

## Myocarditis Associated With Anabolic Steroid Abuse Report of Two Cases

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**Abstract:** *It is obvious that the abuse of anabolic steroids to improve physical performance has been widely implicated in several cardiovascular events, especially myocardial infarction;*

*However, according to the literature, there is currently no report describing myocarditis due to the abuse of anabolic steroids to our knowledge, unless animal experiments or Post mortem autopsy which were carried out at young athletes.*

*We report two cases of young bodybuilders who were complaining about chest pain .*

*They did not have traditional cardiovascular risk factors , however they admitted the intermittent abuse of anabolic steroids.*

*In both instances, electrocardiography, blood chemistry ,Transthoracic echocardiography and cardiac magnetic resonance imaging were performed.*

*Our results support the theory of the cause-effect relationship between anabolic steroid abuse and cardiovascular events, especially their direct cardiac toxicity, by demonstrating myocarditis in both patients through cardiac magnetic resonance imaging.*

**Key Words:** *Myocarditis , Anabolic steroid, Cardiac toxicity.*

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### I. Introduction

Nowadays, the obsession of having a muscular body and improve physical performance, has pushed people to use different types of drugs including anabolic-androgenic-steroids. Recent studies suggest that 6,4% of male and about 1,6 % of female use AAS ,at some time during their lives(1), despite their side effects.Anabolic steroids, also known as anabolic androgenic steroids (SAA), are a group of hormones that include the natural male hormone, testosterone, together with a set of numerous closely related chemical derivatives [1]Testosterone was isolated for the first time in 1930, synthetic derivatives of testosterone quickly followed, and both testosterone and other AAS were widely applied for medical and psychiatric purposes by the end of the following decade[2].

It have been used primarily to treat pathological conditions such as: chronic cachectic conditions like cancer and AIDS or reproductive dysfunction[3].

The use of testosterone derivatives for improvements in physical performance appears to be started in 1954 at a weightlifting competition Russians in Vienna [2]. The intake of steroids became a practice among American weightlifters and the these have been introduced in other sports [2].

Their main role is to increase muscle mass and physical strength and are therefore used in sports, especially in bodybuilding.Their long-term use can have serious health consequences including heart.

The incidence of heart attacks and sudden death in people who abuse anabolic androgenic steroids is underestimated ,because a cause-effect relationship has not been proven, Elsewhere, several cases have been described in the literature since 1988 [4-25].

### II. Case Reports

#### Case 1

A young 24-year-old high-level bodybuilder admitted to emergency. 4 hour after the beginning of crushing retrosternal chest pain without special irradiation or other associated signs. the chest pain has occurred suddenly after a fitness session.

The patient did not have traditional cardiovascular risk factors , however he admitted the intermittent abuse of anabolic steroids orally for 4 years (methandienone 50mg / day) associated with a dietary supplement based on protein powder at at the rate of 60mg / day,the last abuse was for 2 months.

At its admission, the patient was anxious and its vital signs were blood pressure of 120/80 mmHg, heart rate of 90 beats/min, respiratory rate of 17/min and temperature of 36C. Arterial oxygen saturation was

99%. His physical examination was remarkable for his muscular appearance his weight was 82 Kg and his length was 175 cm [body mass index (BMI), 26.7kg/m<sup>2</sup>)

Cardiovascular examination was normal, in particular there was no murmur or *added sounds* in auscultation ,nor signs of heart failure

The electrocardiography (ECG) which was recorded in emergency room revealed *inverted T waves* in the *anterior precordial leads*(Fig.1).

His blood chemistry revealed increased levels of cardiac biomarkers (troponin-I : up to 5 ng/ml )

