Acute myocardial infarction in a young bodybuilder taking anabolic androgenic steroids: A case report and critical review of the literature

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Abstract
We describe a case report of a 30-year-old bodybuilder suffering acute myocardial infarction (AMI). He had been taking stanozolol and testosterone for two months. The coronary angiogram showed high thrombotic burden in the left anterior descending artery without underlying atherosclerosis. Few case reports of AMI in athletes taking anabolic androgenic steroids (AASs) have been reported so far. AAS-related AMI is possibly underreported in the medical literature due to the desire of the affected individuals to hide AAS use. Physicians should always consider the possibility of AAS abuse in the context of a young athlete suffering AMI. AASs can predispose to AMI through the acceleration of coronary atherosclerosis. Additionally, thrombosis without underlying atherosclerosis or vasospasm is highly possible to cause AMI in AAS users. Complications after AMI may be more frequent in AAS users.

Keywords
Anabolic steroids, myocardial infarction, athletes, bodybuilding

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Introduction
Anabolic androgenic steroids (AASs) are synthetic testosterone analogs.¹ Abuse of AASs is usually given publicity in cases of elite athletes who have been caught. Among athletes the highest rates of AAS abuse have been reported for bodybuilders, weightlifters, American football players and throwers.² The published reports of AAS abuse by competitive athletes are just the tip of the iceberg. Indeed, elite athletes account for the smallest group of AAS users, with higher levels of use occurring among recreational sportspeople and those who use AASs for aesthetic purposes.³,⁴

Most of the AAS toxicity can be attributed to the high dosage. Although side effects of AASs are rare at therapeutic doses, abusers typically use five to 15 times the recommended clinical doses of AASs and hence have a greater chance to develop complications.⁵ Even more, multiple AASs are often taken simultaneously by alternately increasing and then tapering their doses either in parallel, in series, or both, for a specific training effect.⁶ Such pyramid schedules, called ‘stacking’, are usually taken over cycles lasting up to 48 weeks. At such high doses AAS abuse has been associated with serious side effects including cardiovascular adverse events, hepatotoxicity, hypogonadism and psychiatric disorders.¹ With regard to the cardiovascular side effects of AAS abuse, acute myocardial infarction (AMI) is possibly the most devastating one. However, few case reports of AMI associated with abuse of AASs have been reported so far.

Case report
A 30-year-old male with no prior medical history presented to the emergency department of a peripheral...
hospital in a Greek island with an acute episode of severe substernal pain accompanied by diaphoresis, which started while he was taking a shower in the afternoon. The pain started 30 min before his arrival at the emergency department. The patient was smoking approximately 60 cigarettes per day. He reported non-medical use of AAS for two months prior to his admission. Specifically, he was taking orally stanozolol 10 mg daily and intramuscularly 250 mg testosterone (30 mg testosterone propionate, 60 mg felinpropi testoster- one, 60 mg testosterone izocaproa, 100 mg testosterone decanoate) twice per week. He was a former boxer and had been training systematically for a bodybuilding competition during the last four months. His training regimen consisted of 2 h resistance training and 1 h aerobic training every day.

In the emergency room, the patient’s vital signs were blood pressure of 155/90 mmHg, heart rate of 106 beats/min, respiratory rate of 17/min and temperature of 36°C. Arterial oxygen saturation was 99%. His physical examination was remarkable for his muscular appearance. The initial electrocardiogram revealed sinus tachycardia with ST-segment elevations up to 0.3 mV in leads II, aVF, III and V3–V6, suggesting an evolving AMI (Figure 1). The patient was given intravenous thrombolysis with tenecteplase and was started an oral therapy with aspirin, clopidogrel, pantoprazole, valsartan, carvedilol and rosuvastatin and subcutaneous therapy with fraxiparine. The patient was monitored in the coronary care unit, where he was asymptomatic and the ST-segment elevations in the electrocardiogram decreased by more than 50%.

His blood chemistry revealed increased levels of cardiac troponin-I (up to 7.6 ng/ml), creatine kinase (up to 5971 IU/l), creatine kinase-MB (up to 231 IU/l), alanine transaminase (up to 146 IU/l), aspartate transaminase (up to 86 IU/l) and lactate dehydrogenase (up to 419 IU/l). Fasting lipid profile revealed very low high-density lipoprotein-cholesterol (HDL-C) of 22 mg/dl and increased low-density lipoprotein-cholesterol (LDL-C) of 167 mg/dl.

