groups.

Parameters / Groups	Testicular weight (g)		Testicular sperm count (million/testis)		Testicular testosterone (ng/testis)		Plasma testosterone (ng/ml)		Serum L.H. (ng/ml)		Serum F.S.H. (ng/ml)	
	M D	P	M D	P	M D	P	M D	P	M D	P	M D	P
1 versus 2	7.000	1.00	1.00	0.98	5.00	0.88	6.00	1.00	2.000	1.00	0.600	1.00
1 versus 3	1.26	0.00	75.00	0.000	477.3	0.00	7.09	1.00	0.73	0.00	1.85	.037
2 versus 3	1.33	0.00	76.00	0.000	482.3	0.00	0.32	0.98	0.75	0.00	2.45	0.004

Group 1 : Blank group (n = 11), rats received nothing.
Group 2 : Vehicle group (n = 10), rats injected IM by 0.1 stafflower oil.
Group 3 : AASs-treated group (n = 20), each rat was injected IM by 5 mg/kg testosterone propionate twice weekly for 8 weeks.
Values are expressed as mean X ± SD (standerd deviation)
M D=mean difference.
L.H. : Lutenizing hormone
F.S.H. : Follicle stimulating hormone. P *< 0.05 is significant.

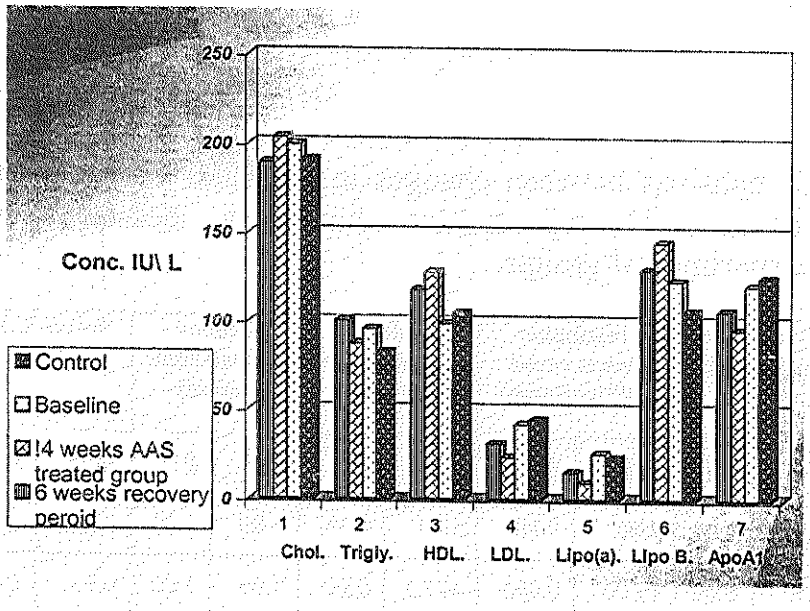

Fig. (1) : Effects of AAS on lipid profiles in male athletes.

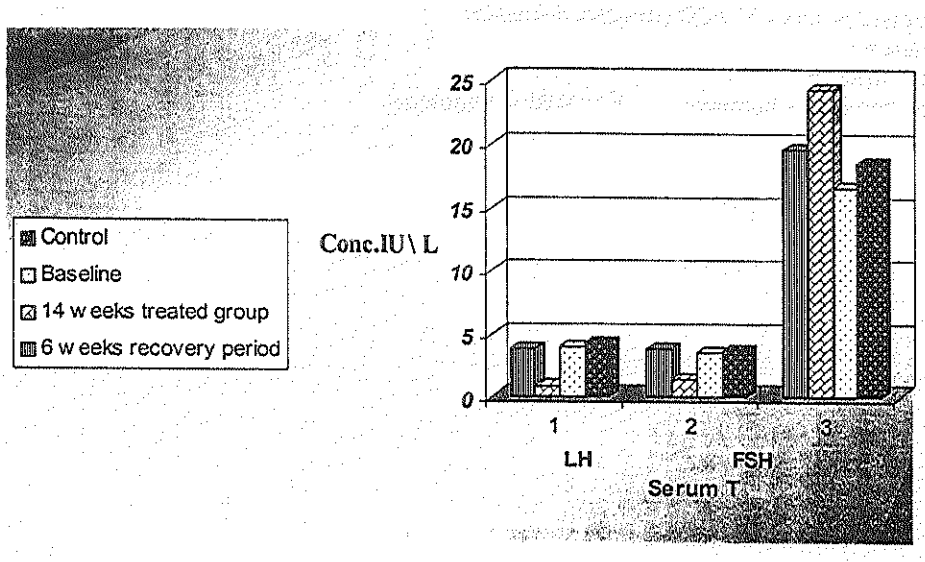

Fig. (2) : Effects of AAS on sex hormones in male athletes.