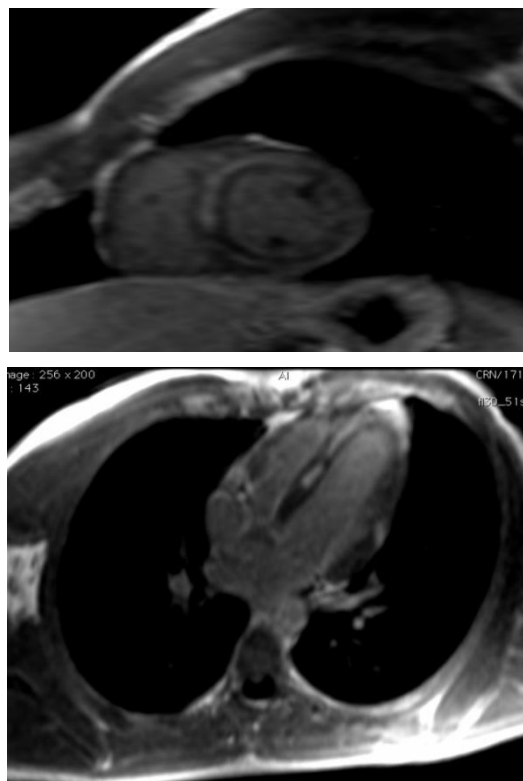
Transthoracic echocardiography did not demonstrate ventricular hypertrophy or myocardial contraction abnormalities *with* preserved ejection fraction (EF=67%).



**Fig.1** inverted T waves in the anterior precordial leads

The patient was monitored in the cardiac intensive care unit, where he was asymptomatic and troponin rate decreased slightly in the light of the young age of the patient the absence of traditional cardiovascular risk factors and the elevation of troponin levels myocarditis has been suspected.

48 hours later, A cardiac magnetic resonance imaging was performed and it showed a persistence of a late intramyocardial nodular enhancement at the anterolateral , inferoseptal anterior and inferior walls , which is a consequence of active myocarditis ;However left ventricular dimensions and function were normal (68%) (Fig.2)



**Fig.2:** persistence of a late intramyocardial nodular enhancement at the anterolateral , inferoseptal anterior and inferior wall

Our patient had no history of influenza-like illness or other infectious signs and there were no extracardiac signs in favor of a systemic disease so the toxic cause was the most evoked.

During his hospitalization there was no anginal recurrence and the troponin decreased; so he was discharged from the hospital on the fifth day with Sports ban instructions.

the evolution was marked by the normalization of the T wave, and troponin nevertheless 6 months later, irm showed the persistence of myocarditis aftereffect with normal left ventricular function .

**case 2**

A young 30-year-old bodybuilder with no traditional cardiovascular risk factors otherwise the patient reported intermittent abuse of several anabolic steroids for 10 years both orally and parenterally:

Oral : methandienone 60mg/day,

Injectable :testosterone 250 mg/2weeks

Decanoate de nandrolone : 50 mg/3weeks

6 months before his admission, the patient presented intermittent chest pain of short duration that did not motivate his consultation, 2 months later he started having asthenia with mild dyspnea associated to slow end irregular palpitations without syncope or lipothymia.

Physical examination found a muscular patient with normal hemodynamic parameters:

Blood pressure of 130/ 70 mmHg, heart rate of 67 beats/min, respiratory rate of 17/min and temperature of 36C.

Arterial oxygen saturation was 99%.

his weight was 111 Kg and his length was 183 cm [body mass index (BMI), 30 kg/m<sup>2</sup>]

Cardiovascular examination was normal, in particular there was no murmur or added sounds in auscultation ,nor signs of heart failure

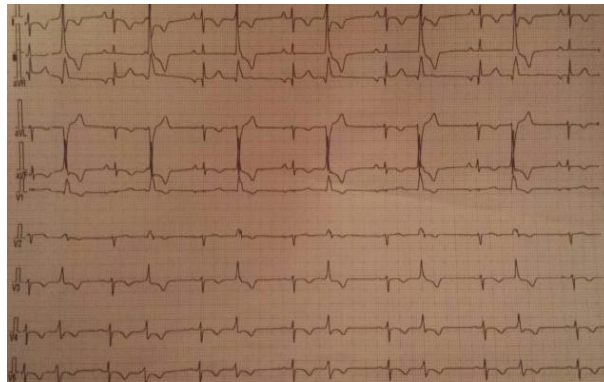
The electrocardiography (ECG) showed ventricular extra systoles with right bundle-branch block appearance(fig.3).