On echocardiographic examination there was concentric left ventricular (LV) hypertrophy with maximum wall thickness 13 mm and LV end-diastolic diameter 45 mm. Ejection fraction was 45% with hypokinesia in the apical segments of LV myocardium. Mild spontaneous echo contrast was detected within the LV cavity. Pulsed wave Doppler of transmitral inflow revealed a ratio of the peak early diastolic (E) to atrial transmitral velocities of 1.3 and a deceleration time of 207 ms. The ratio of E to early diastolic mitral annular velocity was 11, indicating that LV filling pressure was not increased. Pulmonary hypertension was absent.

The man was referred to our tertiary centre to undergo coronary angiography three days after the AMI. Coronary angiography revealed thrombosis in the mid-portion of the left anterior descending coronary artery (LAD) without significant underlying atherosclerosis (Figure 2). Abrupt occlusion of the distal segment of a ‘wrap-around’ LAD was detected with visible embolized thrombus at the distal part of the LAD, just after ‘wrapping around’ the apex towards the inferior wall (Figure 3). This finding can explain the ST-segment elevation in the inferior leads of the electrocardiogram. A conservative strategy with medical therapy was adopted, since no underlying coronary artery stenosis was detected, the burden of coronary thrombus was not high and there were no signs of acute ischaemia during coronary angiography. A second coronary angiography was recommended to be performed in the following days during his initial hospitalization for the reevaluation of coronary anatomy after the anticipated resolution of coronary thrombus. A detailed examination of other possible causes of thrombosis, such as spontaneous dissection of the coronaries with optical coherence tomography, was also scheduled. The patient had an uneventful hospital course. However, the patient refused to stay in hospital any longer on the sixth day after the AMI and he decided not to undergo any further medical examination. The medical treatment at the time of hospital exit included: aspirin 100 mg/day, ticagrelor 90 mg twice daily, bisoprolol 7.5 mg/day, ramipril 5 mg/day, rosuvastatin 10 mg/day and pantoprazole 40 mg/day.

A detailed laboratory analysis for the exclusion of genetic causes of the AMI in this patient was not performed. Therefore, a genetic predisposition for acute coronary events cannot be excluded in this young individual, though there was a negative family history for cardiovascular events. Moreover, smoking may have contributed to the occurrence of AMI in this athlete. Based on the available data of this case report, AAS abuse could be considered only as a predisposing factor, but not the sole cause of AMI in this young bodybuilder.

Discussion

Methodology

A literature search based on PubMed listings up to 27 February 2016 using as the search terms ‘anabolic steroids AND myocardial infarction’ identified 85 articles. Moreover, we examined the reference list of the articles identified by this search strategy and selected those we judged relevant.

Epidemiology of AAS-related AMI

Twenty-three case reports of AAS-related AMI in apparently healthy individuals have been reported so
far\textsuperscript{7–28} (Table 1). All these case reports were similar in that the patients were typically young male athletes in whom other predisposing factors for AMI were excluded and the single remaining possible cause was AAS abuse. Apart from these cases, there were two additional cases of individuals receiving not only AAS, but also other ergogenic substances potentially contributing to AMI (i.e. erythropoietin, amphetamines).\textsuperscript{29,30} Moreover, there was a case report of a bodybuilder chronically using AAS until four weeks before he experienced an AMI and since then he had been taking oral clenbuterol, which is a potent \(\beta_2\)-agonist with anabolic and thermogenic properties.\textsuperscript{31} Two other cases of AMI in older patients with aplastic anaemia who were treated with AAS were reported.\textsuperscript{32} Furthermore, there was one case of AMI in a HIV-infected patient receiving AAS to prevent muscle wasting.\textsuperscript{33}

With regard to the other AAS-induced cardiovascular outcomes apart from AMI, chronic AAS abuse has been linked with a few case reports of ischaemic stroke, as well as with several well-reported cases of sudden cardiac death unrelated to AMI, possibly attributed to a direct myocardial effect.\textsuperscript{34,35}

Limitations of the studies investigating the effects of high dose AAS on human health include the fact that the relevant data have been reported only as case reports or small studies that lack adequate control groups. Moreover, the type and dose of AAS were

\begin{figure}
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\includegraphics[width=\textwidth]{figure1.png}
\caption{Electrocardiogram showing ST segment elevation of the leads V\textsubscript{3}–V\textsubscript{6}, II, III and aVF.}
\end{figure}
often not mentioned in the relevant studies, because they were unknown. The participants of these studies may have also received other ergogenic aids, which can confound the results and increase the potential for adverse events. Furthermore, the AAS-related cardiac events are expected to be underreported in the medical literature due to the desire of the affected individuals to hide AAS use, for legal reasons in the case of an athlete or to avoid being socially stigmatized. Therefore, the reported frequency of AAS-related AMI possibly underestimates the true incidence.