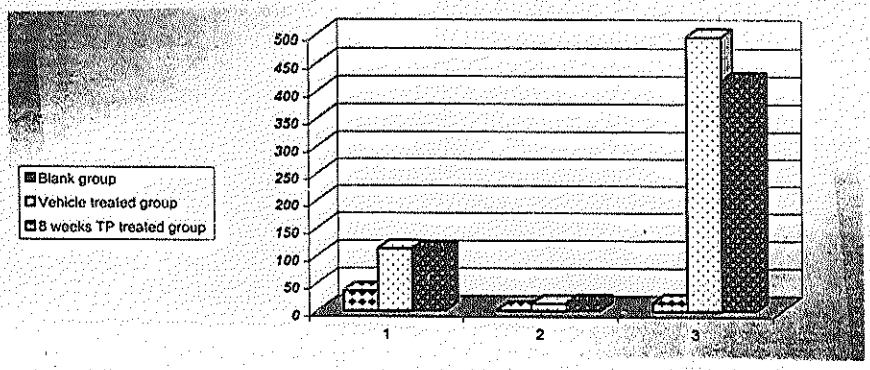

Fig. (3) : Effect of TP injection on sperm count, testicular weight and intratesticular testosterone in rats.

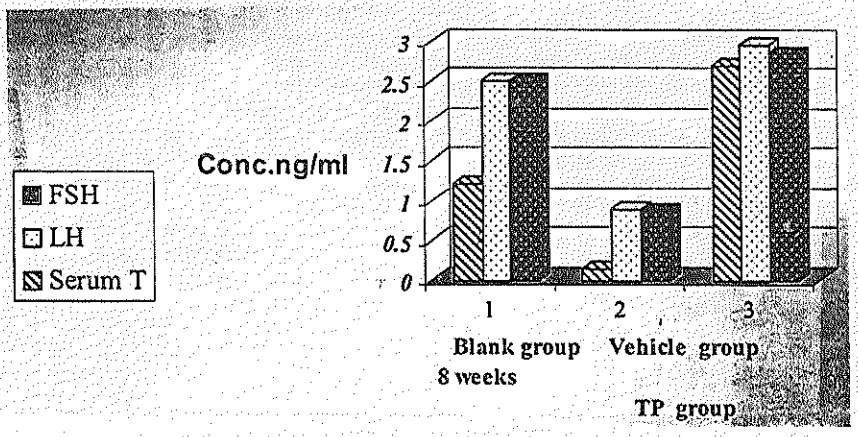

Fig. (4) : Effects of TP injection on rats reproductive hormones.

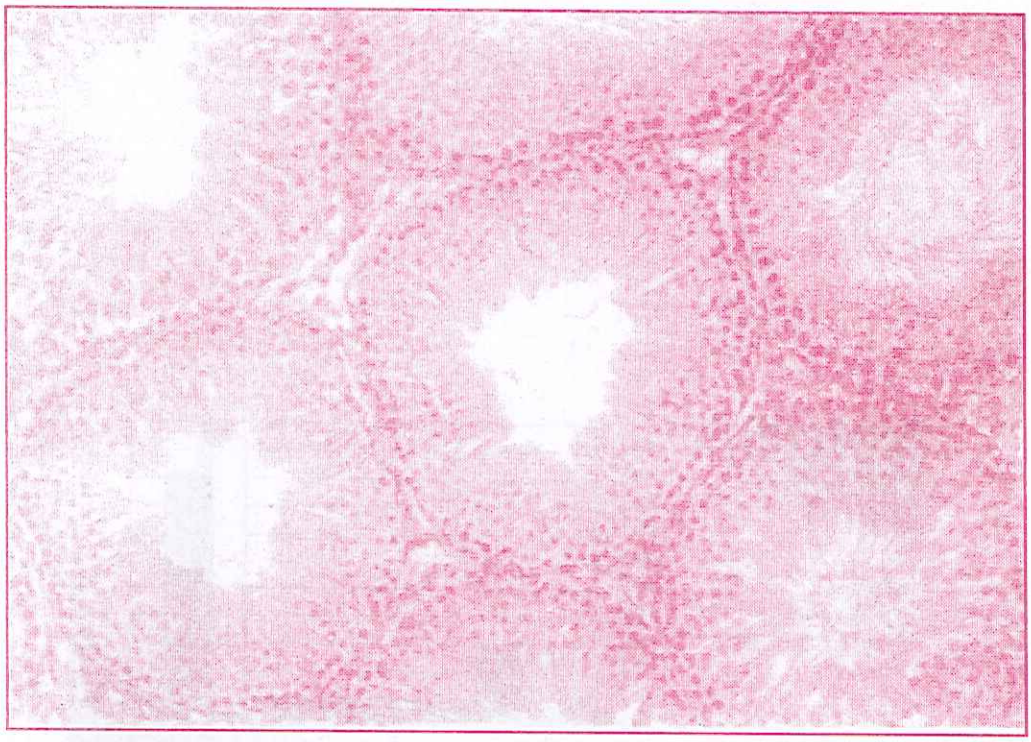

Fig. (5) : A photomicrograph of testis from the control group of rats showing the normal histological structure. *(H & E; X 250).*