His blood chemistry was normal

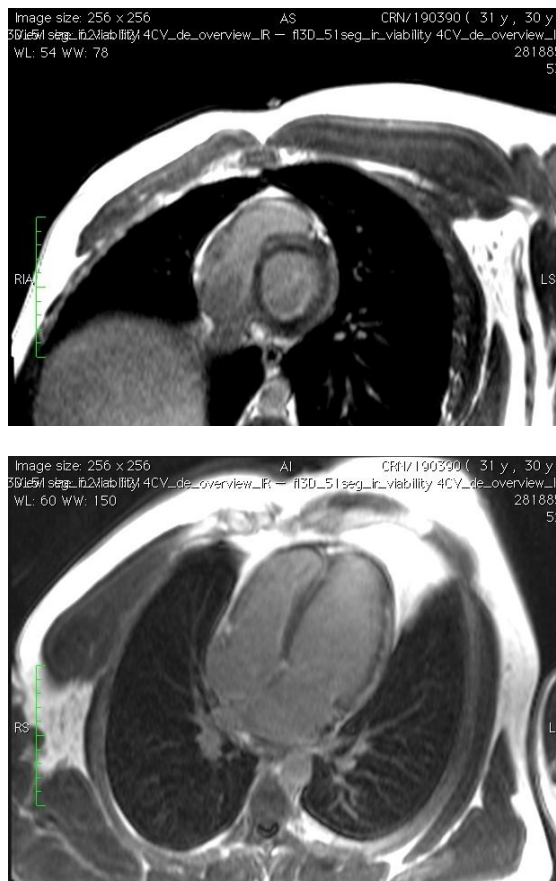
Transthoracique echocardiography objectified a left ventricular dilatation(LVEDd\* =62 mm) without myocardial contraction abnormalities, and ejection fraction was preserved (LFEF=52%)

To investigate this dilatation, A cardiac magnetic resonance imaging was performed and it showed a persistence of focus of fibrosis on the left ventricle free wall and the interventricular septum which is very evocative of myocarditis(Fig.4).

A patient had no history of systemic diseases or infectious diseases ,moreover anabolic steroid abuse may be the cause of myocarditis.



**Fig.3:** ventricular extra systoles with right bundle-branch block appearance



**Fig.4:** A persistence of focus of fibrosis on the left ventricle free wall and the interventricular septum.

The patient has been treated with cordarone, carvedilol, cardioaspirin and ramipril and he was forbidden from sport for 6 months.

### III. Discussion

The most cardiovascular events due to the abuse of anabolic steroids have been dominated by myocardial infarction[25]; where several mechanisms have been incriminated, including: Atherogenic mechanism, Thrombotic mechanism and Vasospasm mechanism.

Atherogenic mechanism: AAS abuse reduce HDL-C and serum lipoprotein(a) levels and increase LDL-C which is providers of atherosclerosis[25,26].

Thrombotic mechanism: anabolic steroids promote thrombosis by several mechanisms which are[27,28]:

the increase in the production of thromboxane A<sub>2</sub>.

the inhibition of prostacyclin (a palatal antiaggregant)

anabolic steroids also cause hyperhomocysteinemia[24,29,30]; which has been implicated for a long time in thromboembolic events Vasospasm mechanism by endothelial dysfunction [31]. moreover Other cardiac abnormalities due to the use of anabolic steroids have been reported .like: arrhythmias [32], ventricular hypertrophy and systolic dysfunction [33], and cardiac autonomic dysfunction[34].

However; according to the literature, No report exists to date describing myocarditis following anabolic androgenic steroid abuse.

all studies describing direct cardiotoxicity were based on animal experiments or on Post mortem autopsy which was carried out at young athletes[35-38].

It has been proven for a long time that the addition of anabolic steroid on myocyte preparation caused their destruction[39,40].

This cytotoxic mechanism would begin with an initial alteration of the energy-producing systems in the mitochondria, this decrease in the energy capacities leading to a decrease in the electrochemical gradients responsible for the activation of the cell lysis systems[39,40].

anabolic steroids act on myocardial cells via specific transcriptional receptors [37,41-43], which explains the direct nature of their cardiotoxicity.

their cardiovascular effect is proportional to the dose and the duration of their administration[37].

These data, derived from animal experiments, serve as a module to explain the changes observed in humans. During the last decade, several autopsy surveys of young athletes anabolic steroid users, have been published [28-30,45,56].

The common characteristics of the deceased autopsies were:

the young age, the absence of traditional cardiovascular risk factors, the sudden death and the anabolic androgenic steroid abuse.