**Figure 2.** Left coronary angiogram (90° lateral projection) showing floating thrombus (white arrow) in the mid-portion of left anterior descending coronary artery.

**Pathogenesis of AAS-induced AMI**

The possible underlying mechanisms of AAS-induced AMI include: a) atherogenic, b) thrombotic and c) vasospastic (Figure 4).

**Atherogenic mechanism.** AAS abuse can reduce HDL-C and serum lipoprotein(a) levels and increase LDL-C. The changes of HDL-C and LDL-C are usually of great magnitude, especially the decreases in HDL-C and HDL2-C, explaining at least in part the atherogenic
potential of AAS. Nevertheless, the acceleration of atherogenesis by the abnormal AAS-induced lipoprotein profile is more likely to occur when there is long-term AAS abuse in the affected individual. Consistently, among the reported cases of AAS-related AMI in athletes, when underlying atherosclerosis was present the AAS abuse was for several years (Table 1). Considering that athletes performing aerobic exercise training are expected to have increased levels of HDL-C, the considerable downregulation of HDL-C (22 mg/dl) in the bodybuilder of our case report raises the suspicion of the possible use of AAS.36

The majority of studies have shown that chronic AAS abuse can increase both systolic and diastolic blood pressure.5,37 When AAS-induced hypertension was found, it was of small magnitude and plausible underlying mechanisms may be renal retention of sodium by AAS, as well as AAS-induced vasospasm.5,38,39

**Thrombotic mechanism.** The specific cause of coronary thrombosis in the patient of the present case report remains unknown, though AAS abuse may have predisposed him to this event. Thrombogenicity of AAS is suggested by experimental data in which animals pretreated with AAS had greater clot size, and lower vessel-occlusion times in response to thrombotic stimuli.40,41 These effects may be mediated through enhanced platelet aggregation, because AASs have been found to potentiate platelet aggregation through both increased production of thromboxane A2, which is a potent platelet aggregator, and decreased

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**Figure 3.** Left coronary angiogram showing abrupt occlusion of the distal segment of a ‘wrap-around’ left anterior descending coronary artery (LAD) (black arrow). Visible embolized thrombus (white arrow) at the distal part of the LAD, just after ‘wrapping around’ the apex towards the inferior wall.
<table>
<thead>
<tr>
<th>Study</th>
<th>Age (years)</th>
<th>Reason for use</th>
<th>Drugs used</th>
<th>Duration of use</th>
<th>Localization of AMI</th>
<th>Death following AMI</th>
<th>Coronary angiography/autopsy</th>
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<tr>
<td>Peoples et al.7</td>
<td>27</td>
<td>Jujitsu</td>
<td>Unknown</td>
<td>Unknown</td>
<td>Anterior</td>
<td>No</td>
<td>LAD (thrombus)</td>
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<td>Ilhan et al.8</td>
<td>41</td>
<td>Bodybuilding</td>
<td>Oxymetholone, methenolone</td>
<td>15 years</td>
<td>Inferior</td>
<td>No</td>
<td>RCA, LAD</td>
</tr>
<tr>
<td>Wysoczanski et al.9</td>
<td>31</td>
<td>Bodybuilding</td>
<td>Unknown</td>
<td>10 years</td>
<td>Inferior</td>
<td>No</td>
<td>LM, RCA</td>
</tr>
<tr>
<td>Stergiopoulos et al.10</td>
<td>44</td>
<td>Weight-lifting</td>
<td>Testosterone</td>
<td>Two years</td>
<td>Inferior</td>
<td>No</td>
<td>RCA (thrombus), LAD</td>
</tr>
<tr>
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<td>43</td>
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<td>Drostanolone, testosterone, methandrostenedolone</td>
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<td>10 years</td>
<td>Anterior</td>
<td>No</td>
<td>LAD</td>
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<tr>
<td>Büttner et al.13</td>
<td>33</td>
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<tr>
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<td>NA</td>
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<td>No</td>
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<td>26</td>
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<td>Unknown</td>
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<td>Inferior</td>
<td>No</td>
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<td>22</td>
<td>College athlete</td>
<td>Unknown</td>
<td>Unknown</td>
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<td>LM (thrombus), LAD (thrombus)</td>
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<td>Ferenchick and Adelman24</td>
<td>37</td>
<td>Weight lifting</td>
<td>Nandrolone, boldenone, testosterone, stanozolol, oxandrolone</td>
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<td>Inferior</td>
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<td>Normal</td>
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<td>28</td>
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<td>&gt;6 months</td>
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<td>Yes</td>
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<td>Bowman26</td>
<td>23</td>
<td>Bodybuilding</td>
<td>Unknown</td>
<td>Unknown</td>
<td>Anterior</td>
<td>No</td>
<td>LAD, RCA</td>
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<tr>
<td>McNutt et al.27</td>
<td>22</td>
<td>Weight lifting</td>
<td>Unknown</td>
<td>Six weeks</td>
<td>Inferior</td>
<td>No</td>
<td>Normal</td>
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<tr>
<td>Shahsavari Nia et al.28</td>
<td>23</td>
<td>Bodybuilding</td>
<td>Trenbolone</td>
<td>One year</td>
<td>Anterior</td>
<td>No</td>
<td>LAD, LCx</td>
</tr>
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</table>