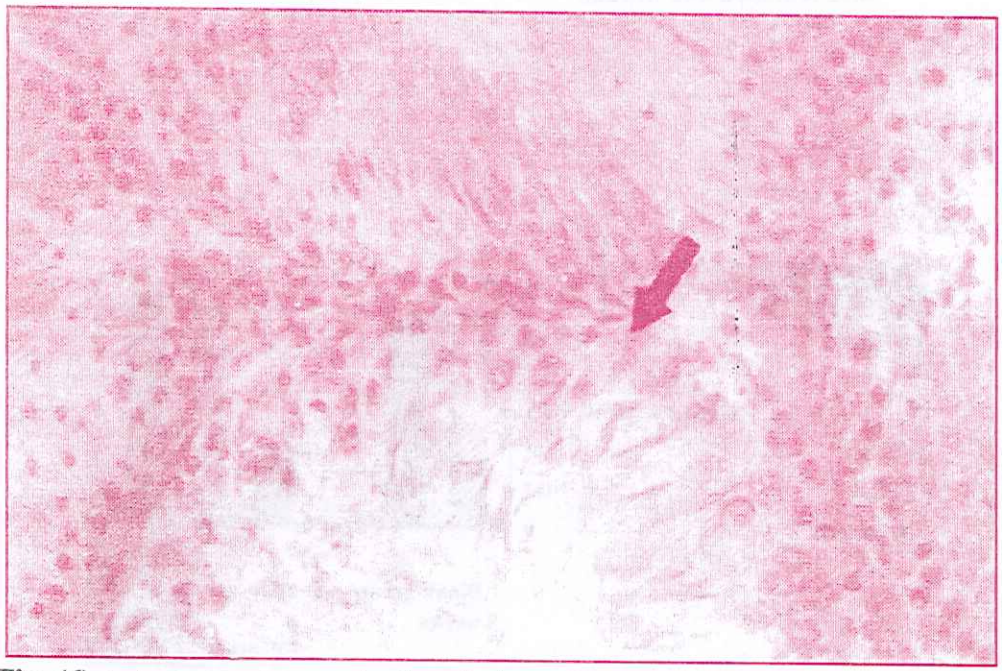

Fig. (6) : A photomicrograph of testis from TP treated group showed severe depletion of the number of leydig cells in the interstitial compartment and sloughing of the germinal epithelium. *(H & E; X 400).*