The investigations performed in deceased male anabolic androgenic steroid users subjected to medico-legal autopsies have concluded that the use of anabolic androgenic steroids can cause cardiac hypertrophy which often exceeds the physiological norms of adaptation to physical exercise [45,46]; which is discordant with the case of our patients where no ventricular hypertrophy has been objectified.

The data of our patients are more consistent

with the study of Fineschi et al [35], where necrosis and myocardial fibrosis were found at the autopsy of 2 young athletes anabolic steroid abuser, in the absence of ventricular hypertrophy.

Fenton et al [36], had also objectified myocarditis disseminated at the autopsy of 2 young athletes using anabolic steroids, although these two deceased were medically controlled before their death.

In the same study the autopsies of the rabbits having received anabolic steroids have objectified multiple myocardial and coronary lesions including myolysis, focal necrosis, disorganized myocyte architecture, misshapen cell nuclei, interstitial fibrosis, without ventricular hypertrophy. All the lesions found in rabbits were evocative of toxic myocarditis. Furthermore, the absence of ventricular hypertrophy was probably due to the short duration of exposure to anabolic steroids (2 months) and the recovery period of 1 month.

Another case of myocarditis has been reported in Montisci series, which further enhances the cause-effect relationship between anabolic steroids abuse and myocarditis,

**Table .1:** Summary table of deceased subjects who presented myocarditis following

study	Age (year)	Reason for use	Weight (kg)	weight of heart(g)	Type of drugs	lesions found
Fineschi 2005	29	Bodybuilding	72	380	Testosterone Nandrolone santanazol	numerous foci of contraction band necrosis+ two microfoci of fibrosis
Fineschi 2005	30	bodybuilding	90	400	Norandrosterone nandrolone	focal myocardial fibrosis (wall thicknesses were normal)
Fanton 2009	25	Bodybuilding	—	460	—	Disseminated myocarditis
Fanton 2009	28	Soccer	—	380	—	Disseminated myocarditis
Montisci 2011	25	Bodybuilding	125	390	Testosterone Epitestosterone nortestosterone	Eosinophilic myocarditis

Anabolic androgenic steroids can be used orally or parenterally by intramuscular injection and transdermally. The most popular mode is the intramuscular route. Oral preparations have a short half-life and are taken daily, whereas injectable androgens are typically used weekly or biweekly [38].

Moreover, the parenteral administration of AAS can be more cardiotoxic than oral route. The alkylated anabolic steroids which are very cardiotoxic, are deactivated by first pass metabolism and are administered only parenterally in opposition to 17- $\alpha$ -alkylated anabolic steroids which are administered orally and are more hepatotoxic.

However; the toxicological investigations done in the deceased anabolic steroid abusers; have objectified the consumption of several types of drug either orally or parenterally which is the same in our patients.

To our knowledge, despite the limited number of our patients, our study is the only one that documented myocarditis by imagery in anabolic steroid abusers.

The results of our investigations tend to confirm the animals data and the discovery of some investigations performed in deceased male anabolic androgenic steroid users, however it is impossible to formulate pathophysiologic theories based on the results obtained from only a small number of cases.

In order to obtain safer results a wider study must be started among young athletes.

#### IV. Conclusion

This is evidence that anabolic steroids have several cardiac and extracardiac side effects. Nevertheless, the causal relationship between ASA and myocarditis can only be established by rigorous studies with well-controlled protocols.

Pending more relevant studies, awareness-raising for young athletes becomes mandatory.

#### Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare

#### Références

- [1]. D. Sagoe et al. The global epidemiology of anabolic-androgenic steroid use: a meta-analysis and meta-regression analysis. *Annals of Epidemiology* 24 (2014) 383-398 .
- [2]. Kanayama, G., Hudson, J. I., Harrison, G., & Pope, Jr. (2008). Long-term psychiatric and medical consequences of anabolic-androgenic steroid abuse: A looming public health concern? *Drug and Alcohol Dependence*, 98(1-2), 1- 12. Doi: 10.1016/j.drugalcdep.2008.05.004.
- [3]. M. Lusetti et al. Pathological changes in anabolic androgenic steroid users *Journal of Forensic and Legal Medicine* 33 (2015) 101e104
- [4]. McNutt RA, Ferenchick GS, Kirlin PC, et al. Acute myocardial infarction in a 22-year-old world class weight lifter using anabolic steroids. *Am J Cardiol* 1988; 62: 164.
- [5]. Bowman S. Anabolic steroids and infarction. *BMJ* 1990;300: 750.
- [6]. Lyngberg KK. Myocardial infarction and death of a body builder after using anabolic steroids. *Ugeskr Laeger* 1991; 153: 587–588
- [7]. Ferenchick GS. Are androgenic steroids thrombogenic? *N Engl J Med* 1990; 322: 476
- [8]. Ferenchick GS and Adelman S. Myocardial infarction associated with anabolic steroid use in a previously healthy 37-year-old weight lifter. *Am Heart J* 1992; 124: 507–508.
- [9]. Kennedy C. Myocardial infarction in association with misuse of anabolic steroids. *Ulster Med J* 1993; 62: 174–176.
- [10]. Kennedy MC, Corrigan AB and Pilbeam ST. Myocardial infarction and cerebral haemorrhage in a young body builder taking anabolic steroids. *Aust N Z J Med* 1993; 23: 713
- [11]. Huie MJ. An acute myocardial infarction occurring in an anabolic steroid user. *Med Sci Sports Exerc* 1994; 26: 408–413.
- [12]. Fisher M, Appleby M, Rittoo D, et al. Myocardial infarction with extensive intracoronary thrombus induced by anabolic steroids. *Br J Clin Pract* 1996; 50: 222–223
- [13]. Hourigan LA, Rainbird AJ and Dooris M. Intracoronary stenting for acute myocardial infarction (AMI) in a 24- year-old man using anabolic androgenic steroids. *Aust N Z J Med* 1998; 28: 838–839
- [14]. Godon P, Bonnefoy E, Gue´rard S, et al. Myocardial infarction and anabolic steroid use. A case report. *Arch Mal Coeur Vaiss* 2000; 93: 879–883.
- [15]. Kokkonen L, Anttonen O, Penttilä O, et al. Protein C deficiency and use of anabolic steroids behind the myocardial infarction in a young man. *Duodecim* 2001; 117: 2279–2281.
- [16]. Fineschi V, Baroldi G, Monciotti F, et al. Anabolic steroid abuse and cardiac sudden death: A pathologic study. *Arch Pathol Lab Med* 2001; 125: 253–255.
- [17]. Tischer KH, Heyny-von Haussen R, Mall G, et al. Coronary thrombosis and ectasia of coronary arteries after long-term use of anabolic steroids. *Z Kardiol* 2003; 92: 326–331.
- [18]. Guñes, Y, Erbas, C, Okuyan E, et al. Myocardial infarction with intracoronary thrombus induced by anabolic steroids. *Anadolu Kardiyol Derg* 2004; 4: 357–358.
- [19]. Halvorsen S, Thorsby PM and Haug E. Acute myocardial infarction in a young man who had been using androgenic anabolic steroids. *Tidsskr Nor Laegeforen* 2004; 124: 170–172.
- [20]. Stergiopoulos K, Brennan JJ, Mathews R, et al. Anabolic steroids, acute myocardial infarction and polycythemia: A case report and review of the literature. *Vasc Health Risk Manag* 2008; 4: 1475–1480.
- [21]. Wysoczanski M, Rachko M and Bergmann SR. Acute myocardial infarction in a young man using anabolic steroids. *Angiology* 2008; 59: 376–378.
- [22]. İlhan E, Demirci D, Guñenc, TS, et al. Acute myocardial infarction and renal infarction in a bodybuilder using anabolic steroids. *Türk Kardiyol Dern Ars* 2010; 38: 275–278.
- [23]. Peoples K, Kobe D, Campana C, et al. Hyperhomocysteinemia-induced myocardial infarction in a young male using anabolic steroids. *Am J Emerg Med* 2014; 32: 948.e1-2.
- [24]. Poorzand, et al. Acute myocardial infarction in a young male wrestler: A case report *ARYA Atheroscler* 2015; Volume 11; Issue 6 :366-369
- [25]. Christou et al. Acute myocardial infarction in a young bodybuilder taking anabolic androgenic steroids: A case report and critical review of the literature. *European Journal of Preventive Cardiology* 0(00) 1–12
- [26]. Garcí a-Esperero ´n C, Herva ´s-Garcí a JV, Jime ´nez-Gonza ´lez M, et al. Ingestion of anabolic steroids and ischaemic stroke. A clinical case report and review of the literature. *Rev Neurol* 2013; 56: 327–331
- [27]. Pilo R, Aharony D and Raz A. Testosterone potentiation of ionophore and ADP induced platelet aggregation: Relationship to arachidonic acid metabolism. *Thromb Haemost* 1981; 46: 538–542
- [28]. Nakao J, Change WC, Murota SI, et al. Testosterone inhibits prostacyclin production by rat aortic smooth muscle cells in culture. *Atherosclerosis* 1981; 39: 203–209
- [29]. F. Borson-Chazot, L. Guadagnino, S. Bernard, Ph. Moulin\* Hyperhomocystéinémie et risque vasculaire *Act. Méd. Int. - Métabolismes - Hormones - Nutrition*, Volume III, n° 1, mars 1999 :31-34
- [30]. Graham MR, Grace FM, Boobier W, et al. Homocysteine induced cardiovascular events: a consequence of long term anabolic-androgenic steroid (AAS) abuse. *Br J Sports Med* 2006;40:644–8.
- [31]. Skogastierna C, Hotzen M, Rane A, et al. A supraphysiological dose of testosterone induces nitric oxide production and oxidative stress. *Eur J Prev Cardiol* 2014; 21: 1049–1054.
- [32]. Sculthorpe N, Grace F, Jones P, Davies B. Evidence of altered cardiac electrophysiology following prolonged androgenic anabolic steroid use. *Cardiovasc Toxicol* 2010;10:239–43.
- [33]. T. Luijckx et al. Anabolic androgenic steroid use is associated with ventricular dysfunction on cardiac MRI in strength trained athletes *International Journal of Cardiology* 167 (2013) 664–668
- [34]. Pereira-Junior PP, Chaves EA, Costa ESRH, Masuda MO, de Carvalho AC, Nascimento JH. Cardiac autonomic dysfunction in rats chronically treated with anabolic steroid. *Eur J Appl Physiol* 2006;96:487–94
- [35]. Fineschi V, Riezzo I, Centini F, Silingardi E, Licata M, Beduschi G, et al. Sudden cardiac death during anabolic steroid abuse: morphologic and toxicologic findings in two fatal cases of bodybuilders. *Int J Leg Med* 2007;121:48e53.