AMI: acute myocardial infarction; LAD: left anterior descending artery; LCx: left circumflex coronary artery; LM: left main coronary artery; NA: no available information; RCA: right coronary artery
production of prostacyclin, which is an inhibitor of platelet aggregation.\textsuperscript{42–45} Although AASs have been consistently found to induce activation of platelet aggregability, their effects on coagulation cascade and fibrinolytic pathway are less clear.\textsuperscript{46} From this point of view, documentation of high thrombus burden in coronary arteries of AAS users having suffered AMI dictates treatment with intravenous IIb/IIIa inhibitors as a reasonable option.

One case report of AAS-related AMI accompanied by hyperhomocysteinemia has been reported.\textsuperscript{7} Indeed, AAS can increase serum homocysteine levels and by this way may promote atherosclerosis and thrombosis in coronary arteries.\textsuperscript{47}

AASs are known to stimulate erythropoiesis, particularly in high doses, resulting in upregulation of haematocrit, which increases blood viscosity and thus can predispose to thrombosis.\textsuperscript{10} Consistently, a case report of AAS-induced AMI with significant polycythaemia (haemoglobin: 22 g/dl, haematocrit: 63\%) has been reported.\textsuperscript{10}

A diligent search for intracardiac thrombi should be performed especially in AAS users suffering AMI, due to the highly thrombogenic milieu produced by AAS.\textsuperscript{8} Thromboembolic events in the setting of AAS-induced AMI have been documented either as stroke or as renal infarction.\textsuperscript{8} The detection of mild spontaneous echo contrast within the LV cavity of our case report was in line with the prothrombotic potential of AAS. Spontaneous echo contrast within the LV cavity is well-reported in the context of severe LV dysfunction or LV aneurysm.\textsuperscript{48} Thus, the mild myocardial dysfunction of the patient of our case report indicates that the possible cause of LV spontaneous echo contrast was a prothrombotic condition, rather than severe wall motion abnormality.

\textbf{Vasospastic mechanism.} One plausible explanation for the documentation of patent coronary arteries in some coronary angiograms and/or autopsies of AAS users having suffered AMI is the AAS-induced coronary vasospasm.\textsuperscript{13,16,24,27} Indeed, chronic treatment of rabbits with AAS was shown to result in enhanced response of aorta to vasoconstrictors and attenuated response to vasodilators, which were attributed to the reduction of nitric oxide-mediated relaxation, through the inhibition of guanylate cyclase.\textsuperscript{38,39} Even more, AAS abuse has been found to induce vascular dysfunction, affecting both endothelial-dependent and endothelial-independent vasodilatation.\textsuperscript{49–51} From this point of view, chronic AAS treatment may promote coronary vasospasm, albeit the effects of AAS on coronary vascular function have not been investigated yet. This issue needs further investigation.

\begin{figure}
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\includegraphics[width=\textwidth]{figure4.png}
\caption{Mechanisms by which anabolic androgenic steroids can cause acute myocardial infarction.}
\end{figure}

\textbf{AAS:} anabolic androgenic steroid; \textbf{AMI:} acute myocardial infarction; \textbf{cGMP:} cyclic guanosine monophosphate; \textbf{HDL-C:} high-density lipoprotein-cholesterol; \textbf{LDL-C:} low-density lipoprotein-cholesterol
Short-term complications after AAS-induced AMI

Athletes abusing AAS may tend to ignore nonsevere coronary artery disease-related warning signals, possibly due to the potential AAS-induced mental changes and the misinterpretation of these symptoms as exercise-induced musculoskeletal injuries. This fact may lead to the late presentation with AMI of athletes abusing AAS, deteriorating their prognosis.