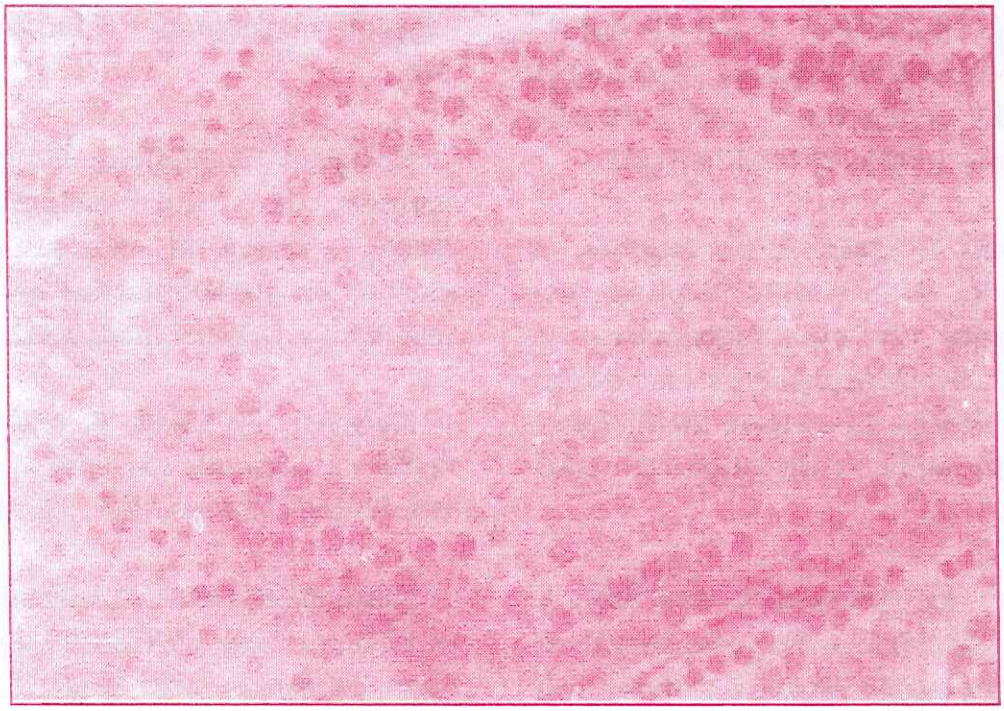

Fig. (7) : A photomicrograph of testis from TP treated group shows arrest of advanced spermatogenesis in the seminiferous tubules. *(H & E; X 400).*

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تأثير أدوية الستيرويدات البنائية على القلب والأوعية الدموية والخصوبة فى الرياضيين الذكور البالغين

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إنتشر إستخدام الستيرويدات البنائية (المنشطات) إنتشاراً وبائياً فى أوساط الرياضيين على إختلاف أنواعهم مما بات خطراً قد يهدد صحتهم على المدى الطويل. ويهدف هذا البحث إلى دراسة التأثير المزمن لأدوية الستيرويدات البنائية على القلب والأوعية الدموية والخصوبة لهؤلاء المتعاطين .

وقسمت هذه الدراسة إلى جزئين : الجزء الأول تم دراسته على ١٨ رياضى يتعاطون عدة أنواع من أدوية الستيرويدات البنائية معاً عن طريق الفم والحقن العضلى لمدة ١٤ إسبوع متتالية، تم قياس نسبة الدهون والبروتينات الدهنية فى دمائهم عند إنتهاء المده وبعد توقف تعاطى هذه الأدوية بستة أسابيع لبيان مدى تأثير هذه الأدوية على الأوعية الدموية والقلب للمتعاطين، أيضاً تم قياس نسبة هرمون التستوستيرون والليوتينيزنج هرمون والهرمون المحفز للحويصلات فى الدم كإشارة إلى تأثير هذه الأدوية على خصوبة هؤلاء الرجال،

أما الجزء الثانى فتم إجرائه على ٤٠ فأراً بالغاً تم تقسيمها إلى مجموعتين مجموعة أولى ضابطة ومجموعة ثانية تم حقنها فى العضل ب ٥ مجم/كجم بهرمون التستوستيرون بروبيونات مرتين إسبوعياً لمدة ٨ أسابيع متتالية، وفى نهاية الدراسة تم وزن الخصية وتحديد عدد الحيوانات المنوية بها وقياس مستوى الهرمونات التناسلية بالدم وبالخصية كما تم فحص نسيج الخصية ميكروسكوبيا لدراسة التغيرات الباثولوجية الحادثة بها.

وأظهرت النتائج التالى : أظهر تحليل الدم الخاص بالرياضيين حدوث زيادة ذات دلالة إحصائية فى الدهون منخفضة الكثافة والأبوليبوبروتين صاحبه نقص ذو دلالة إحصائية فى الدهون عالية الكثافة والأبوليبوبروتين أ١ والليبوبروتين أ. ومن ناحية أخرى وجد نقص ذو دلالة إحصائية فى كل من الليوتينيزنج هرمون والهرمون المحفز للحويصلات فى الدم، وبعد ستة أسابيع من توقف تعاطى هذه الأدوية لم تعد نسبة الدهون والبروتينات الدهنية إلى معدلاتها الطبيعية قبل التعاطى، وأعطت نتائج الجزء التجريبى نقصاً ذو دلالة إحصائية فى كل من وزن الخصية وعدد الحيوانات المنوية وهرمون التستوستيرون بالخصية والليوتينيزنج هرمون والهرمون المحفز للحويصلات بالدم عند مقارنتهم بالمجموعة الضابطة،

كما أظهر فحص نسيج الخصية وجود احباط فى عملية تكوين الحيوانات المنوية فى أنابيب الخصية ونقص شديد فى عدد خلايا لايدج.

وخلص هذا البحث إلى أن تعاطى الستيرويدات البنائية (المنشطات) يؤدى إلى إختلال ضار وخطير فى الدهون والبروتينات الدهنية مما قد يؤدى إلى تأثير مرضى سيىء على القلب والأوعية الدموية كما أن هذه الأدوية مسئولة مسئولية مباشرة عن إنخفاض الخصوبة عند الرجال أثناء تعاطى هذه الأدوية.