- [36]. Fanton L, Belhani D, Vaillant F, Tabib A, Gomez L, Descotes J, et al. Heart lesions associated with anabolic steroid abuse: comparison of post-mortem findings in athletes and norethandrolone-induced lesions in rabbits. *Exp Toxicol Pathol* 2009;61:317e23.
- [37]. M. Montisci et al. Anabolic androgenic steroids abuse and cardiac death in athletes: Morphological and toxicological findings in four fatal cases/ *Forensic Science International* 217 (2012) e13–e18
- [38]. M. Lusetti et al. Pathological changes in anabolic androgenic steroid users . *Journal of Forensic and Legal Medicine* 33 (2015) 101e104
- [39]. MELCHERT R.B. et al effect of anabolic androgenic steroids on primary myocardial cell cultures. *Med. Sci. Sports Exerc.*, 1992, 24 : 206-212.
- [40]. C.-Y. Guezennec .effets des apportsd'hormones exogenes sur la fonction cardiaque à l'effort:problemes cardio-vasculaires posés par le dopage
- [41]. C.M. Kuhn, Anabolic steroids, *Recent Prog. Horm. Res.* 57 (2002) 411–434.
- [42]. J.D. Marsh, M.H. Lehmann, R.H. Ritchie, J.K. Gwathmey, G.E. Green, R.J. Schiebinger, Androgen receptors mediate hypertrophy in cardiac myocytes, *Circulation* 98 (1998) 256–261.
- [43]. P.Y. Liu, A.K. Death, D.J. Handelsman, Androgens and cardiovascular disease, *Endocr. Rev.* 24 (2003) 313–340.
- [44]. H.R.M. Far et al. Cardiac hypertrophy in deceased users of anabolic androgenic steroids: an investigation of autopsy findings. *Cardiovascular Pathology* 21 (2012) 312–316 .
- [45]. I. Thiblin et al. Anabolic steroids and cardiovascular risk: A national population-based cohort study/ *Drug and Alcohol Dependence* 152 (2015) 87–92

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