Ischaemia/reperfusion injury. Chronic treatment of rat hearts with AAS for at least six weeks has been consistently shown to increase myocardial infarct size and myocardial susceptibility to ischaemia/reperfusion injury and to reduce responsiveness to postconditioning cardioprotection. Although studies investigating the causal underlying mechanisms for these effects have not been performed yet, pathways implicated in the process of ischaemia/reperfusion injury have been shown to be differentially regulated in these rat models, suggesting the potential involvement of the relevant pathways in the pathogenesis of AAS-induced adverse myocardial effects after AMI. Specifically, downregulation of both myocardial phosphorylated Akt and phosphatase PP2A levels, which are important enzymes in both cardiomyocyte growth and protection, as well as reduction of antioxidant heart capacity were found in these animal models. Moreover, these adverse post-AMI effects of AAS on rats were accompanied by attenuation of the exercise-induced activation of antioxidant enzymes, implying that athletes abusing AAS may not gain the beneficial antioxidant effects of exercise. Furthermore, the upregulation of tumour necrosis factor-α and myocardial cyclic adenosine monophosphate in these rat models may further contribute to the AAS-induced ischaemia/reperfusion injury.

Myocardial metabolism. AASs have been shown to result in swelling and disintegration of the mitochondria accompanied by adverse structural changes in the sarcomeric units of rat myocardial cells. The extrapolation of experimental data from rats to humans indicates the existence of a possible direct AAS-induced impairment of aerobic mitochondrial metabolism in the myocardium of AAS abusers suffering AMI, which may lower the threshold for the shift of ischaemic myocardium to the less effective anaerobic metabolism.

Myocardial dysfunction after AMI. Chronic AAS abuse has been associated with impaired biventricular diastolic function, as reflected by decreased early diastolic filling, possibly due to decreased ventricular relaxation. The reduction of ventricular compliance by AAS has been shown to be caused by increased fibrosis of the myocardium in rats, which can be attributed to increased crosslink formation between collagen strands, rather than to increased myocardial collagen content. Even more, chronic AAS abuse can induce subtle changes in ventricular systolic function, as reflected by decreased LV strain with preserved ejection fraction. Thus, the total magnitude of myocardial dysfunction after AMI in AAS users results from not only AMI-induced myocardial dysfunction, but also AAS-induced mild myocardial dysfunction, tending to deteriorate further the prognosis of these patients after AMI. Consistently, testosterone pretreatment of rats before experimentally induced AMI resulted in lower fractional shortening, despite unchanged infarct size.

Ventricular arrhythmias. Acute administration of nandrolone 10 min before induction of experimental myocardial ischaemia in rats resulted in a lower fraction of rats surviving ischaemia and higher incidence of ventricular fibrillation, despite no difference in the size of the area of ischaemia and in haemodynamic profile between treatment groups, implying a possible direct proarrhythmic effect of nandrolone in the context of myocardial ischaemia. Hence, AAS abuse possibly increases the risk of life-threatening ventricular arrhythmias, predisposing to sudden cardiac death, albeit the underlying mechanisms remain to be elucidated. Possible underlying mechanisms for the proarrhythmic effects of AAS in the context of myocardial ischaemia include direct myocardial injury with structural myocardial changes, inappropriate adrenergic activation and heterogeneity of repolarization.

Direct myocardial injury. With regard to the AAS-induced direct myocardial injury, the most common pathological finding in autopsied hearts of individuals with chronic AAS abuse is LV hypertrophy, frequently associated with fibrosis and myocytolysis. This pattern of LV hypertrophy may be potentially proarrhythmic, as the myocardial disarray in hypertrophic myocardioptrophy is well-known to be proarrhythmic. AAS can bind to myocardial androgen receptors and may directly cause hypertrophy with echocardiographically detected increase in ventricular wall thickness after periods of AAS use as short as three months. AASs have been demonstrated to have a long lasting hypertrophic effect on the myocardium, as former users still show an increase in LV muscle mass at least 12 months after discontinuing AAS. Ex-users lie between non-users and users with respect to the magnitude of LV hypertrophy, indicating that the hypertrophic effect of AAS decreases over the years. However, the extent of the reversibility of AAS-induced myocardial fibrosis after discontinuation of AAS treatment remains unknown, but its importance lies in the...
potential proarrhythmic effect of fibrous myocardial regions.

There is convincing evidence that AAS-induced myocardial toxicity may be more possible with non-
alylated AASs compared with 17-α-alkylated AASs.57,58 Nonalkylated AASs are deactivated by first-pass metabolism and are administered only parenterally, whereas 17-α-alkylated AASs can escape first-pass metabolism.71 AAS-induced myocardial toxicity appears to be more relevant with the parenterally administered nonalkylated AASs, which contradicts with the greater hepatotoxic potential of the orally taken 17-α-alkylated AASs.58

Adrenergic activation. Chronic AAS abuse has been shown to induce a sympathovagal imbalance in the cardiac autonomic regulation with sympathetic dominance, which may be proarrhythmic in the early period after AMI.72 Upregulation of adrenergic activation after AMI by AAS can be attributed not only to increased catecholamine release, but also to enhanced β2-adrenoceptor expression. Indeed, AASs have been found to inhibit reuptake of catecholamines in extraneuronal tissues, promoting an increment of catecholamine concentrations at receptor sites.73 Neuronal catecholamine transporter is responsible for not only reuptake of noradrenaline, but also nonexocytotic release of noradrenaline from sympathetic nerve terminals during ischaemia.74 Taking into account that increased release of noradrenaline has been shown to enhance ischaemia-induced arrhythmias in rat hearts, the enhanced noradrenaline release in hearts of AAS users after AMI may predispose them to malignant ventricular arrhythmias.74 Even more, AAS administration has been shown to increase β2-adrenoceptor expression and to reverse the normal post-stress down-regulation of adrenergic-response in rat hearts, suggesting increased risk for malignant arrhythmias in AAS users under stressing conditions, such as early after AMI.75

Myocardial repolarization. Chronic AAS use is well-known to induce shortening of QT interval, attributed to increased density of both inward and delayed rectifier potassium currents.76–79 Athletes normally have prolonged QTc interval, which is attributed to the increased LV mass.80 Moreover, although athletes are normally characterized by decreased QT dispersion, athletes using AASs appear to have increased both QT dispersion and interval from the peak to the end of the electrocardiographic T wave, indicating increased heterogeneity of repolarization, possibly predisposing them to malignant ventricular arrhythmias.79,81,82 Thus, taking into account that AMI survivors with increased QT dispersion are at increased risk of developing ventricular arrhythmias and have increased cardiac mortality, the history of recent AAS abuse may predispose them to ventricular arrhythmias and deteriorate prognosis after AMI.83 The proarrhythmic potential of AAS in the period after AMI is possibly further enhanced by the reported increased incidence of late potentials in the signal-averaged electrocardiogram of AAS users, which result in an electrophysiological instability, providing a re-entry substrate for ventricular arrhythmias.84

Long-term complications after AAS-induced AMI

There is only one study, published 40 years ago, which investigated the impact of AAS treatment on the prognosis of humans experiencing AMI.85 This study found no significant difference in the mortality after AMI between the AAS-treated and -untreated patients.85 An important limitation of this study was the fact that the participants received methandrostenolone therapy only after the occurrence of AMI and they were not pretreated with AAS before AMI.85 Therefore, the study design was not relevant to the real life setting of the occurrence of AMI after chronic AAS abuse. Moreover, this study was underpowered to detect significant differences in the cardiovascular events between the treatment group and the control group.

The VALIANT study demonstrated that baseline LV hypertrophy, and especially concentric LV hypertrophy, portends an increased long-term risk for cardiovascular morbidity and mortality following AMI.86 Therefore, AAS-induced concentric LV hypertrophy may predispose AAS users to long-term complications after AMI. The abnormal LV hypertrophy of athletes taking AASs, which is characterized by fibrosis and myocytolysis, may have greater adverse prognostic significance after AMI, compared with the physiological training-induced LV hypertrophy.66

Conclusions

We report a case of a young bodybuilder with no significant past medical history who presented with chest pain due to AMI associated with AAS abuse. Physicians should always consider the possibility of AAS abuse in the context of a young athlete suffering AMI, especially when accompanied by considerable downregulation of HDL-C. Thrombosis without underlying atherosclerosis or vasospasm is highly possible to be the cause of AAS-induced AMI. Moreover, complications may be more frequent in AAS users suffering AMI. Thus, education campaigns are needed to increase public awareness about the serious cardiovascular complications of AAS abuse, in order to discourage AAS abuse.
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